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Part II

Department of Labor

Occupational Safety and Health Administration

29 CFR Part 1910, et al.
Air Contaminants; Proposed Rule

DEPARTMENT OF LABOR

Occupational Safety and Health Administration

29 CFR Parts 1910, 1915, 1917, 1918, 1926, 1928

[Docket No. H-020 A]

Air Contaminants

AGENCY: Occupational Safety and Health Administration (OSHA), Department of Labor.

ACTION: Proposed rule.

SUMMARY: The Occupational Safety and Health Administration (OSHA) proposes to amend its existing air contaminant standards that set permissible exposure limits (PELs) for the maritime, construction and agriculture industry sectors. The existing standards for maritime and construction are based on either the 1968 or 1970 "Threshold Limit Values of Airborne Contaminants" of the American Conference of Government Industrial Hygienists (ACGIH). There are no permissible exposure limits applicable to agricultural employees.

Since those ACGIH recommendations were compiled, there has been new research and many new substances have entered general use. As a result both ACGIH and the National Institute of Occupational Safety and Health (NIOSH) have recommended new or lower exposure limits for many substances.

Based on those recommendations, on June 7, 1988 (53 FR 20960), OSHA proposed new exposure limits for general industry. After reviewing the record of a lengthy public hearing, many comments and extensive scientific data, OSHA issued on January 19, 1989 (54 FR 2332) new exposure limits for general industry. OSHA stated after reviewing feasibility data, it would propose the same new exposure limits for construction, maritime and agricultural employees.

In this notice of proposed rulemaking OSHA is proposing to issue more protective exposure limits for approximately 210 substances currently regulated in the construction and maritime industries and add new exposure limits for approximately 160 chemicals to protect these workers.

OSHA is also proposing that employees in agriculture be covered by these PELs as well as by approximately 220 additional limits which currently exist in general industry, maritime and construction, but do not exist for the agricultural industry. By appropriations act rider, only employees of farms with more than 10 employees are covered by OSHA standards.

OSHA preliminarily concludes that the new or more protective limits will substantially reduce significant risk of material impairment of health for construction, maritime and agriculture workers and are technologically and economically feasible for those industries. Diseases reduced include heart disease, cancer, neurological disorders, lung, liver and kidney disease, eye disorders, moderate and severe irritation and other disorders.

The new standard will prevent approximately 8 to 13 deaths and 31,000 illnesses per year.

The proposed changes will be carried out for the maritime sectors by adding a new § 1915.1000 for shipyards, by adding new § 1917.1000 for marine terminals and by adding a new § 1918.1000 for longshoring along with necessary consequential amendments. For construction § 1926.55 will be revised and for agriculture a new § 1928.1000 will be added.

DATES: Written comments on the proposed standard must be postmarked on or before September 25, 1992. Notices of Intention to Appear at the informal rulemaking hearings on the proposed standard must be postmarked on or before September 11, 1992. Individuals who wish to comment or appear during the public hearings must see Section VIII of this document for specific requirements.

Parties who request more than 10 minutes for their presentation at the informal public hearing and parties who will submit documentary evidence at the hearing must submit the full text of their testimony and all documentary evidence, postmarked on or before September 25, 1992. The informal rulemaking hearing is scheduled for October 20–30, 1992, in Washington, DC, for November 17–20, 1992 in San Diego, California, and on December 8–11, 1992 in Des Moines, Iowa.

ADDRESSES: Written comments should be submitted to OSHA Docket Office, Docket No. H–020A, room N–2634, U. S. Department of Labor, 200 Constitution Avenue NW., Washington, DC 20210, telephone (202) 523–7894.

Notice of intention to appear, testimony and documentary evidence to be submitted at the hearing are to be sent to Mr. Tom Hall, OSHA Division of Consumer Affairs, Docket No. H–020A, room N–3647, U.S. Department of Labor, 200 Constitution Avenue NW., Washington, DC 20210, telephone (202) 523–8615.

The hearing will be held in Washington, DC in the Auditorium, Frances Perkins Department of Labor Building, Third and Constitution Avenue NW. The informal public hearing will begin at 9:30 a.m. The hearing in San Diego will be held at the Holiday Inn on the Bay, 1355 North Harbor Drive, San Diego, California, 92101; Tel: (619) 232–3861. The hearing in Des Moines will be held at the Holiday Inn Des Moines, 1050 Sixth Ave., Des Moines, Iowa, 50314; Tel: (515) 283–0151.

FOR FURTHER INFORMATION CONTACT: Mr. James F. Foster, Director, Office of Information and Consumer Affairs, OSHA, U.S. Department of Labor, room N–3649, 200 Constitution Avenue NW., Washington, DC 20210, Telephone (202) 523–8151.

SUPPLEMENTARY INFORMATION:

Introduction and Organization of this Document

This **Federal Register** notice proposes to update the exposure limits for air contaminants in the construction, maritime and agriculture sectors. As a result there will be more protective limits for approximately 210 substances currently regulated in construction and maritime and new exposure limits for approximately 160 substances in these sectors. These new and more protective limits will also be made applicable to agriculture as will the approximately 220 limits already applicable to other sectors which have not been applied to agriculture. This notice then proposes to add to 29 CFR a new § 1915.1000 for shipyards, a new § 1917.1000 for marine terminals, a new § 1918.1000 for longshoring along with necessary conforming amendments. Section 1926.55 will be revised for construction and a new § 1928.1000 added for agriculture.

The first part of this document (Sections I, II and III of the preamble) discusses the approach, regulatory history and the general need for updated and new health standards in the construction, maritime and agricultural industries. It discusses the legal basis for this rulemaking and the background of the rulemaking which updated exposure limits for general industry (54 FR 2322–2982; January 19, 1989). Various tables are presented identifying the substances to be covered and the changes being proposed. At the beginning of this part of the preamble there are questions to assist the public in commenting on the proposal.

Next, (in Section IV) the preamble discusses the health effects of the substances which OSHA proposes to regulate. This begins with a general discussion of the principles of toxicology

and dose-response analysis. (See Section IV A and B.) The substances covered are divided into categories based on their health effects, e.g. narcotic effects, ocular effects, systemic toxicity. Each category of substances is reviewed in a separate subsection of Section IV. The discussion begins with a general overview of the relationship between toxic substances in that category and the illnesses or material impairments of health which they cause, followed by individual discussions of each substance in the category addressing illnesses or health impairments associated with each substance and the most important studies which demonstrate those effects.

Because OSHA's health discussions draw on the Air Contaminants rulemaking for General Industry, they cover the major studies in OSHA's view and in the views of participants who submitted comments to that rulemaking. In addition, the discussions incorporate or respond to the comments already made by the public in the general industry rulemaking.

The health discussions in this preamble also incorporate newer studies (generally post 1987) which were not available prior to the general industry rulemaking and studies which may have specifically covered workers in the construction, maritime and agriculture sectors. Finally the individual discussions include OSHA's reasoning and preliminary conclusion that the new exposure limit is necessary to substantially reduce or eliminate a significant risk of material impairment of heath or functional capacity.

Section V of the preamble addresses the technologic and economic feasibility of the proposed exposure limits for the construction, maritime and agriculture sectors. A separate analysis is presented for shipyards, and for longshoring and marine terminals. This section also includes an estimate of the overall benefits for purposes of Executive Order 12291, and presents OSHA's preliminary conclusions on the technical and economic feasibility of the proposed new exposure limits.

This section of the preamble (V) then includes an analysis of the impact on small business under the Regulatory Flexibility Act. It also includes an environmental impact assessment.

Section VI contains the Clearance of Information Collection Requirements and the State Plan Applicability. Section VII of the preamble discusses the specific regulatory language for each sector. The regulatory changes are more complex for the maritime sector because of the need to integrate them with detailed existing provisions unique to those sectors. This is followed, in Section VIII, by information on how the public can file written comments and participate in the oral public hearings. The hearing rules are discussed. Reference to authority under which the document was prepared is made in Section IX. The proposed regulatory text and tables are presented in Section X, followed by appendixes in Section XI.

OSHA's intention is that after this rule is completed, all employees under OSHA's jurisdiction will be covered by the PELs. If any subsector has not been evaluated and discussed by OSHA, it should assume that OSHA's intention is to cover it, and it should submit relevant comments.

Table of Contents

I. Background
A. Need and Approach
B. Comments Solicited from Interested Parties
C. History and Need for Revision of the PELs in Construction
D. History and Need for Revision of the PELs in Maritime
E. History and Need for Establishing PELs in Agriculture
F. Substances Included in the Update of PELs for Construction, Maritime and Agriculture
G. Index to Preamble Discussion of Individual Substances
H. Mixture or Computation Formulae
II. Pertinent Legal Authority
III. Glossary
IV. Health Effects of Substances to be Regulated
A. General Principles of Toxicology and Dose-Response
B. Historical Development of Occupational Exposure Limits
C. Description of the Substances for Which Limits Are Being Proposed
1. Substances for Which Proposed Limits Are Based on Avoidance of Neuropathic Effects
2. Substances for Which Proposed Limits Are Based on Avoidance of Narcotic Effects
3. Substances for Which Proposed Limits are Based on Avoidance of Sensory Irritation
4. Substances for Which Proposed Limits are Based on Avoidance of Liver or Kidney Effects
5. Substances for Which Proposed Limits are Based on Avoidance of Ocular Effects
6. Substances for Which Proposed Limits are Based on Avoidance of Respiratory Effects
7. Substances for Which Proposed Limits are Based on Avoidance of Cardiovascular Effects
8. Substances for Which Proposed Limits are Based on Avoidance of Systemic Toxicity
9. Substances for Which Proposed Limits are Based on No Observed Adverse-Effect Levels
10. Substances for Which Proposed Limits are Based on Avoidance of Physical Irritation and Other Effects
11. Substances for Which Proposed Limits are Based on Analogy to Related Substances
12. Substances for Which Proposed Limits are Based on Avoidance of Biochemical/Metabolic Effects
13. Substances for Which Proposed Limits are Based on Avoidance of Sensitization Effects
14. Substances for Which Proposed Limits are Based on Avoidance of Cancer
15. Substances for Which 1987–1988 ACGIH TLVs Are Less Stringent Than Existing OSHA PELs
16. Substances for Which OSHA Is Proposing to Establish Short-Term Exposure Limits
17. Substances for Which OSHA is Proposing to Add Skin Notations
D. References for Section IV
V. Preliminary Feasibility, Regulatory Impact, Regulatory Flexibility and Environmental Impact Analyses
A. Executive Summary
B. Preliminary Regulatory Impact Analysis for Construction
C. Preliminary Regulatory Impact Analysis for Maritime
D. Preliminary Regulatory Impact Analysis for Agriculture
E. Preliminary Regulatory Impact Analysis for General Industry
VI. Clearance of Information Collection Requirements and State Plan Applicability
VII. Summary and Explanation of the Proposed Standard Construction
Maritime
Agriculture
VIII. Public Participation and Public Hearings
IX. Index of Terminology and Authority
X. Standard
XI. Appendices

I. Background

A. Need and Approach

There are 5 million workers in construction and 180,000 workers in the maritime industries who may be currently exposed to toxic substances which have no or inadequately protective exposure limits in these sectors. There are approximately 780,000 workers in agriculture under OSHA jurisdiction who may be exposed to some of the approximately 600 toxic chemicals which have protective exposure limits for workers in the general industry sectors. There are no protective exposure limits for these agricultural workers.

It is among OSHA's highest priorities to extend this needed health protection to all workers under OSHA's jurisdiction. All workers under OSHA's jurisdiction are equally important. The OSH Act states "The Congress declares it to be its purpose and policy * * * to assure so far as possible every working

man and woman in the nation safe and healthful working conditions * * *" (section (2)(b); emphasis added). OSHA estimates that accomplishing this goal will save 8 to 13 lives per year and prevent 31,000 illnesses annually.

In order to promulgate this rule efficiently and in a reasonable time, OSHA intends to utilize analyses previously conducted by OSHA and by other reputable scientific bodies. This information will be integrated with the latest available scientific and feasibility data.

OSHA believes the regulatory approach it has adopted is the best method for accomplishing the goal while meeting legal requirements and being consistent with good science.

The first step in the approach is to make use of the scientific work, public comment and experience gained during the Air Contaminants rulemaking for general industry. In that rulemaking, proposed June 7, 1988 (53 FR 20960) and issued January 19, 1989 (54 FR 2332), OSHA explained that it had issued more protective health standards for only 26 substances in its 18 years. However, there were many hundreds of toxic substances not covered by OSHA regulations and, in addition, many substances regulated by OSHA in its Z-tables were covered by exposure limits that were considered inadequate by the scientific community. It was clear that OSHA needed a more systematic approach to regulation; it had to move beyond substance-by-substance rulemaking.

OSHA determined that setting new exposures limits for a large number of toxic substances was of higher priority for protecting workers than promulgating a few standards with complete ancillary provisions (e.g., medical surveillance, exposure monitoring). It concluded that making use of the existing scientific work of the National Institute for Occupational Safety and Health (NIOSH) and the American Conference of Government Industrial Hygienists (ACGIH) as a starting point in OSHA's analysis, would expedite the regulatory process while still insuring that health decisions were based on sound science.

In developing and promulgating its General Industry Air Contaminants rule, OSHA reviewed the existing literature on control technology and its costs, engaged in a massive survey of 5700 firms in industry, reviewed approximately 1500 public comments and more than 2000 scientific studies, and held extensive public hearings. Consequently, its final decisions were based on full public input and complied with required rulemaking procedures. See discussions at 53 FR 20962–67, 20977–81 and 54 FR 2333–39, 2361–2383 for an extended discussion of OSHA's approach.

OSHA determined that it would be most effective to extend the new exposure limits to the construction, maritime, and agriculture sectors in a second rulemaking. This would permit OSHA to consult with the Construction Advisory Committee and the Shipyard Advisory Committee, to meet with representatives of the longshore and marine terminal sectors, and to make presentations to and have meetings with the agriculture community. In addition, it would give OSHA the time and resources to extensively analyze feasibility for these sectors. See 54 FR 2383. OSHA has completed its advisory committee consultations and economic analyses.

In addition, OSHA will consult with the Departments of Transportation and Agriculture, the Environmental Protection Agency, the Small Business Administration, and the Office of Science and Technology Policy regarding the extension of revised exposure limits to these three major industry sectors, and to small businesses within each sector.

OSHA is only opening the record for construction and maritime for the 376 substances where it concluded new or changed limits were appropriate in general industry. For each substance OSHA is proposing the exposure limit it concluded was appropriate in the 1989 General Industry rulemaking, which was determined after extensive review of scientific literature and public comments.

In addition, OSHA is opening the record for consideration of exposure limits for fiberglass, mineral wool and asphalt for construction, maritime, agriculture and general industry. OSHA did not reach final conclusions on these substances in the general industry rulemaking.

There are a few substances where OSHA has issued substance specific standards for general industry (in 29 CFR 1910.1001–1048), but has not made the exposure limit applicable to construction or maritime. These include lead for construction and cotton dust for both construction and maritime. OSHA is proposing to apply the exposure limit from the substance specific general industry standard to construction and maritime (but not the ancillary provisions which are not being considered in this rulemaking).

There are no exposure limits applicable to agriculture. OSHA believes that the Act, the need for health protection and good health policy all require that agricultural workers should be protected from the effects of toxic chemicals. (By appropriations rider, OSHA cannot issue or enforce regulations on agricultural operations with no more than 10 employees.) Accordingly, OSHA is proposing that agricultural workers be protected by all the exposure limits applicable to workers in other sectors. This includes exposure limits for the 376 substances being covered for construction and maritime, the approximately 220 substances already applicable to other workers, and the substances regulated with single-substance standards.

OSHA wishes to make it absolutely clear that it is considering the group of 220 substances only for agriculture. Opening the record for other sectors would excessively delay this rulemaking and workers already have protection in the other sectors.

In addition, OSHA is not opening the record of the 376 substances for general industry, and will not consider those substances for general industry. OSHA has just completed a review of all of these substances for general industry and it would be inappropriate to review them so shortly after full consideration. The only substances being considered for general industry are fibrous glass, mineral wool and asphalt fumes.

OSHA is not opening the record or considering any comments on ancillary provisions such as medical surveillance and exposure monitoring in this rulemaking. OSHA has already commenced consideration of whether to issue general exposure monitoring and medical surveillance provisions. Advanced notices of proposed rulemaking have been issued, and a major study is in process on medical surveillance. These matters are in the published regulatory agenda as separate rulemakings. It would unnecessarily delay this rulemaking and exposure limit protection for construction, agriculture and maritime workers to attempt to consider ancillary provisions in this rulemaking.

Table I–F lists the substances being considered for each sector and for the maritime subsectors.

In the future OSHA intends to update its Air Contaminant exposure limits at approximately 3 to 5 year intervals. These updates will consider changes to existing limits and inclusion of new substances. They will cover all sectors and workers within OSHA's jurisdiction. This process is listed in the published regulatory agenda. These updates are a more appropriate forum to consider, across the board, changes in current general industry exposure limits.

OSHA concludes, based on its expertise and experience, that this approach to air contaminant regulation is the best method for increasing health protection for all American workers in a reasonable time. As discussed below in the Legal Authority section, OSHA concludes this approach is legal. OSHA will continue to engage in single substance rulemakings as appropriate.

Technical Matters

OSHA is not considering in this rulemaking the 13 substances (29 CFR 1910.1003–16) for which it has work practices but no exposure limits. This rulemaking is designated to cover exposure limits. It is also not considering nine substances currently involved in ongoing single substance rulemakings. OSHA has already completed extensive consideration of these substances in those rulemakings, and it would be wasteful of resources to reconsider them here. It is also not considering a few substances for agriculture (e.g., coke oven emissions) which have absolutely no relevance in that sector.

OSHA will consider in the first future update and not this rulemaking, different limits for the four substances that it has committed to reconsider in response to the legal challenge brought by the AFL/CIO to the General Industry rule. These substances are carbon tetrachloride, vinyl bromide, trichloroethylene and gasoline. It will also reconsider the four substances it agreed to reconsider pursuant to settlement agreements reached with industry. These are nitroglycerin and ethylene glycol dinitrate for the civilian explosives industry and calcium oxide and hydroxide. The first update is a better forum because all industry sectors can be efficiently considered at one time.

Existing exposure limits vary among industry sectors. The existing limits for General Industry, Longshoring and Marine Terminals are based on the 1971 OSHA PELs, which were originally based on the 1968 ACGIH TLVs. The existing limits for Construction and Shipyards are based on the 1970 ACGIH TLVs. (See also the Legal Authority discussion on Shipyards for a further analysis). There are no existing limits for Agriculture. The Transitional Limits in the proposed Z–Tables are the existing limits for each sector, and vary accordingly. The Transitional Limits are effective until the new limits are phased in.

A few of the 376 substances changed for general industry are not being opened for consideration for Construction and Shipyards because the 1970 TLVs (the current construction PELs) already included the change which OSHA adopted in 1989 for General Industry.

Also, OSHA is not opening the record for Construction and Maritime for consideration of the 52 substances it considered but determined not to change for General Industry. It would be an inefficient use of resources to reopen in this proceeding substances for which OSHA so recently concluded, based on a full rulemaking record, that change was inappropriate at that time.

OSHA has attempted to make the tables of exposure limits for Shipyards, Marine Terminals & Longshoring, Construction and Agriculture as useful as possible. In keeping with its practice for General Industry, there will be a Table Z, Construction; a Table Z, Shipyards; a Table Z, Longshoring & Marine Terminals; and a Table Z, Agriculture. However, there will be only one Table Z for each sector, in contrast to the three for General Industry; Table Z–1–A, Z–2 and Z–3. General industry has 3 Tables because (1) Table Z–2 is based on ANSI standards, which utilize a different definition of ceiling than does ACGIH or OSHA. (The Z–2 definition of maximum peak is somewhat similar to OSHA's concept of a STEL.) And (2) Table Z–3 presents mineral dust limits, some of which are formulas, not specific numbers.

OSHA believes that a single table, with the same concepts and specific numbers rather than complex formulae will be easier for the public to use, understand and comply with. Accordingly OSHA is, where necessary for Table Z–2 substances, utilizing the Peak levels as STELs and not incorporating the ceilings. In addition, formulas are being converted to the nearest gravimetric measurement (weight per unit volume).

OSHA believes these slight adjustments are in the nature of technical changes and make no substantive difference in either health or feasibility. OSHA intends to make similar changes for General Industry as technical amendments in the future to achieve uniformity and consistency across all sectors regulated by OSHA.

The new Z–Tables list all substances for which OSHA has exposure limits, including those substances where there are single substance standards with ancillary provisions that are applicable. These substances will be footnoted or textual cross referenced indicating where the ancillary provisions are located. This will make it easier for the public to determine whether a substance is regulated by OSHA, what the exposure limit is, and whether there are ancillary provisions.

Approximately 100 substances listed in the Z–Tables are registered pesticides regulated by EPA. Where an agricultural employer uses any registered pesticide in accordance with the EPA label, OSHA has no jurisdiction pursuant to section (4)(b)(1) of the OSH Act. If the substance is used for non-pesticide purposes or the agricultural employer makes the substance and it is not labelled, then the OSHA exposure limit applies. See the discussion under Legal Authority.

B. Comments Solicited from Interested Parties

OSHA requests comments on all issues raised by this proposal including health effects, feasibility, risk and policy issues. The following are some specific questions which may assist interested parties in preparing their comments. If a commenter has data in a less detailed form than OSHA requests or just partial information to some questions, the information would still be useful to OSHA, and the commenter is requested to submit the data or information in whatever form it exists.

"Risk-Risk Analysis"

OSHA has preliminarily concluded that this standard could prevent approximately 8 to 13 deaths and 31,000 illnesses per year. See also, the "Economic Impacts" section of the PRIA below, V–B (Construction), V–C (Maritime), V–D (Agriculture), V–E (General Industry) for further information on OSHA's preliminary economic impact estimates. OSHA notes that these benefit estimates may change pending consideration of public comments about this proposed rule and the consideration of the issues raised by OMB.

OMB requested that OSHA consider "[h]ow will compliance with the proposed PEL rule affect workers' employment, wages, and therefore health?" OMB asked OSHA to assess comprehensively whether the net health benefit of this rule to workers is positive or negative. The objective of OMB'S request is to weigh the risks to workers from exposure to toxic substances regulated under the rule with the health risks posed to those same workers if compliance with the proposal lowers their incomes or employment levels. OMB asked OSHA to give serious thought to this kind of analysis to determine, in our judgement, if the arguments raised by OMB were valid, and to consider sponsoring additional empirical work in the future.

OMB has requested that the Department study this issue and section 3(a) of Executive Order 12291, states "each agency shall, in connection with every major rule, prepare, and to the extent permitted by law consider, a Regulatory Impact Analysis." If the type of analysis suggested by OMB is able to generate a clearer picture of the total benefits provided workers by regulatory proposals, then the Agency and the public will be better informed of the likely impact of the regulations. We clearly recognize, however, that information generated by any study can be used by OSHA only in accordance with the OSH Act and controlling case law. See the "Pertinent Legal Authority" section, infra, at Section II. The information needs to be relevant to analyzing whether workers are at significant risk of material impairment of health or whether compliance with the rules is feasible for it to be legally relevant to OSHA's decisions regarding the PELs.

The Department has thus agreed to study the questions raised by OMB, to the extent applicable to worker health, and will consider whether weighing the risks to workers' from exposures to toxic substances with the risks associated with lowering those same workers' incomes is appropriate and relevant. OSHA will consider the issue while simultaneously moving forward with the rulemaking.

To assist OSHA is assessing the issues raised by OMB, OSHA is soliciting comments on the following questions and on any other information that the commentator believes will assist OSHA in meeting its statutory obligations.

1. What are the advantages and disadvantages of introducing "risk-risk" analysis into this rule? Specifically, has the methodology been tested and verified?

2. What is the validity of the suggested economic analysis proposed by OMB?

3. How valid is the issue OMB raises with respect to the linkage between income and health?

4. How valid is the issue OMB raises with respect to the linkage between the costs of regulation and workers' incomes?

5. Can one empirically estimate the impact of the cost of a regulation on the incomes of the same workers?

6. Comment is requested on all aspects of whether any such analysis should consider indirect effects only on workers covered by the rule, or those effects on all workers covered by the OSH Act?

7. What general equilibrium considerations, if any, would need to be taken into account in performing such an analysis?

8. What macro-economic considerations, if any, would need to be taken into account in performing such an analysis?

9. Are there any methodological issues, not covered by the above questions, with the suggested economic analysis proposed by OMB?

To contribute to OSHA's effort to examine whether the estimated health benefits of this rule to workers are positive or negative, OSHA solicits public comment on concerns raised by OMB. Specifically, commenters should describe whether there are modelling techniques, data sources, or parameter estimates which provide accurate and unbiased estimates of two key relationships associated with this question: (1) The relationship between OSHA regulations (or this rule in particular) and measures of workers' economic welfare such as financial wealth, income, wages and employment status; (2) The relationship between these measures of workers' economic welfare and measures of health status such as mortality.

Regarding the relationship, if any, between regulatory compliance and changes in workers' economic welfare, commenters may discuss whether this rule is likely to affect workers' real income, employment, and/or productivity. Estimates of potential employment and wage effects due to OSHA regulations in general would also be helpful in this analysis. Most useful in such analysis would be data describing what changes in workers' economic welfare could occur in the industries affected by this rulemaking. Comments may include retrospective analyses of the effect of past OSHA regulations on workers' economic welfare, and discussions of why this rule's effects may be similar or different. Alternatively, commenters may offer prospective analyses of the effects of this rule. All comments should justify the underlying assumptions about key parameters in the analysis. Finally, commenters may discuss whether and how particular changes in variables such as employment or wages are in fact attributable to the rule.

Regarding the relationship, if any, between workers' economic welfare and their health status, OMB is interested in OSHA obtaining information on any research or studies which examine the positive relationship between income and health status. A preliminary review of the literature points to several studies which estimate the size of this income/health relationship. OMB is interested in OSHA obtaining information on all aspects of these studies, including the quality of their estimates and their applicability to the employment context. OMB is interested in OSHA obtaining information about the accuracy of these estimates and the existence of a relationship. This list is not comprehensive, and commenters are encouraged to submit any additional data or studies. Comments which consider international data on how incomes and health, or mortality, vary either over time or across countries are also invited.

OMB has provided OSHA with a summary and analysis of research which OMB cited as relevant to the questions it raised above regarding this proposed rule. OSHA solicits public comment on this research and on the questions raised by OMB to the extent relevant under the OSH Act to workers' health. OMB's summary and analysis of this research is listed as follows.

OMB Summary and Analysis of Selected Studies on Income and Health

The most careful large sample studies by Anderson and Burkhauser, by Duleep, and by Wolfson *et al.*, seem to suggest that losses in wages in the range of $1.9 to $6.5 million dollars annually are associated with one additional fatality annually. Although causality is not proven, an association between income and mortality persists after controlling for prior health status, for educational attainment, and for exposure to quantifiable occupational risks. Further, the association exists, although more weakly, at high income levels. Although most of the more careful studies focus on wages and worker mortality, the general effect that they are trying to measure is the relation between real income broadly defined, and health status of the family.

Several studies are summarized in the following table. To facilitate comparisons of the results of the various studies, the table includes estimates of the income gain necessary to avert one fatality. These estimates of "willingness-to-spend", which are calculated from the effect of income on mortality reported in the papers, are intended to be illustrative and not definitive. As the effect of income on mortality is likely to depend on age, income, and number of years of observations, estimates of willingness-to-spend to save a statistical life similarly depend on these variables. Differences among studies in the estimated willingness to spend depend in part on differences in the means and distributions of the age of respondents,

their income, and the number of years over which mortality was observed.

Among the studies is a commonly cited one by Ralph Keeney (1990), who estimated that under a particular set of assumptions, regulatory costs equal to $7.25 million could induce one fatality. His estimate is based in part on correlations between income and mortality reported in Kitagawa and Hauser (1973), who used 1960 Census data and matched death certificates for the 4 months following the month of the Census.

Another commonly cited study is More Medical Care, Better Health: An Economic Analysis of Mortality Rates, (1982) by Jack Hadley, Co-Director of Community and Family Medicine, Georgetown University. Hadley showed that increase in total family income of 1% were associated with statistically significant declines in mortality of 0.07% for white males between 45 and 64, and of 0.116% for black males between 45 and 64.[1] He used aggregate cross-sectional data for 400 county groups for 1970, and focused on mortality rates by age group, by sex and by race. Income effects were also large for white and black infants of both sexes. For some groups, e.g., black females, increases in income were associated with increases in mortality, but these were not statistically significantly different from zero.

Aggregate cross-sectional analyses, of which Hadley's study is a well-known example, tend to suffer from several well-known difficulties. First, measurement error in the explanatory variables due to the misclassification of data is likely to bias the estimates towards zero. Measurement error may result because current family income is a poor proxy for permanent income with which current mortality should correlate, and because decedents may have crossed county lines prior to death. The failure to correct for geographic variations in the cost of living also introduces measurement error. Second, since income effects on mortality are estimated separately for different subgroups of the populations, e.g., men, the estimated effect likely understates the overall effect of income changes on mortality. In fact the true effect of total family income on mortality may be the sum of the income effects for each subgroup making up a family. Finally, since healthier populations that have lower mortality can work harder and earn more, the estimated elasticity may be exaggerated by failing to correct for reverse causality.[2]

A 1984 study of the Joint Economic Committee (JEC) of Congress entitled "Estimating the Effects of Economic Change on National Health and Social Well-Being" reports that the 3% decline in real per capita income below trend during the 1973 recession is associated with a 3% increase in total mortality. Written by M. Harvey Brenner of Johns Hopkins University, the report examines relationships between economic variables such as changes in income, and unemployment, and indicators of social well-being, including mortality rates. Applying the reported relationships to today's economy implies that a decline in per capita income of 3%, or about $690 per capita would result in an increase in mortality of 3%, or about 65,000 premature deaths. A decline in income of between $1.8 million and $2.7 million would thus be associated with one death.[3]

The estimate by the JEC may be an overstatement of the long-run effect of income on health, since it reflects the effects of transitional income changes. Such income changes are likely to be associated with changes in mortality greater than those caused by permanent changes in income, since the former are associated with more stress than the latter.

Kathryn Anderson and Richard Burkhauser showed that an increase in wages of $1.00 (17%) implies a 4.2% decline in mortality risk over the next 10 years, (p. 321).[4] Their study, published in 1985 in the Journal of Human Resources, used 10 years of mortality data from 4878 male workers aged 58 to 63, to analyze the joint determination of health and retirement from the labor force. A decline in mortality risk of 4.2%, given their sample amounts to 200 fewer deaths over the 10 years, or approximately 20 fewer deaths per year. Thus an increase in wages of $1.00 per hour for the 4878 workers is expected to prevent 20 deaths annually. If workers are paid for 2080 hours annually, then the annual wage gain necessary to avert one death is approximately $510,000 in 1969 dollars. Using the Consumer Price Index to adjust for inflation implies that an increase in wages of $1.9 million would prevent one premature fatality.

Anderson and Burkhauser's study also implies that an increase of $10,000 in the value of worker's homes lowered the risk of death by 0.79%, and prevented 3.8 deaths annually. To translate these changes in the stock of wealth to changes in income flows, note that the annualized equivalent of $10,000 is $890 annually, if borrowed at mortgage rates of 8% for 30 years. Thus gains in income of $4.3 million (4878×$890 of additional income annually) are associated with 3.8 fewer deaths. Using the Consumer Price Index to convert these 1969 dollars to current dollars implies that a gain in income of $4.3 million should be associated with one averted death.

Several qualifications are necessary before interpreting the Anderson and Burkhauser results in this manner. Analyses on the mortality of older persons may underestimate the effect of income on mortality if low income people have already died at younger ages. Finally, as mentioned by Duleep, (1986), income's effect on mortality is dependent on the extent to which death is preventable. At older ages the income effect falls as death becomes unavoidable.

Harriet Duleep, using a sample of 9618 white married men showed that earnings of less than $3000 instead of more than $9000 have a direct effect on the probability of death by age fifty, of 0.089, and an indirect effect, (through the probability of becoming disabled), of 0.028, for a total effect of 0.117. Her study, published in 1986 in the Journal of Human Resources used data on earnings, disability, and mortality of men aged 35 to 64 to estimate the effects of earnings on mortality. Her analysis held constant educational achievement, corrected for the reverse causality of health on income by controlling for whether the workers were initially disabled, and used sophisticated statistical methods. Although Duleep's

[1] See Hadley, p. 112.

[2] Taking Hadley's estimate of 0.07% for the elasticity of middle-aged male mortality with respect to income implies that an income loss of $38 million is necessary to induce one fatality. This figure, which is higher than those mentioned below, is calculated by dividing a hypothetical 1% decline in US GNP, (57 billion), by 1500 deaths, the 0.07% increase in mortality that is predicted to occur by applying Hadley's elasticity estimate to U.S. mortality. Since lower elasticity estimates result in larger required income losses to induce one fatality, the fact that the $38 million estimate seems high may be ascribed to the various factors that appear to lower the estimated elasticity.

[3] The higher figure from applying the elasticity of unity to 1991 income and mortality data. In particular, a 1% decline in current GNP is a loss of about $57 billion, and this would be associated with a 1% rise in mortality, or about 21,000 premature deaths. Thus the dollar losses necessary to induce one fatality equal approximately $2.7 million, (2,7000,00 = $57 billion/21,000). The lower follows from applying the elasticity to 1975 data, and inflating to current dollars.

[4] The coefficient from their logit analysis is −.21, while the probability of death in their sample is .28. Applying the formula to interpret logit coefficients found on p. 791 of Judge *et.al* (1988) gives 4.2% as an estimate of the change in the risk of death attributable to an additional dollar of hourly wage income.

work seems to suggest that the observed relation may be causal, the effect of income on mortality is significant only in the lower income categories. For workers with earning greater than the mean, the effect of income on mortality is insignificantly different from zero. Her other key findings include the following:

(1) Having earnings of less than $3000, instead of more than $9000, raises the probability of becoming disabled between ages 25 and 35 by 8 percentage points, and the probability of becoming disabled between the ages of 25 and 50 by about 28 percentage points.

(2) "The probability of death (during a six year period) for a 50 year-old man in the lowest earnings category with a high school education is more than twice as high as for his counterpart in the highest earnings category." (See p. 242)

To estimate the wage increases necessary to avert one fatality, first note that an increase in wages to between $6000 and $9000 from between $3,000 and $6,000 lowers mortality risk by 0.023 over the six years, or by about 0.0038 over one year, assuming that the incremental risk is uniformly distributed over the period.[5]

On average, raising annual wages by $3000 for each of 264 workers currently earning between $3000 and $6000 would avert one fatality per year, (.38% × 264 = 1). Converting these figures to current 1992 dollars, a rise in wages of $2.5 million is on average associated with one averted fatality annually.

Duleep (1989), found that income was significantly related to mortality throughout six income levels. In particular, mortality was higher for men with annual incomes between $36,000 and $47,000, (in 1992 dollars), than for similar men with incomes above $47,000. The effect of income on mortality was stronger in the lower income ranges than in the higher income ranges. Comparing the third and fourth income categories, her results imply that an increase in income of $6.5 million annually is associated with one less fatality per year. Her study covered 13,954 white married men aged 25 to 64 during the years 1973 to 1978. Comparing the relationship between mortality and income with the findings of Kitagawa and Hauser who used 1960 data, she found an apparent lack of improvement in the relative mortality experience of low income men.

In a more recent paper, Duleep (1991) investigates whether low income is associated with higher mortality because poorly paid workers face higher occupational risks to health and safety. Using information from the National Occupational Hazards Survey on the distribution of potential employee exposures to chemical substances and physical hazards, she found that each of six different measures of occupational hazard has only statistically insignificant effects on mortality. Furthermore, the effects of income on mortality are changed only slightly when occupational hazards are controlled for. Her results indicate that wage gains of $3.9 million in current dollars are associated with one less premature fatality.[6]

Thus it appears that the effect of low income on mortality is not a consequence of lower paid workers being exposed to greater on the job hazards.

Wolfson, *et al.*, in a forthcoming article in the Journal of Gerontology, show that income effects on mortality of 500,000 older Canadian males exist even between the highest and second highest income quintiles. Since all Canadians had access to public health care during the sample period, it appears that access to medical care is not the key mechanism linking income to mortality. Wolfson *et al.*, show that the mortality risk of the married males retiring at age 65 with incomes in the 95th percentile is only 0.86 of the risk for the similar males with median average earnings. Given that the risk of death by age 70 is about 0.15 for married males with median income, a relative risk of 0.86 implies that an increase in income from the median to the 95th percentile is associated with declines in mortality of about 2%. To express this in terms of dollars per life, note that an increase in income from the median to the 95th percentile is about 25,000 Canadian dollars annually. Converting to U.S. dollars, and adjusting for inflation, this amounts to $24,000 1991 U.S. dollars. An increase in incomes by this amount for married workers close to 65 would decrease mortality by two percentage points. Thus increases of $24,000 per year for each of 50 such workers earning the median income would be expected to avert one fatality during the years when these workers are age 65 to 70. Increase in income of $24,000 per year for each of 250 workers ($6 million in total) would thus be expected to avert one fatality per year, assuming for simplicity that the fatalities are uniformly distributed over the years in question. Wolfson concludes that "the results cast doubt on the primacy of causal explanations such as reverse causality and health selection".

The National Institutes of Health of the U.S. Department of Health and Human Services has studied extensively the relation between mortality and various demographic, social and economic factors.[7] They report that higher incomes are associated with lower mortality in the vast majority of the many cohorts that they considered. Using a follow-up study of one million Americans over the 3 years from 1979 to 1981, they calculate simple correlations between mortality and family income, for particular age, sex, and race cohorts. Their study does not use multiple regression techniques that might control for reversal causality by including important variables such as prior health status. They also do not control for education while considering the effects of income on mortality. They find, for example, that the mortality rates for white males aged 25–34 with family incomes of between $5,000 and $9,999 annually were 28% higher than for all whites in this age group. For white males aged 25–34 with family incomes between $25,000 and $49,999, mortality rates were only 57% as high as for all white males in this age group. Similarly, for black females aged 55–64 with family incomes of $5,000 to $9,999 females in this age group. For black females aged from 55–64 with family incomes of $5,000 to $9,999 annually, mortality rates were 8% higher than for all black females in this age group. For black females aged from 55–64 with family incomes of $25,000 to $49,999 annually, mortality rates were 46% as high as for all black females in this age group. Similar patterns between family income and mortality seem to exist for most cohorts.

These data permit a rough estimate of the income gains necessary to avert one death. Focusing on white males age 35–44, increasing income from the $15,000 to $19,999 category to the $20,000 to $24,999 category lowers mortality rates over the three years from .45% to .38%. Thus the decline in mortality rates is about 0.023% per year. Shifting 10,000 families from the low income category to the

[5] See Table 1, column 5 in Duleep (1988).

[6] This estimate is derived by considering the effect of a change in income from the category of $3,000–$5,999 to the category $6,000–$8,999, as reported in the first column of Duleep's Table 1. Applying again the formula for interpreting logit coefficients found on p. 791 of Judge *et al.*, implies that the decline in the risk of mortality is 1.6% over the 6 years. Taking one sixth of this change as the annual effect implies that annual income gains of $1.1 million would be associated with one less mortality annually. Expressed in current dollars a gain of $3.8 million in workers income in this income range would reduce mortality by one.

[7] National Institutes of Health, A Mortality Study of One Million Persons: by Demographic Social, and Economic Factors: 1979–1981 Follow-up First Data Book, U.S. National Longitudinal Mortality Study, March 1988.

higher income category would save on average 2.3 adult male lives per year. Improvements in income sufficient to shift families from the $15,000 to $19,999 category to the $20,000 to $24,999 category would also lower the death rates for other family members. For girls aged less than 15 it would fall by .028%, for boys aged less than 15 it would decline by 0.028%, while for women aged 35–44 it would fall by 0.09%. Thus shifting 10,000 families all composed of two parents, one boy and one girl, would reduce mortality by 17 lives. Since improvements in income would range from $1 to $10,000 we take $5,000 as the midpoint. Thus income gains of $50,000,000 save 17 lives. Converting from 1980 dollars to current dollars using the Consumer Price Index gives $4.9 million per life. Although this estimate considers the relation between family income and family mortality, it does so only over a short period, (3 years), without controlling for education, or prior health status.

All these studies estimate imperfectly the effect of income on mortality. Since these studies, with the exception of the NIH report, consider only the association between wage earnings and mortality of the individual, (or of the worker), rather than the relation between family income and mortality of family members, they tend to underestimate the relationship between family income and mortality generally, including that of all family members.

SUMMARY OF SELECTED STUDIES ON INCOME AND HEALTH

Study	Data	Implicit income gains necessary to avert one death (millions)	Comments
Keeny (1990)	Used income and mortality correlations form Kitagawa and Hauser, (1960) data, and others.	$7.25	Cited in *UAW* v. *OSHA*.
Hadley (1982)	400 counties, family income and county-wide mortality rates.	$38	Aggregate cross-sectional study. Measurement error may exaggerate estimate.
Joint Economic Committee (1984)	Aggregate U.S. income, employment, mortality, and morbidity; 1950–1980.	$1.8 to 2.7	Reflects income loss from recession of 1974–75.
Anderson and Burkhauser (1985)	4878 male workers over 10 years, 1969–1979.	$1.9 (wages) $4.3 (other income)	Older workers aged 58–63. Measured effects of wages and of value of one's home on mortality.
Duleep (1986)	9618 white married male workers age 35–64 over 6 years, 1973–1978.	$2.6	Controls for prior disability, and educational attainment.
Duleep (1989)	13,954 white married male workers aged 25–64 over 6 years, 1973–1978.	$6.5	
Duleep (1991)	9618 white married male workers aged 35–64 over 6 years, 1973–1978.	$3.9	Same as above. Also corrects for exposure to occupational hazards.
Wolfson (1992)	500,000 Canadian workers, over 10–20 years.	$6	Investigates longevity rather than mortality. Finds income effects at highest quintiles of income.
National Institutes of Health (1988)	1,000,000 Americans, all ages, 1979–1981	$4.9	

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Additional Questions for Public Comment

General (Apply to Construction, Maritime & Agriculture)

1. Are there any exposures to substances considered in this rulemaking other than those identified by OSHA? If so, which activities? Which substances? How many employees are exposed? To what exposure levels? What controls are used?

2. Please submit any additional exposure monitoring data to supplement the data OSHA already has. If possible, the results should be accompanied by a

description of the activity and any controls used.

3. Is there information regarding laboratory analytical procedures which may be used in lieu of those suggested by OSHA to determine exposure to air contaminants?

4. Are there any circumstances in which the proposed changes in PELs would have significant impacts beyond those identified by OSHA?

5. What controls are available to reduce exposures below existing levels in addition to those identified by OSHA, and what are the estimated costs associated with these controls?

6. OSHA requests comments on all aspects of and approaches to the regulation of fibrous glass, including refractory ceramic fibers, and mineral wool. This includes the scientific evidence on nonmalignant respiratory disease, carcinogenicity, and irritant effects, feasibility data, and various approaches to regulation based on these factors.

7. OSHA's proposed final limit for silica is based on gravimetric measurement. The 1970 TLV for silica is measured in m.p.p.c.f. (millions of particles per cubic foot of air, based on impinger samples counted by light-field techniques). Should OSHA convert this sampling methodology to the more commonly utilized gravimetric measurement of mg/m^3, for the purpose of listing in the transitional table of the final rule on the effective date of the final rule? The advantages of using gravimetric measurement will occur sooner and OSHA does not foresee feasibility problems as this is the common practice.

8. Comments are requested on all aspects of the regulation of asphalt fumes including issues as to its respiratory effects and carcinogenicity, and other issues relating to its regulation.

9. A risk assessment for asphalt fumes is presented based on the Hanson study. There is scientific disagreement on whether the exposed employees were exposed significantly to coal tar pitch as well as asphalt as discussed in the preamble. Is this study appropriate for risk assessment? Is the quantitative risk assessment methodology used appropriate? Have there been any other quantitative risk assessments conducted for asphalt fumes? If so, OSHA requests all relevant information.

10. OSHA proposed that the PEL for asphalt fumes be measured and expressed as total particulate. Another view is that use of the "benzene-soluble fraction" is more appropriate. OSHA solicits comments on this issue.

11. OSHA is proposing effective dates of 6 months for compliance with any means of engineering, word practice controls, and personal protective equipment, and 4 years for engineering controls for longshoring and marine terminals. For agriculture the first date is 1 year and the second is 4 years. For shipyards and construction the dates are 3 months and 4 years based in part on the Advisory Committee recommendations. Are these dates appropriate? Should they be longer or shorter?

12. Based on the assumption that a 1 percent excess mortality rate among workers in the construction industry would result from non-compliance with the proposed PELs, OSHA has estimated that 8 to 13 extra deaths per year would occur without this rule (see PRIA, V–2). OSHA requests comment on this assessment methodology, especially data which confirm or deny the assumed excess mortality rate.

13. In assessing the number of illnesses that would occur in the absence of this regulation, OSHA has assumed that BLS illness estimates for this industry are under-reported by a factor of five (See PRIA, V–2B). OSHA requests comment on this estimate, especially data which confirm or deny the magnitude of any under-reporting in reported illness statistics.

14. OSHA preliminarily concludes that the proposed PELs are economically and technologically feasible for all sectors. OSHA requests comments on the technological and economic feasibility of attaining these PELs in industry sector where working conditions may be different from those in general industry. Commenters may address feasibility issues on an industry or sub-industry basis.

15. In order to assess the benefits associated with this rulemaking, OSHA has preliminarily projected that the proposed PELs will cause an 80 percent reduction in the incidence of occupationally related illnesses (see PRIA). OSHA requests comments about whether an eighty percent effectiveness rate is a reasonable estimate of the rule's efficacy.

Construction

1. In the process of estimating risk to workers, OSHA has estimated the extent of exposures in the construction industry as the number of full-time equivalent employees exposed (See PRIA, page VB–58). This is to say that the total risk of 100 workers exposed above the PEL full-time is equivalent to the risk to 1000 workers each exposed for one-tenth the time. OSHA requests comments on the validity of this assumption and on all other aspects of converting exposure estimates into risk estimates. Should strict linearity be assumed?

Maritime

1. OSHA has some data on what percentage of workers currently wear respirators. Additional data on what percentage of tank cleaners, welders and painters currently wear personal protective equipment, including respirators, and on what percentage of other occupational groups wear personal protective equipment, including respirators is requested.

2. The use of "real time" measurements may be useful in tank cleaning and other confined space entry situations. Identify methods or instruments used for making "real time" measurements, in addition to those identified by OSHA. How accurate are these methods? What costs are involved?

3. Ship repairing and shipbreaking reference the 1970 ACGIH TLVs. Ship building references OSHA's Z–1 Table. OSHA proposes that, as of the effective date of the standard, all of the previously mentioned subgroups within the maritime sector utilize the 1970 TLVs as transitional limits. This will facilitate conversion to the same levels for these groups and provide consistency. The Shipyard Employment Standards Advisory Committee generally supports this proposal. OSHA also solicits comments from other interested members of the public.

Agriculture

1. OSHA requests additional information concerning the controls and work practices presently used to reduce exposures to respirable dust during silo filling, grain dust during the filling of transport vehicles and respirable silica during the harvesting of grapes.

2. OSHA requests any information regarding exposure to pesticidal residue during cotton ginning or other post-harvest agricultural processing.

3. OSHA requests information concerning activities at large agricultural establishments which present exposure to hazardous substances not identified in OSHA's preliminary analysis.

4. In addition to those substances identified by OSHA, what other substances found in the Z Table for Agriculture are used in agriculture? During what operations or activities?

5. OSHA is proposing that agricultural workers be protected by all the same exposure limits as workers in other sectors since they face the same risks

when exposed. (The use of EPA-registered pesticides pursuant to the label is regulated by EPA.) OSHA intends, through non-mandatory appendices, special publications and/or outreach programs, to inform agricultural employers and employees of the more common substances for which there is exposure in agriculture and appropriate precautions to take. OSHA requests information on the best way to supply this information to the agricultural community and the substances for which this program would be most useful.

6. OSHA has proposed to set Permissible Exposure Limits for over 100 pesticides that are regulated under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). These PELs would only apply if labelling instruction were not followed or if unlabelled pesticides were used. OSHA requests comment on whether additional regulation of registered pesticides is necessary, given the penalties of FIFRA. Would PELs cause confusion in the agricultural sector as to which regulatory requirements would take precedence or would the additional information provided by the exposure limits tend to reduce the number of poisonings from pesticides?

C. History and Need for Revision of PELs in Construction

Construction work sites are complex and varied, and may create special problems for identifying and controlling hazards (Bertinuson and Weinstein, (1978) Occupational Hazards of Construction). Construction sites are temporary and change constantly as the work moves ahead and as new trades arrive on the site. Several trades may share the same work area, exposing each other to toxic substances. The work is usually rushed, increasing the chances that an accident might occur or that a harmful chemical exposure goes unnoticed. Construction workers change jobs often, and some work for a number of employers during the same year. All of these factors contribute to the complex array of activities and related exposures, increasing the potential risk of accident or illness.

According to the Bureau of Labor Statistics, in 1989 the rate of work-related injuries in construction was the highest of any sector at 14.2 per 100 full-time workers. The average rate for all sectors combined was 8.2 injuries per 100 full-time workers. Construction also leads the other sectors in lost workdays, a measure of severity of injuries.

A construction worker is at least four times as likely to die on the job as workers in general. Occupational fatalities occurred at the rate of 5.4 in the private sector in 1989, with the rate for construction being 22.4 per 100,000 full-time workers according to BLS data for 1989.

Rates of occupational illnesses reported to BLS in construction appear to be lower than other sectors. There are a number of reasons for this. One is specifically related to the nature of occupational illnesses, which are more difficult to recognize, often have a long latency between exposure and development of disease, and are more difficult to relate to the workplace than injuries. As a result, injuries and fatalities, which are easy to recognize, have a short latency period, and are often more easily related to the workplace, are much more likely to be reported than illnesses.

Exposure profiles for construction workers vary from jobsite to jobsite, and typically include exposures to multiple substances, some of which they may not be working with directly. Workers tend to be employed on certain jobs or with particular contractors for short periods of time.

The difficulties inherent in identifying occupational illnesses and the transient nature of both the worksite and the workforce are the two major reasons for lack of reporting of occupational illnesses in construction.

The above analysis applies to BLS data. Recognizing this, OSHA has made its best estimate of illnesses and deaths avoided in construction. These are 22,000 illnesses and 15 to 20 deaths avoided per year. See the regulatory analysis for the approach used.

Construction Standards

Standards relevant to construction, primarily safety standards, are found in 29 CFR part 1926. Section 1926.20 of subpart C—General Safety and Health Provisions, under paragraph (a), Contractor requirements states:

(1) Section 107 of the Act requires that it shall be a condition of each contract which is entered into under legislation subject to Reorganization Plan number 14 of 1950 (64 Stata. 1267), as defined in § 1926.12, and is for construction, alteration, and/or repair, including painting and decorating, that no contractor or subcontractor for any part of the contract work shall require any laborer or mechanic employed in the performance of the contract to work in surroundings or under working conditions which are unsanitary, hazardous, or dangerous to his health or safety."

The above regulation was first issued under the Construction Safety Act on April 17, 1971 at 36 FR 7340, and in 1971 became an occupational safety and health standard for all construction workers. (See pertinent Legal Authority.)

Section 1926.55 regulates exposure to gases, vapors, fumes, dusts, and mists, and references the "Threshold Limit Values of Airborne Contaminants for 1970" of the American Conference of Governmental Industrial Hygienists. These are the limits that currently exist and are enforced in construction. Much scientific knowledge in the area of health effects of toxic substances has been gained in the past twenty years. OSHA is proposing to update these limits. The levels that are being proposed in construction are those levels that are currently in effect in general industry. The objective of this rulemaking is to produce the same exposure limits across all sectors, resulting in the same level of protection for all workers.

In addition, the scope of many of the substance-specific health standards found in 29 CFR part 1910 includes coverage of the construction sector. Lead, 1910.1025, and coke oven emissions are the only substance-specific standards which specify exposure limits that do not cover construction. However, lead is covered in construction to the degree that the 1970 TLV for lead is 200 $\mu g/m^3$. And while the standard for coke oven emissions does not apply to construction, the coal tar pitch volatile TLV of 200 $\mu g/m^3$ does apply.

OSHA is proposing, as part of this current rulemaking, a PEL of 50 $\mu g/m^3$ for lead in construction. This is the level that has existed in general industry since 1976; this is also the level effective in the maritime sector. The ancillary provisions (e.g. medical surveillance, exposure monitoring) are outside the scope of this rulemaking. However, OSHA is currently developing a proposal to address all of the other issues in a comprehensive standard addressing workplace exposure to lead in the construction industry.

As per its legal mandate to do so, OSHA has consulted with the Advisory Committee on Construction Safety and Health (ACCSH) in regard to this proposal. On June 20–1, 1990 ACCSH met and unanimously passed the following motion:

The Advisory Committee on Construction Safety and Health recommends to the Assistant Secretary of Occupational Safety and Health the adoption of the recommendations presented in the report of the workgroup on Permissible Exposure Limits for Construction and proceed with rulemaking to promulgate the D list (Table Z, Construction) as a 1926 construction standard and include in Table D provisions for mineral

wool, fibrous glass, asphalt fume, lead at 50 micrograms per cubic meter and cotton dust at 500 micrograms per cubic meter.

D. History and Need for Establishing PELs in Maritime

The Department of Labor has had some authority for many years for the maritime industry under the Longshore and Harbor Workers Compensation Act (33 U.S.C. 901 et seq.). Specific authority was granted prior to the OSH Act under Public Law 89–742, August 22, 1958, 33 U.S.C. 941 for the Secretary of Labor to issue regulations to protect the health and safety of longshoremen, marine terminal workers, ship repairers, shipbuilders and shipbreakers (See also 33 U.S.C. 902). Pursuant to section 4(b)(2) of the OSH Act, standards issued pursuant to 33 U.S.C. 941 became OSH standards on May 29, 1971 at 36 FR 10466.

At that time the Shipyard standards were in three Parts of 29 CFR part 1915 for ship repairing, part 1916 for shipbuilding and part 1917 for shipbreaking. These were recodified with the same basic organization on October 19, 1977 at 37 FR 22458.

On April 20, 1982 at 47 FR 16984, parts 1915, 1916 and 1917 were consolidated into a new part 1915, Shipyards, covering shipbuilding, ship repairing and shipbreaking. As a consequence of their history, the permissible exposure limits applicable to the new part 1915, Shipyards are complex. The shipbreaking and ship repairing subsectors and the use of toxic solvents and removers in the shipbuilding and ship repair subsectors are covered by the 1970 TLVs of the ACGIH. See §§ 1915.5, 1915.11, 1915.12, 1915.32 and 1915.33. For shipbuilding operations not involving toxic solvents and removers, the 1971 OSHA PELs apply. No specific exposure limits are specified for shipbuilding outside those 2 circumstances. When no standard is specified for a subsector, and there is a standard for general industry, then the general industry standard applies to the subsector. See 29 CFR 1910.5(c). The OSHA general industry standard for air contaminants are the Z Tables in 29 CFR 1910.1000. OSHA did not make the updated (1989) PELs applicable to maritime awaiting the completion of this rulemaking. See 29 CFR 1910.1000(f)(3)(ii). In addition, a number of OSHA single-substance standards (1910.1001–1048) are applicable to the maritime sectors. See the individual standards and 29 CFR 1910.19.

Clearly it is confusing both to the shipyard industry, its employees and for OSHA enforcement for slightly different standards to apply to different operations in the same work place. (The 1970 TLVs and the 1968 PELs do differ for some substances, though usually not by significant amounts.) Consequently, for shipyards, OSHA feels the existence of different standards for different subsectors should be eliminated as soon as possible. Accordingly, OSHA is proposing that the new PELs will take effect for all of shipyards on the effective date of the standard, to be achieved by any reasonable means of compliance. The preference for engineering controls in the transition period will apply to the 1970 TLVs for all subsectors of shipyards.

Pursuant to the Longshoremen and Harbor Worker Compensation Acts 1958 amendments (33 U.S.C. 941), OSHA in 1960 issued regulations protecting longshore employees (25 FR 1569). These regulations also covered marine terminal employees. They were adopted as OSHA standards on May 29, 1971 at 36 FR 10466. They were recodified as 29 CFR part 1918 on May 19, 1977 at 37 FR 22530.

On July 5, 1983 (48 FR 30886), OSHA issued a final standard specifically covering marine terminals separately from longshoring. The Marine Terminals Standard was designated 29 CFR part 1917. (It should be recalled that the earlier part 1917 covering shipbreaking had been recodified as part of part 1915—Shipyards.) The Marine Terminal Standard basically provides that employees not be exposed to air contaminants over the limit set in the 1971 Z Tables of CFR 1910.1000. See §§ 1917.2(p), 1917.22, 23, 25.

Longshoring operations continue to be regulated by 29 CFR part 1918. OSHA has consistently interpreted that the air contaminant exposure limits set forth in 1910.1000 are applicable pursuant to § 1910.5(c) to longshoring because no quantitative exposure limits are set forth for most air contaminants. Section 1918.93 only sets forth specific numerical limits for carbon monoxide. It is OSHA's intention to clarify in this rulemaking that all the air contaminant exposure limits in the 1971 Z Tables in 1910.1000 are applicable and will remain applicable to longshoring during the transition period.

OSHA is proposing to update the air contaminants standards used in the maritime sector. Ship repairing and shipbreaking are currently covered by the 1970 TLVs, and shipbuilding, marine terminals and longshoring are covered by the 1971 OSHA PELs-subpart Z. The product of this rulemaking will be one table used by all subsectors of Maritime. Thus, this proposed rulemaking will produce consistency across all subsectors of the maritime sector.

This proposal for maritime is necessary for several reasons. First, the existing limits are out-of-date and not sufficiently protective for 376 substances. Second, workers are regularly exposed to a variety of toxic substances in this sector. Third, current standards are inconsistent among the various subsectors of maritime leading to confusion. The Shipyard Advisory Committee has discussed and is supportive of these changes.

E. History and Need for Establishing PELs in Agriculture

Historically, OSHA has developed and applied few health and safety regulations in the agricultural sector. The OSH Act is intended to apply to agricultural workers, though Congress has exempted the employers of farms with ten or fewer employees by appropriations rider since 1977. See the discussion in the Legal Authority section.

When OSHA was formed, the Agency was given two years to adopt "consensus" standards, under section 6(a) of the OSH Act, so that it would have some regulations with which to start enforcement procedures. The issuance of very few standards for "agricultural operations" appears to have been based on the view that, since the 1910 consensus standards of the time were primarily ANSI and NFPA standards, and these standards had been developed by the industrial sector, this raised the question of whether these could be considered "consensus" standards when adopted to fit agricultural situations.

Part 1928 (source: 40 FR 18257, Apr. 25, 1975) of the CFR refers to those standards currently applicable in agriculture. Subpart B, 1928.21 specifically cites those standards in 29 CFR part 1910 (General Industry) which apply to agricultural operations:

(1) Temporary labor camps—§ 1910.142
(2) Storage and handling of anhydrous ammonia—§ 1910.111(a) and (b);
(3) Pulpwood logging—§ 1910.266;
(4) Slow-moving vehicles—§ 1910.145;
(5) Hazard communication—§ 1910.1200

In addition, standards which apply to Roll-Over Protective Structures in agriculture are found in subpart C of 29 CFR part 1928. Part 1928 subpart D addresses the use of guarding for agricultural equipment (Safety for Agricultural Equipment). Field Sanitation, 1928.110, is found in Subpart I—General Environmental Controls. 29 CFR 1928.21(b) specifically states that "(e)xcept to the extent specified in paragraph (a) of this section, the

standards contained in subparts B through T and subpart Z of 1910 of this title do not apply in agricultural operations."

There are no Permissible Exposure Limits (PELs) that apply to agriculture. OSHA states in its Air Contaminants Final Rule, (Federal Register, Vol. 54, No. 12, p. 2383): "In the future OSHA will consider, based on relevance, priorities and administrative resources, whether or not it is appropriate to consider coverage for agriculture." The Agency has considered this issue and concluded that it is appropriate to provide the same level of protection from toxic substances to workers employed in the agricultural sector as workers employed in other sectors, e.g. general industry or construction.

For purposes of analysis of agriculture for this standard, OSHA has relied primarily on the classifications in OMB's Standard Industrial Classification (SIC) manual. The Standard Industrial Classification manual assigns agriculture the following SIC codes: Major Group 01, Agricultural Production—Crops; Major Group 02, Agricultural Production—Livestock; Major Group 07, Agricultural Services. The SIC manual defines agriculture as including an "establishment (e.g. farms, ranches, dairies, greenhouses, nurseries, orchards, hatcheries) primarily engaged in the production of crops, plants, vines or trees (excluding forestry operations); and the keeping, grazing, or feeding of livestock for the sale of livestock * * * also establishments primarily engaged in the production of mushrooms, bulbs, * * * seeds and in the growing of hydroponic crops." Nurseries and greenhouses are considered to be agricultural establishments only if the products they sell are grown primarily at the establishment. Otherwise these establishments are considered retail trade establishments. Forestry (08), (but not logging) comes under agriculture and is being considered in this rulemaking. Fishing (09) is considered to fall under general industry. However, fishing may wish to submit data, which OSHA will then consider.

OSHA has studied Major Group 07, Agricultural Services, but parts of that sector may be covered by the General Industry standard. Any subsector which believes it is covered but has not been studied, should submit appropriate comments. OSHA's intention is that after after this rule is completed, that all employees under OSHA's jurisdiction will be covered. If any sector believes it has feasibility matters it wishes to bring to OSHA's attention, which OSHA has not considered, it should submit this information.

Agriculture is one of the most hazardous of U.S. industries, with 1,300 deaths and 120,000 worker injuries in 1989, according to National Safety Council figures. These estimates indicate that agriculture has the second highest death rates of any major industry division, second only to mining, but greater than construction.

In terms of illnesses, the illness incidence rate among agricultural workers for 1989, the latest year reported by the Bureau of Labor Statistics, was 45.5 per 10,000 full-time workers, higher than mining and construction.

In addition, these statistics are believed to underestimate agricultural morbidity by a large margin because OSHA does not require recordkeeping and reporting by farms with 10 or fewer employees and because workers' compensation laws in most states do not cover farms with few employees. As a consequence, the reporting of agricultural injuries and illnesses on a national level is very poor. For illnesses that are the result of agricultural exposures, the statistics are further understated due to the problems inherent in identifying and reporting occupational illnesses.

The single most hazardous group of substances to which agricultural workers are exposed is pesticides. In the United States, any chemical that is to be used for pesticidal purposes must be registered in accordance with EPA, and in particular with Federal Insecticide, Fungicide and Rodenticide Act (FIFRA), requirements. In addition to requiring registration, FIFRA has the authority to regulate conditions of use, including worker safety and health, for pesticidal substances. EPA first regulated pesticide worker safety in 1974, when it promulgated a Worker Safety standard (still being enforced) that applies only to workers performing hand labor at agricultural establishments. EPA has recently proposed to update and expand its worker protection standard for agricultural pesticides. The proposed standard also would apply to pesticide users at farm sites, nurseries, greenhouses, and forest areas used for the commercial production of wood fiber and timber products. EPA has left open the question of the size-class of agricultural establishments to be covered by these regulations and has solicited input on this issue. In contrast to the 1974 rule, the proposed standard would apply to workers engaged in all pesticide handling activities (e.g. mixing, loading, and application) conducted at agricultural establishments. The FIFRA proposal addresses the following areas: specific information to be provided to workers; training and labeling requirements; selection of personal protective equipment; avoidance of exposure to non-applicators; minimum reentry times; availability of water, soap, and towels; and biological monitoring of cholinesterase levels for pesticide handlers exposed to certain organophosphates for specified durations.

Once FIFRA's Worker Safety standard becomes final, EPA will have exercised its jurisdiction authorized under FIFRA, regulation of many safety and health issues related to worker exposure to use of labelled pesticides. In consequence, OSHA will not, in many cases, have authority to regulate pursuant to section 4(b)(1) of the OSHA Act when a labelled pesticide is used according to its label. See the discussion under Legal Authority.

In 1980, about 600 active ingredients (or groups of active ingredients) were registered under FIFRA (54 FR 7740). Since 1980, EPA has issued 194 registration standards (documents describing EPA's regulatory position and the Agency's underlying rationale for regulating particular pesticides); these 194 standards have addressed approximately 350 chemical substances used either as active or inert pesticide ingredients.

Some of the pesticides currently registered under FIFRA appear on OSHA's Z-Table. For the sake of completeness, and for use as a reference, a short discussion of these substances is included in the appropriate health effects section of the preamble. Where the substance has a FIFRA-registered application, that information is included. In many cases there are non-pesticidal uses of these substances that result in occupational exposures. Again, see the discussion under Legal Authority for OSHA jurisdictional issues.

A number of the substances on OSHA's Z Table, Agriculture are fertilizers. These are among the substances that are covered by this rulemaking. The following are OSHA-regulated substances used as fertilizers in agriculture:

Chemical	CAS No.
Ammonia	7664-41-7
Borax (borates, tetra, decahydrate)	1303-96-4
Calcium cyanamide	156-62-7
Diatomaceous earth (amorphous silicates)	617490-53-2
Dolomite (calcium carbonate)	1317-65-3
Furfural	98-01-1

Chemical	CAS No.
Gypsum	3397-24-5
Hydrochloric acid (hydrogen chloride)	7647-01-0
Limestone (calcium carbonate)	1317-65-3
Nitric acid	7647-01-0
Perlite	N/A
Phosphoric acid	7664-38-2
Sulfuric acid	7664-93-9

Source: Farm Chemicals Handbook.

Farm workers use many different cleansers and disinfectants that are regulated by OSHA in its Z–1–A Table. Among the most important are: Caustic soda (sodium hydroxide), used to clean milking equipment and containers; and ammonium hydroxide, used to neutralize formaldehyde, another substance commonly used.

Farm workers regularly use solvents, paints, acids, coatings, adhesives and other materials that contain toxic substances. They are thus exposed to heavy metals (e.g., in paints), solvents (when cleaning machinery and tools), fuels (when fueling farm vehicles), resins (in coatings and adhesives), welding fumes (when welding), and toxic gases (from manure pits), and a host of other contaminants. In addition, these workers are routinely exposed to grain dust, a major cause of pulmonary dysfunction in farm workers.

The agricultural work environment is often quite dusty. Agricultural dusts in the farm environment may come from any animal or plant source associated either specifically with agricultural production or in general with the rural environment. Agricultural dusts vary with the season, climate, weather conditions, and type of agricultural operation (Mutel et al., 1986 as cited in: Donham, K., (1986) Hazardous Agents in Agricultural Dusts and Methods of Evaluation, Am J of Ind Med 10:205–220). They are typically complex, including a mixture of both inorganic and organic particles and perhaps aerosolized pesticides and chemical fertilizers (Mutel et al., as cited in: Donham, K., (1986) Hazardous Agents in Agricultural Dusts and Methods of Evaluation, Am J of Ind Med 10:205–220).

Various agricultural activities can result in exposure to dusts. Harvesting, transporting, or storing grain, milling grain, mixing feed or feeding animals, during various activities. Swine and poultry confinement buildings are sources of especially high exposures. Trucking or otherwise moving grain or hay, cleaning out livestock structures, storing grain, unloading the top of silage from nonairtight silos, and harvesting grain can create exposure to dusts (Darke et al., 1976; Donham et al., 1978, 1983; Lacey and Lacey, 1964; Mutel et al., 1986; as cited in: Donham, K, (1986) Hazardous Agents in Agricultural Dusts and Methods of Evaluation, Am J Ind Med, 10:205–220).

Potential for exposure to animal-related dusts is encountered during routine animal care, including feeding, cleaning animal quarters, sorting and handling animals, administering medications, milking dairy cows, or replacing their bedding (Donham et al., 1977, as cited in: Donham, K, (1986) Hazardous Agents in Agricultural Dusts and Methods of Evaluation, Am J Ind Med, 10:205–220). An even greater potential for significant exposure to dusts exists when generated in an enclosed structure, such as a barn, silo, or livestock confinement structure.

The Air Contaminants Final Rule (FR Vol 54, No. 12, Jan. 19, 1989) adopted 164 new PELs where none previously existed and 212 PELs that were more protective than the old. There were approximately 160 substances that had been in effect since the publication of the Z–Table and were not considered for change during this rulemaking because there was no difference between the old PEL (Z-1 Table value) and the 1987–88 TLV. (One of the criteria for consideration of updating of a particular PEL was that the 1987–8 ACGIH TLV be significantly different from the old OSHA PEL.)

The situation in agriculture, however, is very different. Because there have been no Z–Tables, and thus no PELs in effect in agriculture, the approximately 160 substances referenced in the earlier paragraph are being proposed for the first time in agriculture. In addition, there are approximately 50 substances which OSHA is not proposing to change for construction and maritime because OSHA determined in the general industry rulemaking that change was not appropriate at that time. These are being proposed for agriculture also. There is the same need for health protection from the toxic effects of these chemicals in agricultural applications as in other sectors. Health write-ups are provided.

In addition, there are ten substances for which OSHA has developed comprehensive substance-specific standards. These standards are not limited to a Permissible Exposure Limit, but typically have other provisions (e.g. medical surveillance, exposure monitoring requirements, medical removal protection) which further reduce the risk of illness to workers exposed to these substances. None of the substance-specific standards has been applied to the agricultural sector. OSHA is attempting to correct this situation during this rulemaking. However, since this rulemaking is covering only the Permissible Exposure Limits and not other provisions, only the PELs for these substances will be proposed. The same PELs that have been previously established in other sectors are being proposed in agriculture for the following substances: Asbestos, vinyl chloride, inorganic arsenic, lead, benzene, cotton dust, 1,2'-dibromo-3-chloropropane (DBCP), acrylonitrile, ethylene oxide and formaldehyde.

It is important to note that farming operations that do not maintain a temporary labor camp and that employ 10 or fewer employees are exempt from OSHA regulations. In addition, immediate family members of farm employers are not regarded as employees when making the determination as to number. These situations are exempted by appropriations rider. See the discussion under Legal Authority.

Small farms constitute the majority (96%, see Preliminary Regulatory Impact Analysis) of American farms. Addressing the injuries, illnesses and fatalities which have become part of daily life on these farms is an area OSHA feels it does have a responsibility, albeit not a regulatory one. The focus of OSHA's current effort is to work with and through the U.S. Department of Agriculture to reach out to states, land grant universities, Farm Bureaus, Granges, 4–H Clubs, and others such as the National Safety Council and National Farmers Union in furthering education and awareness in agricultural health and safety. OSHA is also collaborating closely with NIOSH to facilitate information collection and dissemination to the benefit of both agencies and ultimately the agricultural community. Although a fair amount of groundwork has already been laid, OSHA is still in the early stages of its agricultural outreach initiative.

TABLE I.F.—SUBSTANCES INCLUDED IN THE UPDATE OF PELs FOR CONSTRUCTION, MARITIME, AND AGRICULTURE

Substance	CAS No.	Substances for which limits are being proposed in construction	Substances for which limits are being proposed in maritime			Substances for which limits are being proposed in agriculture	Substances for which limits are being proposed in general industry
			Ship repairing and ship breaking	Ship building	Marine terminals and longshoring		
Abate (see Temephos)	3383-96-8						
Acetaldehyde	75-07-0	X	X	X	X	X	
Acetic acid	64-19-7					X	
Acetic anhydride	108-24-7	X	X	X	X	X	
Acetone	67-64-1	X	X	X	X	X	
Acetonitrile	75-05-8	X	X	X	X	X	
Acetylene dichloride; see 1,2-Dichloroethylene.							
Acetylene tetrabromide	79-27-6					X	
Acetylsalicylic acid (Aspirin)	50-78-2	X	X	X	X	X	
Acrolein	107-02-8	X	X	X	X	X	
Acrylamide	79-06-1	X	X	X	X	X	
Acrylic acid	79-06-1	X	X	X	X	X	
Acrylonitrile; see 1910.1045	107-13-1					X	
Aldrin	309-00-2					X	
Allyl alcohol	107-18-6	X	X	X	X	X	
Allyl chloride	107-05-1	X	X	X	X	X	
Allyl glycidyl ether (AGE)	106-92-3	X	X	X	X	X	
Allyl propyl disulfide	2179-59-1	X	X	X	X	X	
alpha-Alumina Total dust	1344-28-1	X	X	X	X	X	
Respirable fraction						X	
Aluminum (as Al) Metal	7429-90-5						
Total dust						X	
Respirable fraction						X	
Pyro powders			X	X	X	X	
Aluminum (con't.) welding fumes			X	X	X	X	
Soluble salts		X	X	X	X	X	
Alkyls		X	X	X	X	X	
2-Aminoethanol; see Ethanolamine							
2-Aminopyridine	504-29-0					X	
Amitrole	61-82-5	X	X	X	X	X	
Ammonia	7664-41-7	X	X	X	X	X	
Ammonium chloride fume	12125-02-9	X	X	X	X	X	
Ammonium sulfamate	7773-06-0						
Total dust		X	X	X	X	X	
Respirable fraction						X	
n-Amyl acetate	628-63-7					X	
sec-Amyl acetate	626-38-0					X	
Aniline and homologs	62-53-3	X	X	X	X	X	
Anisidine (o-, p-isomers)	29191-52-4					X	
Antimony and compounds (as Sb)	7440-36					X	
ANTU (alpha naphthylthiourea)	86-88-4					X	
Arsenic, inorganic compounds (as As); see 1910.1018.	7440-38-2					X	
Arsenic, organic compounds (as As)	7440-38-2					X	
Arsine	7784-42-1					X	
Asbestos; see 1910.1001; 1910.1101	(1)					X	
Asphalt (petroleum) fumes	8052-42-4	X	X	X	X	X	X
Atrazine	1912-24-9	X	X	X	X	X	
Azinphos-methyl	86-50-0					X	
Barium, soluble compounds (as Ba)	7440-39-3					X	
Barium sulfate	7727-43-7						
Total dust (as Ba)		X	X	X	X	X	
Respirable fraction						X	
Benomyl	17804-35-2						
Total dust		X	X	X	X	X	
Respirable fraction						X	
Benzene; see 1910.1028	71-43-2					X	
p-Benzoquinone; see Quinone							
Benzo(a)pyrene; see coal tar pitch volatiles							
Benzoyl peroxide	94-36-0					X	
Benzyl chloride	100-44-7					X	
Beryllium and beryllium compounds (as Be).	7440-41-7					X	
Biphenyl; see Diphenyl							
Bismuth telluride, undoped	1304-82-1						
Total dust						X	
Respirable fraction						X	
Bismuth telluride, Se-doped		X	X	X	X	X	
Borates, tetra, sodium salts							
Anhydrous	1330-43-4	X	X	X	X	X	
Decahydrate	1303-96-4	X	X	X	X	X	
Pentahydrate	12179-04-3	X	X	X	X	X	
Boron oxide	1303-86-2						

TABLE I.F.—SUBSTANCES INCLUDED IN THE UPDATE OF PELS FOR CONSTRUCTION, MARITIME, AND AGRICULTURE—Continued

Substance	CAS No.	Substances for which limits are being proposed in construction	Substances for which limits are being proposed in maritime			Substances for which limits are being proposed in agriculture	Substances for which limits are being proposed in general industry
			Ship repairing and ship breaking	Ship building	Marine terminals and longshoring		
Total dust		X	X	X	X	X	
Boron tribromide	10294-33-4	X	X	X	X	X	
Boron trifluoride	7637-07-2					X	
Bromacil	314-40-9	X	X	X	X	X	
Bromine	7726-95-6	X	X	X	X	X	
Bromine pentafluoride	7783-30-2			X	X	X	
Bromoform	75-25-2					X	
Butane	106-97-8	X	X	X	X	X	
Butanethiol; see Butyl mercaptan							
2-Butanone (Methyl ethyl ketone)	78-93-3	X	X	X	X	X	
2-Butoxyethanol	111-76-2	X	X	X	X	X	
n-Butyl-acetate	123-86-4	X	X	X	X	X	
sec-Butyl acetate	105-46-4					X	
tert-Butyl acetate	540-88-5					X	
Butyl acrylate	141-32-2	X	X	X	X	X	
n-Butyl alcohol	71-36-3	X	X	X	X	X	
sec-Butyl alcohol	78-92-2	X	X	X	X	X	
tert-Butyl alcohol	75-65-0	X	X	X	X	X	
Butylamine	109-73-9					X	
tert-Butyl chromate (as CrO_3)	1189-85-1					X	
n-Butyl glycidyl ether (BGE)	2426-08-6	X	X	X	X	X	
n-Butyl lactate	138-22-7	X	X	X	X	X	
Butyl mercaptan	109-79-5			X	X	X	
o-sec-Butylphenol	89-72-5	X	X	X	X	X	
p-tert-Butyltoluene	98-51-1	X	X	X	X	X	
Calcium carbonate	1317-65-3						
Total dust						X	
Respirable fraction						X	
Calcium cyanamide	156-62-7	X	X	X	X	X	
Calcium hydroxide	1305-62-0	X	X	X	X	X	
Calcium oxide	1305-78-8					X	
Calcium silicate	1344-95-2						
Total dust						X	
Respirable fraction						X	
Calcium sulfate	7778-18-9						
Total dust						X	
Respirable fraction						X	
Camphor, synthetic	76-22-2					X	
Caprolactam	105-60-2						
Dust		X	X	X	X	X	
Vapor		X	X	X	X	X	
Captafol (Difolatan)	2425-06-1	X	X	X	X	X	
Captan	133-06-2	X	X	X	X	X	
Carbaryl (Sevin)	63-25-2					X	
Carbofuran (Furadan)	1563-66-2	X	X	X	X	X	
Carbon black	1333-86-4					X	
Carbon dioxide	124-38-9	X	X	X	X	X	
Carbon disulfide	75-15-0	X	X	X	X	X	
Carbon monoxide	630-08-0	X	X	X	X	X	
Carbon tetrabromide	558-13-4	X	X	X	X	X	
Carbon tetrachloride	56-23-5	X	X	X	X	X	
Carbonyl fluoride	353-50-4	X	X	X	X	X	
Catechol (Pyrocatechol)	120-80-9	X	X	X	X	X	
Cellulose	9004-34-6					X	
Total dust						X	
Respirable fraction						X	
Cesium hydroxide	21351-79-1	X	X	X	X	X	
Chlordane	57-74-9					X	
Chlorinated camphene	8001-35-2	X	X	X	X	X	
Chlorinated diphenyl oxide	55720-99-5					X	
Chlorine	7782-50-5	X	X	X	X	X	
Chlorine dioxide	10049-04-4	X	X	X	X	X	
Chlorine trifluoride	7790-91-2					X	
Chloroacetaldehyde	107-20-0					X	
a-Chloroacetophenone (Phenacyl chloride).	532-27-4					X	
Chloroacetyl chloride	79-04-9	X	X	X	X	X	
Chlorobenzene	108-90-7					X	
o-Chlorobenzylidene malononitrile	2698-41-1					X	
Chlorobromomethane	74-97-5					X	
2-Chloro-1,3-butadiene; see beta-Chloroprene							
Chlorodifluoromethane	74-45-6	X	X	X	X	X	
Chlorodiphenyl (42% Chlorine) (PCB)	53469-21-9					X	
Chlorodiphenyl (54% Chlorine) (PCB)	11097-69-1					X	

TABLE I.F.—SUBSTANCES INCLUDED IN THE UPDATE OF PELS FOR CONSTRUCTION, MARITIME, AND AGRICULTURE—Continued

Substance	CAS No.	Substances for which limits are being proposed in construction	Substances for which limits are being proposed in maritime			Substances for which limits are being proposed in agriculture	Substances for which limits are being proposed in general industry
			Ship repairing and ship breaking	Ship building	Marine terminals and longshoring		
1-Chloro,2,3-epoxypropane; see Epichlorohydrin							
2-Chloroethanol; see Ethylene chlorohydrin							
Chloroform (Trichloromethane)	67-66-3	X	X	X	X	X	
1-Chloro-1-nitropropane	600-25-9	X	X	X	X	X	
Chloropentafluoroethane	76-15-3	X	X	X	X	X	
Chloropicrin	76-06-2					X	
beta-Chloroprene	126-99-8	X	X	X	X	X	
o-Chlorostyrene	2039-87-4	X	X	X	X	X	
o-Chlorotoluene	95-49-8	X	X	X	X	X	
2-Chloro-6-trichloromethyl pyridine	1929-82-4						
Total dust						X	
Respirable fraction						X	
Chlorpyrifos	2921-88-2	X	X	X	X	X	
Chromic acid and chromate (as CrO_3)	(1)					X	
Chromium (II) compounds (as Cr)	7440-47-3					X	
Chromium (III) compounds (as Cr)	7440-47-3					X	
Chromium metal	7440-47-3					X	
Chrysene; see Coal tar pitch volatiles							
Clopidol	2971-90-6						
Total dust						X	
Respirable fraction						X	
Coal dust (less than 5% SiO_2), Respirable quartz fraction.		X	X	X	X	X	
Coal dust (greater than or equal to 5% SiO_2), Respirable quartz fraction.		X	X	X	X	X	
Coal tar pitch volatiles (benzene soluble fraction), anthracene, BaP, phenanthrene, acridine, chrysene, pyrene.	65966-93-2					X	
Cobalt metal, dust, and fume (as Co)	7440-48-4	X	X	X	X	X	
Cobalt carbonyl (as Co)	10210-68-1	X	X	X	X	X	
Cobalt hydrocarbonyl (as Co)	16842-03-8	X	X	X	X	X	
Coke oven emissions; see 1910.1029		X					
Copper	7440-50-8						
Fume (as Cu)					X		
Dusts and mists (as Cu)						X	
Cotton dust		X	X	X	X	X	
Crag herbicide	136-78-7						
(Sesone) Total dust		X	X	X	X	X	
Respirable fraction						X	
Cresol, all isomers	1319-77-3					X	
Crotonaldehyde	123-73-9					X	
	4170-30-3						
Crufomate	299-86-5	X	X	X	X	X	
Cumene	98-82-8					X	
Cyanamide	420-04-2	X	X	X	X	X	
Cyanides (as Cn)	(1)					X	
Cyanogen	460-19-5			X	X	X	
Cyanogen chloride	506-77-4	X	X	X	X	X	
Cyclohexane	110-82-7					X	
Cyclohexanol	108-93-0	X	X	X	X	X	
Cyclohexanone	108-94-1	X	X	X	X	X	
Cyclohexene	110-83-8					X	
Cyclohexylamine	108-91-8	X	X	X	X	X	
Cyclonite (RDX)	121-82-4			X	X	X	
Cyclopentadiene	542-92-7					X	
Cyclopentane	287-92-3	X	X	X	X	X	
Cyhexatin	13121-70-5	X	X	X	X	X	
2,4-D (Dichlorophenoxyacetic acid)	94-75-7					X	
Decaborane	17702-41-9	X	X	X	X	X	
Demeton (Systox)	8065-48-3					X	
Diacetone alcohol (4-Hydroxy-4-methyl-2-pentanone).	123-42-2					X	
1,2-Diaminoethane; see Ethylenediamine.							
Diazinon	333-41-5	X	X	X	X	X	
Diazomethane	334-88-3					X	
Diborane	19287-45-7					X	
1,2-Dibromo-3-chloropropane; see 1910.1044.	96-12-8		X	X	X	X	
2-N-Dibutylaminoethanol	102-81-8	X	X	X	X	X	
Dibutyl phosphate	107-66-4	X	X	X	X	X	

TABLE I.F.—SUBSTANCES INCLUDED IN THE UPDATE OF PELs FOR CONSTRUCTION, MARITIME, AND AGRICULTURE—Continued

Substance	CAS No.	Substances for which limits are being proposed in construction	Substances for which limits are being proposed in maritime			Substances for which limits are being proposed in agriculture	Substances for which limits are being proposed in general industry
			Ship repairing and ship breaking	Ship building	Marine terminals and longshoring		
Dibutyl phthalate	84-74-2					X	
Dichloroacetylene	7572-29-4			X	X	X	
o-Dichlorobenzene	95-50-1					X	
p-Dichlorobenzene	106-46-7	X	X	X	X	X	
Dichlorodifluoromethane	75-71-8					X	
1,3-Dichloro-5,5-dimethyl hydantoin	118-52-5	X	X	X	X	X	
Dichlorodiphenyltrichloroethane (DDT)						X	
1,2-Dichloroethane see Ethylene dichloride							
1,2-Dichloroethylene	540-59-0					X	
Dichloroethyl ether	111-44-4	X	X	X	X	X	
Dichloromonofluoromethane	75-43-4	X	X	X	X	X	
1,1-Dichloro-1-nitroethane	594-72-9	X	X	X	X	X	
1,2-Dichloropropane; see Propylene dichloride							
1,3-Dichloropropene	542-75-6	X	X	X	X	X	
2,2-Dichloropropionic acid	75-99-0	X	X	X	X	X	
Dichlorotetrafluoroethane	76-14-2					X	
Dichlorvos (DDVP)	62-73-7					X	
Dicrotophos	141-66-2	X	X	X	X	X	
Dicyclopentadiene	77-73-6	X	X	X	X	X	
Dicyclopentadienyl iron	102-54-5						
Total dust		X	X	X	X	X	
Respirable fraction						X	
Dieldrin	60-57-0					X	
Diethanolamine	111-42-2	X	X	X	X	X	
Diethylamine	109-89-7	X	X	X	X	X	
2-Diethylaminoethanol	100-37-8					X	
Diethylene triamine	111-40-0	X	X	X	X	X	
Diethyl ether; see Ethyl ether							
Diethyl ketone	96-22-0	X	X	X	X	X	
Diethyl phthalate	84-66-2	X	X	X	X	X	
Difluorodibromomethane	75-61-6					X	
Diglycidyl ether (DGE)	2238-07-5	X	X	X	X	X	
Dihydroxybenzene; see Hydroquinone							
Diisobutyl ketone	108-83-8	X	X	X	X	X	
Diisopropylamine	108-18-9					X	
Dimethoxymethane; see Methylal							
Dimethyl acetamide						X	
Dimethylamine	124-40-3					X	
Dimethylaminobenzene; see Xylidine							
Dimethylaniline (N,N-Dimethylaniline)	121-69-7	X	X	X	X	X	
Dimethylbenzene; see Xylene							
Dimethyl-1,2-dibromo-2,2-dichloroethyl phosphate.	300-76-5	X	X	X	X	X	
Dimethylformamide	68-12-2					X	
2,6-Dimethyl-4-heptanone; see Diisobutyl ketone.							
1,1-Dimethylhydrazine	57-14-7					X	
Dimethylphthalate	131-11-3					X	
Dimethyl sulfate	77-78-1	X	X	X	X	X	
Dinitolmide (3,5-Dinitro-o-toluamide)	148-01-6	X	X	X	X	X	
Dinitrobenzene (alpha, meta, para isomers).	528-29-0					X	
	99-65-0						
	100-25-4						
Dinitro-o-cresol	534-52-1					X	
Dinitrotoluene	25321-14-6					X	
Dioxane (Diethylene dioxide)	123-91-1	X	X	X	X	X	
Dioxathion (Delnav)	78-34-2	X	X	X	X	X	
Diphenyl (Biphenyl)	92-52-4					X	
Diphenylamine	122-39-4			X	X	X	
Diphenylmethane diisocyanate; see Methylene bisphenyl isocyanate							
Dipropylene glycol methyl ether	34590-94-8	X	X	X	X	X	
Dipropyl ketone	123-19-3	X	X	X	X	X	
Diquat	85-00-7	X	X	X	X	X	
Di-sec octyl phthalate (Di-2-ethylhexylphthalate).	117-81-7	X	X	X	X	X	
Disulfiram	97-77-8	X	X	X	X	X	
Disulfoton	298-04-4	X	X	X	X	X	
2,6-Di-tert-butyl-p-cresol	1128-37-0	X	X	X	X	X	
Diuron	330-54-1	X	X	X	X	X	
Divinyl benzene	1321-74-0	X	X	X	X	X	
Emery	112-62-9						
Total dust		X	X	X	X	X	

TABLE I.F.—SUBSTANCES INCLUDED IN THE UPDATE OF PELs FOR CONSTRUCTION, MARITIME, AND AGRICULTURE—Continued

Substance	CAS No.	Substances for which limits are being proposed in construction	Substances for which limits are being proposed in maritime			Substances for which limits are being proposed in agriculture	Substances for which limits are being proposed in general industry
			Ship repairing and ship breaking	Ship building	Marine terminals and longshoring		
Respirable fraction						X	
Endosulfan	115-29-7			X	X	X	
Endrin	72-20-8					X	
Epichlorohydrin	106-89-8	X	X	X	X	X	
EPN	2104-64-5					X	
1,2-Epoxypropane; see Propylene oxide							
2,3-Epoxy-1-propanol; see Glycidol							
Ethanethiol; see Ethyl mercaptan							
Ethanolamine	141-43-5	X	X	X	X	X	
Ethion	563-12-2	X	X	X	X	X	
Ethyl acetate	141-78-6					X	
Ethyl acrylate	140-88-5	X	X	X	X	X	
Ethyl alcohol (Ethanol)	64-17-5					X	
Ethylamine	75-04-7					X	
Ethyl amyl ketone (5-Methyl-3-heptanone).	541-85-5					X	
Ethyl benzene	100-41-4	X	X	X	X	X	
Ethyl bromide	74-96-4	X	X	X	X	X	
Ethyl butyl ketone (3-Heptanone)	106-35-4					X	
Ethyl chloride	75-500-3					X	
Ethyl ether	60-29-7	X	X	X	X	X	
Ethyl formate	109-94-4					X	
Ethyl mercaptan	75-08-1			X	X	X	
Ethyl silicate	78-10-4	X	X	X	X	X	
Ethylene chlorohydrin	107-07-3	X	X	X	X	X	
Ethylenediamine	107-15-3					X	
Ethylene dichloride (1,2-Dichloroethane).	107-06-2	X	X	X	X	X	
Ethylene glycol	107-21-1	X	X	X	X	X	
Ethylene glycol dinitrate	628-96-6	X	X	X	X	X	
Ethylene oxide; see 1910.1047	75-21-8					X	
Ethylidene norbornene	16219-75-3	X	X	X	X	X	
N-Ethylmorpholine	100-74-3	X	X	X	X	X	
Fenamiphos	22224-92-6	X	X	X	X	X	
Fensulfothion (Dasanit)	115-90-2	X	X	X	X	X	
Fenthion	55-38-9	X	X	X	X	X	
Ferbam	14484-64-1						
Total dust		X	X	X	X	X	
Ferrovanadium dust	12604	X	X	X	X	X	
Fibrous Glass (incl. RCFs)		X	X	X	X	X	X
Fluorides (as F)	(1)					X	
Fluorine	7782-41-4					X	
Fluorotrichloromethane (Trichlorofluoromethane).	75-69-4	X	X	X	X	X	
Fonofos	944-22-9	X	X	X	X	X	
Formaldehyde; see 1910.1048	50-00-00					X	
Formamide	75-12-7	X	X	X	X	X	
Formic acid	64-18-6					X	
Furfural	98-01-1	X	X	X	X	X	
Furfuryl alcohol	98-00-0	X	X	X	X	X	
Gasoline	8006-61-9	X	X	X	X	X	
Germanium tetrahydride	7782-65-2	X	X	X	X	X	
Glutaraldehyde	111-30-8	X	X	X	X	X	
Glycerin (mist)	56-81-5						
Total dust		X	X	X	X	X	
Respirable fraction						X	
Glycidol	556-52-5	X	X	X	X	X	
Grain dust (oat, wheat, barley)		X	X	X	X	X	
Graphite, natural Respirable dust	7782-42-5	X	X	X	X	X	
Graphite, synthetic	7782-42-5						
Total dust		X	X	X	X	X	
Respirable fraction						X	
Guthion; see Azinphos methyl							
Gypsum	7778-18-9						
Total dust						X	
Respirable fraction						X	
Hafnium	7440-58-6					X	
Heptachlor	76-44-8					X	
Heptane (n-Heptane)	142-82-5	X	X	X	X	X	
Hexachlorobutadiene	87-68-3	X	X	X	X	X	
Hexachlorocyclopentadiene	77-47-4	X	X	X	X	X	
Hexachloroethane	67-72-1					X	
Hexachloronaphthalene	1335-87-1					X	
Hexafluoroacetone	684-16-2	X	X	X	X	X	

TABLE I.F.—SUBSTANCES INCLUDED IN THE UPDATE OF PELs FOR CONSTRUCTION, MARITIME, AND AGRICULTURE—Continued

Substance	CAS No.	Substances for which limits are being proposed in construction	Substances for which limits are being proposed in maritime			Substances for which limits are being proposed in agriculture	Substances for which limits are being proposed in general industry
			Ship repairing and ship breaking	Ship building	Marine terminals and longshoring		
n-Hexane	110-54-3	X	X	X	X	X	
Hexane isomers	(1)	X	X	X	X	X	
2-Hexanone (Methyl n-butyl ketone)	591-78-6	X	X	X	X	X	
Hexone (Methyl isobutyl ketone)	108-10-1	X	X	X	X	X	
sec-Hexyl acetate	108-84-9					X	
Hexylene glycol	107-41-5	X	X	X	X	X	
Hydrazine	302-01-2	X	X	X	X	X	
Hydrogenated terphenyls	61788-32-7	X	X	X	X	X	
Hydrogen bromide	10035-10-6	X	X	X	X	X	
Hydrogen chloride	7647-01-0					X	
Hydrogen cyanide	74-90-8	X	X	X	X	X	
Hydrogen fluoride (as F)	7664-39-3	X	X	X	X	X	
Hydrogen peroxide	7722-84-1					X	
Hydrogen selenide (as Se)	7783-07-5					X	
Hydrogen sulfide	7783-06-4	X	X	X	X	X	
Hydroquinone	123-31-9					X	
2-Hydroxypropyl acrylate	999-61-1	X	X	X	X	X	
Indene	95-13-6			X	X	X	
Indium and compounds (as In)	7440-74-6			X	X	X	
Iodine	7553-56-2					X	
Iodoform	75-47-8	X	X	X	X	X	
Iron oxide fume(as Fe)	1309-37-1					X	
Iron pentacarbonyl (as Fe)	13463-40-6	X	X	X	X	X	
Iron salts (soluble)(as Fe)	(1)			X	X	X	
Isoamyl acetate	123-92-2					X	
Isoamyl alcohol (primary and secondary).	123-51-3	X	X	X	X	X	
Isobutyl acetate	110-19-0					X	
Isobutyl alcohol	78-83-1	X	X	X	X	X	
Isooctyl alcohol	26952-21-6	X	X	X	X	X	
Isophorone	78-59-1	X	X	X	X	X	
Isophorone diisocyanate	4098-71-9	X	X	X	X	X	
2-Isopropoxyethanol	109-59-1	X	X	X	X	X	
Isopropyl acetate	108-21-4	X	X	X	X	X	
Isopropyl alcohol	67-63-0	X	X	X	X	X	
Isopropylamine	75-31-0	X	X	X	X	X	
N-Isopropylaniline	768-52-5	X	X	X	X	X	
Isopropyl ether	108-20-3					X	
Isopropyl glycidyl ether (IGE)	4016-14-2	X	X	X	X	X	
Kaolin							
Total dust		X	X	X	X	X	
Respirable fraction						X	
Ketene	463-51-4	X	X	X	X	X	
Lead, inorganic; see 1910.1025	7439-92-1	X				X	
Limestone	1317-65-3						
Total dust						X	
Respirable fraction						X	
Lindane	~~58-89-9~~					X	
Lithium hydride	7580-67-8					X	
L.P.G. (Liquefied petroleum gas)	68476-85-7					X	
Magnesite	546-93-0						
Total dust						X	
Respirable fraction						X	
Magnesium oxide fume	1309-48-4						
Total particulate		X	X	X	X	X	
Malathion	121-75-5						
Total dust		X	X	X	X	X	
Maleic anhydride	108-31-6					X	
Manganese compounds (as Mn)	7439-96-5					X	
Manganese fume (as Mn)	7439-96-5	X	X	X	X	X	
Manganese cyclopentadienyl tricarbonyl (as Mn).	12079-65-1	X	X	X	X	X	
Manganese tetroxide (as Mn)	1317-35-7	X	X	X	X	X	
Marble	1317-65-3						
Total dust						X	
Respirable fraction						X	
Mercury (aryl and inorganic) (as Hg)	7439-97-6					X	
Mercury (organo) alkyl compounds (as Hg).	7439-97-6	X	X	X	X	X	
Mercury (vapor) (as Hg)	7439-97-6	X	X	X	X	X	
Mesityl oxide	141-79-7	X	X	X	X	X	
Methacrylic acid	79-41-4	X	X	X	X	X	
Methanethiol; see Methyl mercaptan							
Methomyl (Lannate)	16752-77-5	X	X	X	X	X	
Methoxychlor Total dust	72-43-5	X	X	X	X	X	

TABLE I.F.—SUBSTANCES INCLUDED IN THE UPDATE OF PELS FOR CONSTRUCTION, MARITIME, AND AGRICULTURE—Continued

Substance	CAS No.	Substances for which limits are being proposed in construction	Substances for which limits are being proposed in maritime			Substances for which limits are being proposed in agriculture	Substances for which limits are being proposed in general industry
			Ship repairing and ship breaking	Ship building	Marine terminals and longshoring		
4-Methoxyphenol	150-76-5	X	X	X	X	X	
Methyl acetate	79-20-9	X	X	X	X	X	
Methyl acetylene (Propyne)	74-99-7					X	
Methyl acetylene-propadiene mixture (MAPP).		X	X	X	X	X	
Methyl acrylate	96-33-3					X	
Methylacrylonitrile	126-98-7	X	X	X	X	X	
Methylal (Dimethoxy-methane)	109-87-5					X	
Methyl alcohol	67-56-1	X	X	X	X	X	
Methylamine	74-89-5					X	
Methyl amyl alcohol; see Methyl isobutyl carbinol							
Methyl n-amyl ketone	110-43-0					X	
Methyl bromide	74-83-9	X	X	X	X	X	
Methyl butyl ketone; see 2-Hexanone							
Methyl chloride	74-87-3	X	X	X	X	X	
Methyl chloroform (1,1,1-Trichloroethane).	71-55-6	X	X	X	X	X	
Methyl 2-cyanoacrylate	137-05-3	X	X	X	X	X	
Methylcyclohexane	108-87-2	X	X	X	X	X	
Methylcyclohexanol	25639-42-3	X	X	X	X	X	
o-Methylcyclohexanone	583-60-8	X	X	X	X	X	
Methylcyclopentadienyl manganese tricarbonyl (as Mn).	12108-13-3	X	X	X	X	X	
Methyl demeton	8022-00-2	X	X	X	X	X	
4,4'-Methylene bis (2-chloroaniline) (MBOCA).	101-14-4	X	X	X	X	X	
Methylene bis (4-cyclohexylisocyanate).	5124-30-1	X	X	X	X	X	
Methyl ethyl ketone (MEK); see 2-Butanone							
Methyl ethyl ketone peroxide (MEKP)	1338-23-4	X	X	X	X	X	
Methyl formate	107-31-3	X	X	X	X	X	
Methyl hydrazine (Monomethyl hydrazine).	60-34-4					X	
Methyl iodide	74-88-4	X	X	X	X	X	
Methyl isoamyl ketone	110-12-3	X	X	X	X	X	
Methyl isobutyl carbinol	108-11-2	X	X	X	X	X	
Methyl isobutyl ketone; see Hexone							
Methyl isocyanate	624-83-9					X	
Methyl isopropyl ketone	563-80-4	X	X	X	X	X	
Methyl mercaptan	74-93-1			X	X	X	
Methyl methacrylate	80-62-6					X	
Methyl parathion	298-00-0	X	X	X	X	X	
Methyl propyl ketone; see 2-Pentanone							
Methyl silicate	681-84-5	X	X	X	X	X	
Alpha-Methyl styrene	98-83-9	X	X	X	X	X	
Methylene bisphenyl isocyanate (MDI).	101-68-8					X	
Metribuzin	21087-64-9	X	X	X	X	X	
Mica; see Silicates							
Mineral wool		X	X	X	X	X	X
Molybdenum (as Mo)	7439-98-7						
Soluble compounds						X	
Insoluble compounds							
Total dust		X	X	X	X	X	
Monocrotophos (Azodrin)	6923-22-4	X	X	X	X	X	
Monomethyl aniline	100-61-8	X	X	X	X	X	
Morpholine	110-91-8	X	X	X	X	X	
Naphtha (Coal tar)	8030-30-6					X	
Naphthalene	91-20-3	X	X	X	X	X	
Nickel, metal and insoluble compounds (as Ni).	7440-02-0					X	
Nickel, soluble compounds (as Ni)	7440-02-0	X	X	X	X	X	
Nicotine	54-11-5					X	
Nitric acid	7697-37-2	X	X	X	X	X	
Nitric oxide	10102-43-9					X	
p-Nitroaniline	100-01-6	X	X	X	X	X	
Nitrobenzene	98-95-3					X	
p-Nitrochlorobenzene	100-00-5					X	
Nitroethane	79-24-3					X	
Nitrogen dioxide	10102-44-0	X	X	X	X	X	
Nitrogen trifluoride	7783-54-2					X	
Nitroglycerin	55-63-0	X	X	X	X	X	

TABLE I.F.—SUBSTANCES INCLUDED IN THE UPDATE OF PELS FOR CONSTRUCTION, MARITIME, AND AGRICULTURE—Continued

Substance	CAS No.	Substances for which limits are being proposed in construction	Substances for which limits are being proposed in maritime			Substances for which limits are being proposed in agriculture	Substances for which limits are being proposed in general industry
			Ship repairing and ship breaking	Ship building	Marine terminals and longshoring		
Nitromethane	75-52-5					X	
1-Nitropropane	108-03-2					X	
2-Nitropropane	79-46-9	X	X	X	X	X	
Nitrotoluene							
m-isomer	99-08-1						
o-isomer	88-72-2						
p-isomer	99-99-0	X	X	X	X	X	
Nitrotrichloromethane; see Chloropicrin							
Nonane	111-84-2	X	X	X	X	X	
Octachloronaphthalene	2234-13-1	X	X	X	X	X	
Octane	111-65-9	X	X	X	X	X	
Oil mist, mineral	8012-95-1					X	
Osmium tetroxide (as Os)	20816-12-0	X	X	X	X	X	
Oxalic acid	144-62-7	X	X	X	X	X	
Oxygen difluoride	7783-41-7	X	X	X	X	X	
Ozone	10028-15-6	X	X	X	X	X	
Paraffin wax fume	8002-74-2	X	X	X	X	X	
Paraquat, respirable dust	4685-14-7	X	X	X	X	X	
	1910-42-5						
	2074-50-2						
Parathion	56-38-2					X	
Particulates not otherwise regulated							
Total dust	—	X	X	X	X	X	
Respirable fraction	—	X	X	X	X	X	
Pentaborane	19624-22-7	X	X	X	X	X	
Pentachloronaphthalene	1321-64-8					X	
Pentachlorophenol	87-86-5					X	
Pentaerythritol	115-77-5						
Total dust		X	X	X	X	X	
Respirable fraction						X	
Pentane	109-66-0	X	X	X	X	X	
2-Pentanone (Methyl propyl ketone)	107-87-9	X	X	X	X	X	
Perchloroethylene (Tetrachloroethylene).	127-18-4	X	X	X	X	X	
Perchloromethyl mercaptan	594-42-3					X	
Perchloryl fluoride	7616-94-6	X	X	X	X	X	
Perlite	93763-70-3						
Total dust						X	
Respirable fraction						X	
Petroleum distillates (Naphtha)		X	X	X	X	X	
Phenol	108-95-2					X	
Phenothiazine	92-84-2	X	X	X	X	X	
p-Phenylene diamine	106-50-3					X	
Phenyl ether, vapor	101-84-8					X	
Phenyl ether-biphenyl mixture, vapor						X	
Phenylethylene; see Styrene							
Phenyl glycidyl ether (PGE)	122-60-1	X	X	X	X	X	
Phenylhydrazine	100-63-0	X	X	X	X	X	
Phenyl mercaptan	108-98-5	X	X	X	X	X	
Phenylphosphine	638-21-1	X	X	X	X	X	
Phorate	298-02-2	X	X	X	X	X	
Phosdrin (Mevinphos)	7786-34-7	X	X	X	X	X	
Phosgene (Carbonyl chloride)	75-44-5					X	
Phosphine	7803-51-2	X	X	X	X	X	
Phosphoric acid	7664-38-2	X	X	X	X	X	
Phosphorus (yellow)	7723-14-0					X	
Phosphorus oxychloride	10025-87-3	X	X	X	X	X	
Phosphorus pentachloride	10026-13-8					X	
Phosphorus pentasulfide	1314-80-3	X	X	X	X	X	
Phosphorus trichloride	7719-12-2	X	X	X	X	X	
Phthalic anhydride	85-44-9	X	X	X	X	X	
m-Phthalodinitrile	626-17-5	X	X	X	X	X	
Picloram	1918-02-1						
Total dust		X	X	X	X	X	
Respirable fraction						X	
Picric acid	88-89-1					X	
Piperazine dihydrochloride	142-64-3	X	X	X	X	X	
Pindone (2-Pivalyl-1,3-indandione)	83-26-1					X	
Plaster of Paris	26499-65-0						
Total dust						X	
Respirable fraction						X	
Platinum (as Pt) Metal	7440-06-4	X	X	X	X	X	
Soluble salts						X	
Portland cement	65997-15-1						

TABLE I.F.—SUBSTANCES INCLUDED IN THE UPDATE OF PELS FOR CONSTRUCTION, MARITIME, AND AGRICULTURE—Continued

Substance	CAS No.	Substances for which limits are being proposed in construction	Substances for which limits are being proposed in maritime			Substances for which limits are being proposed in agriculture	Substances for which limits are being proposed in general industry
			Ship repairing and ship breaking	Ship building	Marine terminals and longshoring		
Total dust		X	X	X	X	X	
Respirable fraction						X	
Potassium hydroxide	1310-58-3	X	X	X	X	X	
Propargyl alcohol	107-19-7			X	X	X	
Propionic acid	79-09-4	X	X	X	X	X	
Propoxur (Baygon)	114-26-1	X	X	X	X	X	
n-Propyl acetate	109-60-4	X	X	X	X	X	
n-Propyl alcohol	71-23-8	X	X	X	X	X	
n-Propyl nitrate	627-13-4	X	X	X	X	X	
Propylene dichloride	78-87-5	X	X	X	X	X	
Propylene glycol dinitrate	6423-43-4	X	X	X	X	X	
Propylene glycol monomethyl ether	107-98-2	X	X	X	X	X	
Propylene imine	75-55-8					X	
Propylene oxide	75-56-9	X	X	X	X	X	
Propyne; see Methyl acetylene							
Pyrethrum	8003-34-7					X	
Pyridine	110-86-1					X	
Quinone	106-51-4					X	
RDX; see Cyclonite							
Resorcinol	108-46-3	X	X	X	X	X	
Rhodium (as Rh), metal fume and insoluble compounds.	7440-16-6					X	
Rhodium (as Rh), soluble compounds	7440-16-6					X	
Ronnel	299-84-3			X	X	X	
Rosin core solder pyrolysis products, as formaldehyde.		X	X	X	X	X	
Rotenone	83-79-4					X	
Rouge							
Total dust		X	X	X	X	X	
Respirable fraction						X	
Selenium compounds (as Se)	7782-49-2					X	
Selenium hexafluoride (as Se)	7783-79-1					X	
Silica, amorphous, precipitated and gel.	112926-00-8	X	X	X	X	X	
Silica, amorphous, diatomaceous earth, containing less than 1% crystalline silica.	61790-53-2	X	X	X	X	X	
Silica, crystalline cristobalite, respirable dust.	14464-46-1	X	X	X	X	X	
Silica, crystalline quartz, respirable dust.	14808-60-7	X	X	X	X	X	
Silica, crystalline tripoli (as quartz), respirable dust.	1317-95-9	X	X	X	X	X	
Silica, crystalline tridymite, respirable dust.	15468-32-3	X	X	X	X	X	
Silica, fused, respirable dust	60676-86-0	X	X	X	X	X	
Silicates (less than 1% crystalline silica) Mica (respirable dust).	12001-26-2	X	X	X	X	X	
Soapstone, total dust		X	X	X	X	X	
Soapstone, respirable		X	X	X	X	X	
Talc (containing asbestos); use asbestos limit.						X	
Talc (containing no asbestos), Respirable dust.	14807-96-6	X	X	X	X	X	
Tremolite						X	
Silicon	7440-21-3						
Total dust		X	X	X	X	X	
Respirable fraction						X	
Silicon carbide	409-21-2						
Total dust		X	X	X	X	X	
Respirable fraction						X	
Silicon tetrahydride	7803-62-5	X	X	X	X	X	
Silver, metal and soluble compounds (as Ag).	7440-22-4					X	
Soapstone; see Silicates.							
Sodium azide	26628-22-8						
(as HN_3)		X	X	X	X	X	
(as NaN_3)		X	X	X	X	X	
Sodium bisulfite	7631-90-5	X	X	X	X	X	
Sodium fluoroacetate	62-74-8	X	X	X	X	X	
Sodium hydroxide	1310-73-2	X	X	X	X	X	
Sodium metabisulfite	7681-57-4	X	X	X	X	X	
Starch	9005-25-8						
Total dust						X	
Respirable fraction						X	

TABLE I.F.—SUBSTANCES INCLUDED IN THE UPDATE OF PELs FOR CONSTRUCTION, MARITIME, AND AGRICULTURE—Continued

Substance	CAS No.	Substances for which limits are being proposed in construction	Substances for which limits are being proposed in maritime			Substances for which limits are being proposed in agriculture	Substances for which limits are being proposed in general industry
			Ship repairing and ship breaking	Ship building	Marine terminals and longshoring		
Stibine	7803-52-3					X	
Stoddard solvent	8052-41-3	X	X	X	X	X	
Strychnine	57-24-9					X	
Styrene	100-42-5	X	X	X	X	X	
Subtilisins (Proteolytic enzymes)	9014-01-1	X	X	X	X	X	
Sucrose	57-50-1						
Total dust						X	
Respirable fraction						X	
Sulfur dioxide	7446-09-5	X	X	X	X	X	
Sulfur hexafluoride	2551-62-4					X	
Sulfuric acid	7664-93-9					X	
Sulfur monochloride	10025-67-9	X	X	X	X	X	
Sulfur pentafluoride	5714-22-7	X	X	X	X	X	
Sulfur tetrafluoride	7783-60-0	X	X	X	X	X	
Sulfuryl fluoride	2699-79-8	X	X	X	X	X	
Sulprofos	35400-43-2	X	X	X	X	X	
Systox, see Demeton							
2,4,5-T (Trichlorophenoxyacetic acid)	93-76-5					X	
Talc; see Silicates							
Tantalum, metal and oxide dust	7440-25-7					X	
TEDP (Sulfotep)	3689-24-5					X	
Tellurium and compounds (as Te)	13494-80-9					X	
Tellurium hexafluoride (as Te)	7783-80-4					X	
Temephos (Abate)	3383-96-8						
Total dust		X	X	X	X	X	
Respirable fraction		X	X			X	
TEPP	107-49-3					X	
Terphenyls	26140-60-3	X	X	X	X	X	
1,1,1,2-Tetrachloro-2,2-difluoroethane	76-11-9					X	
1,1,2,2-Tetrachloro-1,2-difluoroethane	76-12-0					X	
1,1,2,2-Tetrachloroethane	79-34-5	X	X	X	X	X	
Tetrachloroethylene; see Perchloroethylene							
Tetrachloromethane; see Carbon tetrachloride							
Tetrachloronaphthalene	1335-88-2					X	
Tetraethyl lead (as Pb)	78-00-2	X	X			X	
Tetrahydrofuran	109-99-9	X	X	X	X	X	
Tetramethyl lead (as Pb)	75-74-1	X	X			X	
Tetramethyl succinonitrile	3333-52-6					X	
Tetranitromethane	509-14-8					X	
Tetrasodium pyrophosphate	7722-88-5	X	X	X	X	X	
Tetryl (2,4,6-Trinitrophenyl-methylnitramine)	479-45-8	X	X	X	X	X	
Thallium, soluble compounds (as Tl)	7440-28-0					X	
4,4'-Thiobis(6-tert, Butyl-m-cresol)	96-69-5						
Total dust		X	X	X	X	X	
Respirable fraction						X	
Thioglycolic acid	68-11-1	X	X	X	X	X	
Thionyl chloride	7719-09-7	X	X	X	X	X	
Thiram	137-26-8					X	
Tin, inorganic compounds (except oxides) (as Sn)	7440-31-5					X	
Tin, organic compounds (as Sn)	7440-31-5	X	X	X	X	X	
Tin oxide (as Sn)	21651-19-4	X	X	X	X	X	
Titanium dioxide	13463-67-7						
Total dust		X	X	X	X	X	
Toluene	108-88-3	X	X	X	X	X	
Toluene-2,4-diisocyanate (TDI)	584-84-9	X	X	X	X	X	
m-Toluidine	108-44-1	X	X	X	X	X	
o-Toluidine	95-53-4					X	
p-Toluidine	106-49-0	X	X	X	X	X	
Toxaphene; see Chlorinated camphene							
Tremolite; see Silicates							
Tributyl phosphate	126-73-8	X	X	X	X	X	
Trichloroacetic acid	76-03-9	X	X	X	X	X	
1,2,4-Trichlorobenzene	120-82-1	X	X	X	X	X	
1,1,1-Trichloroethane; see Methyl chloroform							
1,1,2-Trichloroethane	79-00-5					X	
Trichloroethylene	79-01-6	X	X	X	X	X	
Trichloromethane; see Chloroform							
Trichloronaphthalene	1321-65-9					X	
1,2,3-Trichloropropane	96-18-4	X	X	X	X	X	

TABLE I.F.—SUBSTANCES INCLUDED IN THE UPDATE OF PELs FOR CONSTRUCTION, MARITIME, AND AGRICULTURE—Continued

Substance	CAS No.	Substances for which limits are being proposed in construction	Substances for which limits are being proposed in maritime			Substances for which limits are being proposed in agriculture	Substances for which limits are being proposed in general industry
			Ship repairing and ship breaking	Ship building	Marine terminals and longshoring		
1,1,2-Trichloro-1,2,2-trifluoroethane	76-13-1	X	X	X	X	X	
Triethylamine	121-44-8	X	X	X	X	X	
Trifluorobromomethane	75-63-8					X	
Trimellitic anhydride	552-30-7	X	X	X	X	X	
Trimethylamine	75-50-3	X	X	X	X	X	
Trimethyl benzene	25551-13-7			X	X	X	
Trimethyl phosphite	121-45-9	X	X	X	X	X	
2,4,6-Trinitrophenyl; see Picric acid							
2,4,6-Trinitrophenylmethy nitramine; see Tetryl							
2,4,6-Trinitrotoluene (TNT)	118-96-7	X	X	X	X	X	
Triorthocresyl phosphate	78-30-8					X	
Triphenyl amine	603-34-9	X	X	X	X	X	
Triphenyl phosphate	115-86-6					X	
Tungsten (as W)	7440-33-7						
Insoluble compounds		X	X	X	X	X	
Soluble compounds		X	X	X	X	X	
Turpentine	8006-64-2					X	
Uranium (as U)	7440-61-1						
Soluble compounds		X	X	X	X	X	
Insoluble compounds		X	X	X	X	X	
n-Valeraldehyde	110-62-3	X	X	X	X	X	
Vanadium	1314-62-1						
Respirable dust (as V_2O_5)		X	X	X	X	X	
Fume (as V_2O_5)		X	X	X	X	X	
Vegetable oil mist							
Total dust						X	
Respirable fraction						X	
Vinyl acetate	108-05-4	X	X	X	X	X	
Vinyl benzene; see Styrene							
Vinyl bromide	593-60-2	X	X	X	X	X	
Vinyl chloride; see 1910.1017	75-01-4					X	
Vinyl cyanide; see Acrylonitrile							
Vinyl cyclohexene dioxide	106-87-6	X	X	X	X	X	
Vinylidene chloride (1,1-Dichloroethylene).	75-35-4	X	X	X	X	X	
Vinyl toluene	25013-15-4					X	
VM & P Naphtha	8032-32-4	X	X	X	X	X	
Warfarin	81-81-2					X	
Welding fumes (total particulate)		X	X	X	X	X	
Wood dust, all soft and hardwoods, except Western red cedar.		X	X	X	X	X	
Wood dust, Western red cedar		X	X	X	X	X	
Xylenes (o-, m-, p- isomers)	1330-20-7	X	X	X	X	X	
m-Xylene alpha, alpha'-diamine	1477-55-0	X	X	X	X	X	
Xylidine	1300-73-8	X	X	X	X	X	
Yttrium	7440-65-5					X	
Zinc chloride fume	7646-85-7	X	X	X	X	X	
Zinc chromate (as CrO_3)	(1)	X	X	X	X	X	
Zinc oxide fume (as Zn)	1314-13-2	X	X	X	X	X	
Zinc oxide (as Zn)	1314-13-2						
Total dust		X	X	X	X	X	
Respirable fraction						X	
Zinc stearate	557-05-1						
Total dust		X	X	X	X	X	
Respirable fraction						X	
Zirconium compounds (as Zr)	7440-67-7	X	X	X	X	X	

[1] Varies with compounds.

TABLE I.G.—INDEX TO PREAMBLE DISCUSSION OF INDIVIDUAL SUBSTANCES

H.S. No.	Substance name	CAS No.	Primary basis for limits	Preamble section
1001	Acetaldehyde	75-07-0	Sensory irritation	IV.C.3
1002	Acetic acid	64-19-7	Sensory irritation	IV.C.3
1003	Acetic anhydride	108-24-7	Analogy	IV.C.11
1004	Acetone	67-64-1	Sensory irritation	IV.C.3
1005	Acetonitrile	75-05-8	Systemic toxicity	IV.C.8
2001	Acetylene tetrabromide	79-27-6	Liver and kidney effects	IV.C.4
1006	Acetylsalicylic acid (aspirin)	50-78-2	Systemic toxicity	IV.C.8
1007	Acrolein	107-02-8	Sensory irritation	IV.C.3
1008	Acrylamide	79-06-1	Cancer	IV.C.14

TABLE I.G.—INDEX TO PREAMBLE DISCUSSION OF INDIVIDUAL SUBSTANCES—Continued

H.S. No.	Substance name	CAS No.	Primary basis for limits	Preamble section
1009	Acrylic acid	79-10-7	Analogy	IV.C.11
2002	Acrylonitrile	107-13-1	Cancer	IV.C.14
2003	Aldrin	309-00-2	Liver and kidney effects	IV.C.4
1010	Allyl alcohol	107-18-6	Sensory irritation	IV.C.3
1011	Allyl chloride	107-05-1	Liver and kidney effects	IV.C.4
1012	Allyl glycidyl ether (age)	106-92-3	Sensory irritation	IV.C.3
1013	Allyl propyl disulfide	2179-59-1	Sensory irritation	IV.C.3
1014	Alpha-alumina	1344-28-1	Physical irritation	IV.C.10
1015	Aluminum (alkyls)	7429-90-5	Analogy	IV.C.11
1016	Aluminum (metal)	7429-90-5	Physical irritation	IV.C.10
1017	Aluminum (pyro powders)	7429-90-5	Respiratory effects	IV.C.6
1018	Aluminum (soluble salts)	7429-90-5	Analogy	IV.C.11
1019	Aluminum (welding fumes)	7429-90-5	Systemic toxicity	IV.C.8
2004	2-Aminopyridine	504-29-0	Systemic toxicity	IV.C.8
1020	Amitrole (3-amino-1,2,4-triazole)	61-82-5	Cancer	IV.C.14
1021	Ammonia	7664-41-7	Sensory irritation	IV.C.3
1022	Ammonium chloride (fume)	12125-02-9	Sensory irritation	IV.C.3
1024	Ammonium sulfamate (ammate)	7773-06-0	Physical irritation	IV.C.10
2005	N-amyl acetate	628-63-7	Sensory irritation	IV.C.3
2006	Sec-amyl acetate	626-38-0	Analogy	IV.C.11
1025	Aniline	62-53-3	Biochemical/metabolic effects	IV.C.12
2007	Anisidine (O, P-isomers)	29191-52-4	Systemic toxicity	IV.C.8
2008	Antimony and compounds	7440-36-0	Cardiovascular effects	IV.C.7
2009	Antu	86-88-4	Cancer	IV.C.14
2010	Arsine	7784-42-1	Systemic toxicity	IV.C.8
2011	Arsenic, inorganic compounds	7440-38-2	Cancer	IV.C.14
2012	Arsenic, organic compounds	7440-36-0	Systemic toxicity	IV.C.8
2013	Asbestos	Varies	Cancer	IV.C.14
1028	Asphalt fumes	8052-42-4	Respiratory effects	IV.C.6
1029	Atrazine	1912-24-9	Noael	IV.C.9
2014	Azinphos-methyl	86-50-0	Noael	IV.C.9
2015	Barium (soluble compounds)	7440-39-3	Systemic toxicity	IV.C.8
1031	Barium sulfate	7727-43-7	Physical irritation	IV.C.10
1032	Benomyl	17804-35-2	Physical irritation	IV.C.10
2016	Benzene	71-43-2	Cancer	IV.C.14
2017	Benzoyl peroxide	94-36-0	Sensory irritation	IV.C.3
2018	Benzyl chloride	100-44-7	Sensory irritation	IV.C.3
1033	Beryllium & compounds	7440-41-7	Cancer	IV.C.14
2019	Biphenyl (diphenyl)	92-52-4	Neuropathy	IV.C.1
1034	Bismuth telluride (SE-doped)	1304-82-1	Respiratory effects	IV.C.6
1035	Bismuth telluride (undoped)	1304-82-1	Physical irritation	IV.C.10
1036	Borates, tetra, sodium (anhydrous)	1330-43-4	Sensory irritation	IV.C.3
1037	Borates, tetra, sodium (decahydrate)	1303-96-4	Sensory irritation	IV.C.3
1038	Borates, tetra, sodium (pentahydrate)	12179-04-3	Sensory irritation	IV.C.3
1039	Boron oxide	1303-86-2	Physical irritation	IV.C.10
1040	Boron tribromide	10294-33-4	Analogy	IV.C.11
2020	Boron trifluoride	7637-07-2	Sensory irritation	IV.C.3
1041	Bromacil	314-40-9	Noael	IV.C.9
1042	Bromine	7726-95-6	Sensory irritation	IV.C.3
1043	Bromine pentafluoride	7789-30-2	Analogy	IV.C.11
2021	Bromoform	75-25-2	Sensory irritation	IV.C.3
1044	Butane	106-97-8	Narcosis	IV.C.2
1045	2-Butanone (mek)	78-93-3	Sensory irritation	IV.C.3
1046	2-Butoxyethanol	111-76-2	Systemic toxicity	IV.C.8
1047	N-butyl acetate	123-86-4	Sensory irritation	IV.C.3
2022	Sec-butyl acetate	105-46-4	Analogy	IV.C.11
2023	Tert-butyl acetate	540-88-5	Analogy	IV.C.11
1048	Butyl acrylate	141-32-2	Analogy	IV.C.11
1049	Sec-butyl alcohol	78-92-2	Narcosis	IV.C.2
1050	Tert-butyl alcohol	75-65-0	Narcosis	IV.C.2
1051	N-butyl alcohol	71-36-3	Neuropathy	IV.C.1
2025	Butylamine	109-73-9	Sensory irritation	IV.C.3
2024	Tert-butyl chromate	1189-85-1	Systemic toxicity	IV.C.8
1052	N-butyl glycidyl ether (BGE)	2426-08-6	Systemic toxicity	IV.C.8
1053	N-butyl lactate	138-22-7	Sensory irritation	IV.C.3
1054	Butyl mercaptan	109-79-5	Sensory irritation	IV.C.3
1055	O-sec-butyl phenol	89-72-5	Analogy	IV.C.11
1056	P-tert-butyltoluene	98-51-1	Noael	IV.C.9
1057	Calcium carbonate	1317-65-3	Physical irritation	IV.C.10
1058	Calcium cyanamide	156-62-7	Biochemical/metabolic effects	IV.C.12
1059	Calcium hydroxide	1305-62-0	Analogy	IV.C.11
1060	Calcium oxide	1305-78-8	Analogy	IV.C.11
1061	Calcium silicate, total dust	1344-95-2	Physical irritation	IV.C.10
1062	Calcium sulfate	7778-18-9	Physical irritation	IV.C.10
1063	Camphor (synthetic)	76-22-2	Sensory irritation	IV.C.3
1064	Caprolactam (dust)	105-60-2	Sensory irritation	IV.C.3
1065	Caprolactam (vapor)	105-60-2	Sensory irritation	IV.C.3
1066	Captafol (difolatan)	2425-06-1	Sensitization effects	IV.C.13
1067	Captan	133-06-2	Systemic toxicity	IV.C.8

TABLE I.G.—INDEX TO PREAMBLE DISCUSSION OF INDIVIDUAL SUBSTANCES—Continued

H.S. No.	Substance name	CAS No.	Primary basis for limits	Preamble section
2026	Carbaryl (sevin)	63-25-2	Systemic toxicity	IV.C.8
1068	Carbofuran (furadan)	1563-66-2	Biochemical/metabolic effects	IV.C.12
2027	Carbon black	1333-86-4	Respiratory effects	IV.C.6
1069	Carbon dioxide	124-38-9	Biochemical/metabolic effects	IV.C.12
1070	Carbon disulfide	75-15-0	Cardiovascular effects	IV.C.7
1071	Carbon monoxide	630-08-0	Biochemical/metabolic effects	IV.C.12
1072	Carbon tetrabromide	558-13-4	Liver and kidney effects	IV.C.4
1073	Carbon tetrachloride	56-23-5	Cancer	IV.C.14
1074	Carbonyl fluoride	353-50-4	Analogy	IV.C.11
1075	Catechol (pyrocatechol)	120-80-9	Analogy	IV.C.11
1076	Cellulose	9004-34-6	Physical irritation	IV.C.10
1077	Cesium hydroxide	21351-79-1	Sensory irritation	IV.C.3
2028	Chlordane	57-74-9	Liver and kidney effects	IV.C.4
1078	Chlorinated camphene	8001-35-2	Neuropathy	IV.C.1
2029	Chlorinated diphenyl oxide	55720-99-5	Liver and kidney effects	IV.C.4
1079	Chlorine	7782-50-5	Sensory irritation	IV.C.3
1080	Chlorine dioxide	10049-04-4	Respiratory effects	IV.C.6
2030	Chlorine trifluoride	7790-91-2	Sensory irritation	IV.C.3
2031	Chloroacetaldehyde	107-20-0	Sensory irritation	IV.C.3
2032	Alpha-chloroacetophenone	532-27-4	Sensory irritation	IV.C.3
1083	Chloroacetyl chloride	79-04-9	Sensory irritation	IV.C.3
2033	Chlorobenzene	108-90-7	Liver and kidney effects	IV.C.4
1084	O-Chlorobenzylidene malononitrile	2698-41-1	Sensory irritation	IV.C.3
2034	Chlorobromomethane	74-97-5	Narcosis	IV.C.2
1085	Chlorodifluoromethane	75-45-6	Noael	IV.C.9
2035	Chlorodiphenyl 42% (aroclor 1242)	53469-21-9	Liver and kidney effects	IV.C.4
2036	Chlorodiphenyl 54% (aroclor 1254)	11097-69-1	Liver and kidney effects	IV.C.4
1086	Chloroform	67-66-3	Cancer	IV.C.14
1081	1-Chloro-1-nitropropane	600-25-9	Analogy	IV.C.11
1087	Chloropentafluoroethane	76-15-3	Cardiovascular effects	IV.C.7
2037	Chloropicrin	76-06-2	Sensory irritation	IV.C.3
1088	Chloroprene	126-99-8	Systemic toxicity	IV.C.8
1089	O-Chlorostyrene	2039-87-4	Liver and kidney effects	IV.C.4
1090	O-Chlorotoluene	95-49-8	Noael	IV.C.9
1082	2-Chloro-6-trichloromethyl pyridine (nitrapyrin)	1929-82-4	Physical irritation	IV.C.10
1091	Chlorpyrifos	2921-88-2	Biochemical/metabolic effects	IV.C.12
1092	Chromic acid & chromates	Varies	Cancer	IV.C.14
2038	Chromium (II) compounds	7440-47-3	Respiratory effects	IV.C.6
2038A	Chromium (III) compounds	7440-47-3	Respiratory effects	IV.C.6
1093	Chromium, metal	7440-47-3	Respiratory effects	IV.C.6
1094	Chromyl chloride	14977-61-8	Cancer	IV.C.14
1095	Clopidol (coyden)	2971-90-6	Physical irritation	IV.C.10
1096	Coal dust, < 5% quartz	None	Respiratory effects	IV.C.6
1097	Coal dust, > 5% quartz	None	Respiratory effects	IV.C.6
2039	Coal tar pitch volatiles	65966-93-2	Cancer	IV.C.14
1098	Cobalt carbonyl	10210-68-1	Analogy	IV.C.11
1099	Cobalt hydrocarbonyl	16842-03-8	Analogy	IV.C.11
1100	Cobalt metal, fume, dust	7440-48-4	Sensitization effects	IV.C.13
2039A	Coke oven emissions	None	Cancer	IV.C.14
1101	Copper (fume)	7440-50-8	Sensory irritation	IV.C.3
2040	Copper (dusts and mists)	7440-50-8	Sensory irritation	IV.C.3
2041	Cotton dust	None	Respiratory effects	IV.C.6
1102	Crag® herbicide (sesone)	136-78-7	Physical irritation	IV.C.10
2042	Cresol (o, m, p-isomers)	1319-77-3	Analogy	IV.C.11
2043	Crotonaldehyde	123-73-9; 4170-30-3	Analogy	IV.C.11
1103	Crufomate	299-86-5	Biochemical/metabolic effects	IV.C.12
2044	Cumene	98-82-8	Narcosis	IV.C.2
1104	Cyanamide	420-04-2	Biochemical/metabolic effects	IV.C.12
2045	Cyanides	Varies	Systemic toxicity	IV.C.8
1105	Cyanogen	460-19-5	Sensory irritation	IV.C.3
1106	Cyanogen chloride	506-77-4	Sensory irritation	IV.C.3
2046	Cyclohexane	110-82-7	Sensory irritation	IV.C.3
1107	Cyclohexanol	108-93-0	Change in skin designation only	IV.C.16
1108	Cyclohexanone	108-94-1	Liver and kidney effects	IV.C.4
2047	Cyclohexene	110-83-8	Sensory irritation	IV.C.3
1109	Cyclohexylamine	108-91-8	Systemic toxicity	IV.C.8
1110	Cyclonite	121-82-4	Noael	IV.C.9
2048	Cyclopentadiene	542-92-7	Sensory irritation	IV.C.3
1111	Cyclopentane	287-92-3	Narcosis	IV.C.2
1112	Cyhexatin	13121-70-5	Systemic toxicity	IV.C.8
2050	2,4-D (Dichlorophenoxyacetic acid)	94-75-7	Systemic toxicity	IV.C.8
1113	DDT	50-29-3	Systemic toxicity	IV.C.8
2051	DDVP (dichlorvos)	62-73-7	Biochemical/metabolic effects	IV.C.12
1114	Decaborane	17702-41-9	Neuropathy	IV.C.1
2049	Demeton	8065-48-3	Analogy	IV.C.11
2052	Diacetone alcohol	123-42-2	Sensory irritation	IV.C.3
1116	Di-sec-octyl-phthalate	117-81-7	Neuropathy	IV.C.1

TABLE I.G.—INDEX TO PREAMBLE DISCUSSION OF INDIVIDUAL SUBSTANCES—Continued

H.S. No.	Substance name	CAS No.	Primary basis for limits	Preamble section
1117	2,6-Di-tert-butyl-p-cresol	128-37-0	Noael	IV.C.9
1118	Diazinon	333-41-5	Analogy	IV.C.11
2053	Diazomethane	334-88-3	Sensory irritation	IV.C.3
2054	Diborane	19287-45-7	Neuropathy	IV.C.1
2055	1,2-Dibromo-3-chloropropane (DBCP)	96-12-8	Cancer	IV.C.14
1119	Dibutyl phosphate	107-66-4	Sensory irritation	IV.C.3
1120	2-N-Dibutylaminoethanol	102-81-8	Systemic toxicity	IV.C.8
2056	Dibutylphthalate	84-74-2	Noael	IV.C.9
1123	Dichloroacetylene	7572-29-4	Neuropathy	IV.C.1
2057	O-Dichlorobenzene	95-50-1	Sensory irritation	IV.C.3
1125	P-Dichlorobenzene	106-46-7	Analogy	IV.C.11
2058	Dichlorodifluoromethane	75-71-8	Cardiovascular effects	IV.C.7
1126	1,1-Dichloroethane	75-34-3	Liver and kidney effects	IV.C.4
2059	1,2-Dichloroethylene	540-59-0	Noael	IV.C.9
1127	Dichloroethyl ether	111-44-4	Sensory irritation	IV.C.3
1122	1,3-Dichloro-5, 5-Dimethyl-hydantoin	118-52-5	Sensory irritation	IV.C.3
1128	Dichloromonofluoromethane	75-43-4	Analogy	IV.C.11
1121	1,1-Dichloro-1-nitroethane	594-72-9	Analogy	IV.C.11
1129	1,3-Dichloropropene	542-75-6	Liver and kidney effects	IV.C.4
1130	2,2-Dichloropropionic acid	75-99-0	Sensory irritation	IV.C.3
2060	Dichlorotetrafluoroethane	76-14-2	Cardiovascular effects	IV.C.7
1131	Dicrotophos (bidrin)	141-66-2	Biochemical/metabolic effects	IV.C.12
1132	Dicyclopentadiene	77-73-6	Liver and kidney effects	IV.C.4
1133	Dicyclopentadienyl iron	102-54-5	Physical irritation	IV.C.10
2061	Dieldrin	60-57-1	Systemic toxicity	IV.C.8
1134	Diethanolamine	111-42-2	Noael	IV.C.9
2062	Diethlaminoethanol	100-37-8	Sensory irritation	IV.C.3
1135	Diethyl ketone	96-22-0	Analogy	IV.C.11
1136	Diethylphthalate	84-66-2	Noael	IV.C.9
1137	Diethylamine	109-89-7	Sensory irritation	IV.C.3
1138	Diethylene triamine	111-40-0	Analogy	IV.C.11
2063	Difluorodibromomethane	75-61-6	Systemic toxicity	IV.C.8
1139	Diglycidyl ether (DGE)	2238-07-5	Systemic toxicity	IV.C.8
1140	Diisobutyl ketone	108-83-8	Sensory irritation	IV.C.3
2064	Diisopropylamine	108-18-9	Systemic toxicity	IV.C.8
2065	Dimethyl acetamide	127-19-5	Liver and kidney effects	IV.C.4
2066	Dimethylamine	124-40-3	Sensory irritation	IV.C.3
1141	Dimethyl 1,2-dibromo-2, 2-Di-chloro-ethyl phosphate	300-76-5	Change in skin designation only	IV.C.16
2067	Dimethylformamide	68-12-2	Systemic toxicity	IV.C.8
2068	1,1-Dimethylhydrazine	57-14-7	Systemic toxicity	IV.C.8
2069	Dimethyl phthalate	131-11-3	Sensory irritation	IV.C.3
1142	Dimethyl sulfate	77-78-1	Cancer	IV.C.14
1143	Dimethylaniline	121-69-7	Biochemical/metabolic effects	IV.C.12
1144	Dinitolmide (3,5-Dinitro-o-tolu-amide)	148-01-6	Noael	IV.C.9
2071	Dinitrobenzene (Ortho) (meta) (para)	528-29-9; 99-65-0; 100-25-4	Analogy	IV.C.11
2070	Dinitro-o-cresol	534-52-1	Systemic toxicity	IV.C.8
2072	Dinitrotoluene	25321-14-6	Analogy	IV.C.11
1145	Dioxane (diethylene dioxide)	123-91-1	Liver and kidney effects	IV.C.4
1146	Dioxathion (delnav)	78-34-2	Biochemical/metabolic effects	IV.C.12
1147	Diphenylamine	122-39-4	Noael	IV.C.9
1148	Dipropyl ketone	123-19-3	Analogy	IV.C.11
1149	Dipropylene glycol methyl ether	34590-94-8	Neuropathy	IV.C.1
1150	Diquat	85-00-7	Analogy	IV.C.11
1151	Disulfiram	97-77-8	Biochemical/metabolic effects	IV.C.12
1152	Disulfoton	298-04-4	Analogy	IV.C.11
1153	Diuron	330-54-1	Noael	IV.C.9
1154	Divinyl benzene	1321-74-0	Analogy	IV.C.11
1155	Emery	112-62-9	Physical irritation	IV.C.10
1156	Endosulfan	115-29-7	Analogy	IV.C.11
2073	Endrin	72-20-8	Noael	IV.C.9
1158	Epichlorohydrin	106-89-8	Sensory irritation	IV.C.3
2074	EPN	2104-64-5	Analogy	IV.C.11
1159	Ethanolamine	141-43-5	Systemic toxicity	IV.C.8
1160	Ethion (nialate)	563-12-2	Biochemical/metabolic effects	IV.C.12
2075	Ethyl acetate	141-78-6	Sensory irritation	IV.C.3
1161	Ethyl acrylate	140-88-5	Respiratory effects	IV.C.6
2076	Ethyl alcohol	64-17-5	Narcosis	IV.C.2
2081	Ethylamine	75-04-7	Sensory irritation	IV.C.3
2080	Ethyl sec-amyl ketone	541-85-5	Sensory irritation	IV.C.3
2077	Ethyl butyl ketone	106-35-4	Sensory irritation	IV.C.3
1162	Ethyl benzene	100-41-4	Sensory irritation	IV.C.3
1163	Ethyl bromide	74-96-4	Narcosis	IV.C.2
2078	Ethyl chloride	75-00-3	Narcosis	IV.C.2
2082	Ethylenediamine	107-15-3	Systemic toxicity	IV.C.8
2083	Ethylene oxide	75-21-8	Cancer	IV.C.14
1164	Ethyl ether	60-29-7	Sensory irritation	IV.C.3
2079	Ethyl formate	109-94-4	Sensory irritation	IV.C.3

TABLE I.G.—INDEX TO PREAMBLE DISCUSSION OF INDIVIDUAL SUBSTANCES—Continued

H.S. No.	Substance name	CAS No.	Primary basis for limits	Preamble section
1165	Ethyl mercaptan	75-08-1	Sensory irritation	IV.C.3
1166	Ethyl silicate	78-10-4	Liver and kidney effects	IV.C.4
1167	Ethylene chlorohydrin	107-07-3	Systemic toxicity	IV.C.8
1168	Ethylene dichloride (1,2-dichloro-ethane)	107-06-2	Liver and kidney effects	IV.C.4
1169	Ethylene glycol	107-21-1	Sensory irritation	IV.C.3
1170	Ethylene glycol dinitrate	628-96-6	Cardiovascular effects	IV.C.7
1171	Ethylidene norbornene	16219-75-3	Sensory irritation	IV.C.3
1172	N-ethylmorpholine	100-74-3	Ocular effects	IV.C.5
1173	Fenamiphos	22224-92-6	Biochemical/metabolic effects	IV.C.12
1174	Fensulfothion (dasanit)	115-90-2	Biochemical/metabolic effects	IV.C.12
1175	Fenthion	55-38-9	Biochemical/metabolic effects	IV.C.12
1176	Ferbam	14484-64-1	Physical irritation	IV.C.10
1177	Ferrovanadium dust	12604-58-9	Respiratory effects	IV.C.6
1178	Fibrous glass dust	None	Respiratory effects	IV.C.6
2084	Fluoride (as f)	Varies	Systemic toxicity	IV.C.8
1179	Fluorine	7782-41-4	Sensory irritation	IV.C.3
1180	Fluorotrichloromethane	75-69-4	Cardiovascular effects	IV.C.7
1181	Fonofos	944-22-9	Analogy	IV.C.11
2085	Formaldehyde	50-00-0	Cancer	IV.C.14
1182	Formamide	75-12-7	Analogy	IV.C.11
2080	Formic acid	64-18-6	Sensory irritation	IV.C.3
1183	Furfural	98-01-1	Sensory irritation	IV.C.3
1184	Furfuryl alcohol	98-00-0	Sensory irritation	IV.C.3
1185	Gasoline	8006-61-9	Narcosis	IV.C.2
1186	Germanium tetrahydride	7782-65-2	Analogy	IV.C.11
1187	Glutaraldehyde	111-30-8	Sensory irritation	IV.C.3
1188	Glycerin (mist)	56-81-5	Physical irritation	IV.C.10
1189	Glycidol (2,3-epoxy-1-propanol)	556-52-5	Systemic toxicity	IV.C.8
1190	Grain dust	None	Respiratory effects	IV.C.6
1191	Graphite, natural (containing <1% quartz)	7782-42-5	Respiratory effects	IV.C.6
1191A	Graphite, synthetic (containing <1% quartz)	None	Physical irritation	IV.C.10
1192	Gypsum, total dust	13397-24-5	Physical irritation	IV.C.10
2087	Hafnium	7440-58-6	Liver and kidney effects	IV.C.4
2088	Heptachlor	76-44-8	Systemic toxicity	IV.C.8
1194	N-heptane	142-82-5	Narcosis	IV.C.2
1195	Hexachlorobutadiene	87-68-3	Liver and kidney effects	IV.C.4
1196	Hexachlorocyclopentadiene	77-47-4	Sensory irritation	IV.C.3
1197	Hexachloroethane	67-72-1	Sensory irritation	IV.C.3
2089	Hexachloronaphthalene	1335-87-1	Liver and kidney effects	IV.C.4
1198	Hexafluoroacetone	684-16-2	Systemic toxicity	IV.C.8
1200	N-hexane	110-54-3	Neuropathy	IV.C.1
1201	Hexane isomers	Varies	Narcosis	IV.C.2
1202	2-hexanone	591-78-6	Neuropathy	IV.C.1
1203	Hexone (methyl isobutyl ketone)	108-10-1	Liver and kidney effects	IV.C.4
2090	Sec-hexyl acetate	108-84-9	Sensory irritation	IV.C.3
1204	Hexylene glycol	107-41-5	Sensory irritation	IV.C.3
1205	Hydrazine	302-01-2	Liver and kidney effects	IV.C.4
1206	Hydrogen bromide	10035-10-6	Sensory irritation	IV.C.3
2091	Hydrogen chloride	7647-01-0	Sensory irritation	IV.C.3
1207	Hydrogen cyanide	74-90-8	Systemic toxicity	IV.C.8
1208	Hydrogen fluoride	7664-39-3	Sensory irritation	IV.C.3
2092	Hydrogen peroxide (90%)	7722-84-1	Sensory irritation	IV.C.3
2093	Hydrogen selenide (As Se)	7783-07-5	Systemic toxicity	IV.C.8
1209	Hydrogen sulfide	7783-06-4	Ocular effects	IV.C.5
1210	Hydrogenated terphenyls	61788-32-7	Systemic toxicity	IV.C.8
2094	Hydroquinone	123-31-9	Ocular effects	IV.C.5
1211	2-Hydroxypropyl acrylate	999-61-1	Sensory irritation	IV.C.3
1212	Indene	95-13-6	Analogy	IV.C.11
1213	Indium & compounds	7440-74-6	Respiratory effects	IV.C.6
2095	Iodine	7553-56-2	Sensory irritation	IV.C.3
1214	Iodoform	75-47-8	Analogy	IV.C.11
1215	Iron oxide (dust and fume)	1309-37-1	Respiratory effects	IV.C.6
1216	Iron pentacarbonyl	13463-40-6	Neuropathy	IV.C.1
1217	Iron salts (soluble)	Varies	Sensory irritation	IV.C.3
2096	Isoamyl acetate	123-92-2	Sensory irritation	IV.C.3
1218	Isoamyl alcohol	123-51-3	Narcosis	IV.C.2
2097	Isobutyl acetate	110-19-0	Analogy	IV.C.11
1219	Isobutyl alcohol	78-83-1	Analogy	IV.C.11
1220	Isooctyl alcohol	26952-21-6	Analogy	IV.C.11
1221	Isophorone	78-59-1	Narcosis	IV.C.2
1222	Isophorone diisocyanate	4098-71-9	Sensitization effects	IV.C.13
1223	2-Isopropoxyethanol	109-59-1	Systemic toxicity	IV.C.8
1224	Isopropyl acetate	108-21-4	Sensory irritation	IV.C.3
1225	Isopropyl alcohol	67-63-0	Sensory irritation	IV.C.3
1226	Isopropyl ether	108-20-3	Sensory irritation	IV.C.3
1227	Isopropyl glycidyl ether	4016-14-2	Systemic toxicity	IV.C.8
1228	Isopropylamine	75-31-0	Sensory irritation	IV.C.3
1229	N-Isopropylaniline	768-52-5	Analogy	IV.C.11
1230	Kaolin, total dust	1332-58-7	Physical irritation	IV.C.10

TABLE I.G.—INDEX TO PREAMBLE DISCUSSION OF INDIVIDUAL SUBSTANCES—Continued

H.S. No.	Substance name	CAS No.	Primary basis for limits	Preamble section
1231	Ketene	463-51-4	Analogy	IV.C.11
2098	Lead, inorganic	7439-92-1	Systemic toxicity	IV.C.8
1232	Limestone, total dust	1317-65-3	Physical irritation	IV.C.10
2100	Lindane	58-89-9	Neuropathy	IV.C.1
2101	Lithium hydride	7580-67-8	Sensory irritation	IV.C.3
2099	L.P.G. (liquefied petroleum gas)	68476-85-7	Noael	IV.C.9
1233	Magnesite, total dust	546-93-0	Physical irritation	IV.C.10
1234	Magnesium oxide fume*	1309-48-4	Physical irritation	IV.C.10
1235	Malathion*	121-75-5	Physical irritation	IV.C.10
2102	Maleic anhydride	108-31-6	Sensory irritation	IV.C.3
2103	Manganese dust and compounds	7439-96-5	Neuropathy	IV.C.1
1236A	Manganese, fume	7439-96-5	Neuropathy	IV.C.1
1237	Manganese cyclopentadienyl tricarbonyl	12079-65-1	Neuropathy	IV.C.1
1238	Manganese tetroxide	1317-35-7	Neuropathy	IV.C.1
1239	Marble, total dust	1317-65-3	Physical irritation	IV.C.10
1240	Mercury (aryl and inorganic compounds)	Varies	Neuropathy	IV.C.1
1241	Mercury (vapor)	7439-97-6	Neuropathy	IV.C.1
1242	Mercury, (organo) alkyl compounds	Varies	Neuropathy	IV.C.1
1243	Mesityl oxide	141-79-7	Sensory irritation	IV.C.3
1244	Methacrylic acid	79-41-4	Analogy	IV.C.11
1245	Methomyl (lannate)	16752-77-5	Biochemical/metabolic effects	IV.C.12
1246	Methoxychlor*	72-43-5	Physical irritation	IV.C.10
1247	4-Methoxyphenol	150-76-5	Analogy	IV.C.11
1249	Methyl acetate	79-20-9	Noael	IV.C.9
2104	Methyl acetylene (propyne)	74-99-7	Neuropathy	IV.C.1
1250	Methyl acetylene/propadiene mixture	None	Analogy	IV.C.11
2105	Methyl acrylate	96-33-3	Sensory irritation	IV.C.3
1251	Methyl acrylonitrile	126-98-7	Neuropathy	IV.C.1
2106	Methylal (dimethoxymethane)	109-87-5	Analogy	IV.C.11
2110	Methylamine	74-89-5	Analogy	IV.C.11
1252	Methyl alcohol	67-56-1	Ocular effects	IV.C.5
1253	Methyl bromide	74-83-9	Neuropathy	IV.C.1
1254	Methyl chloride	74-87-3	Narcosis	IV.C.2
1255	Methyl chloroform (1,1,1-trichloro-ethane)	71-55-6	Narcosis	IV.C.2
1248	Methyl 2-cyanoacrylate	137-05-3	Sensory irritation	IV.C.3
1256	Methyl demeton	8022-00-2	Analogy	IV.C.11
1257	Methyl ethyl ketone peroxide	1338-23-4	Analogy	IV.C.11
1258	Methyl formate	107-31-3	Analogy	IV.C.11
1259	Methyl iodide	74-88-4	Analogy	IV.C.11
1260	Methyl isoamyl ketone	110-12-3	Analogy	IV.C.11
1261	Methyl isobutyl carbinol	108-11-2	Sensory irritation	IV.C.3
2106	Methyl isocyanate	624-83-9	Sensory irritation	IV.C.3
1262	Methyl isopropyl ketone	563-80-4	Analogy	IV.C.11
1263	Methyl mercaptan	74-93-1	Sensory irritation	IV.C.3
2107	Methyl methacrylate	80-62-6	Sensory irritation	IV.C.3
1264	Methyl N-amyl ketone	110-43-0	Sensory irritation	IV.C.3
1265	Methyl parathion	298-00-0	Analogy	IV.C.11
1266	Methyl silicate	681-84-5	Ocular effects	IV.C.5
1267	Alpha-methyl styrene	98-83-9	Sensory irritation	IV.C.3
2108	Methylene bisphenyl isocyanate (MDI)	101-68-8	Analogy	IV.C.11
1268	Methylcyclohexane	108-87-2	Analogy	IV.C.11
1269	Methylcyclohexanol	25639-42-3	Liver and kidney effects	IV.C.4
1270	O-Methylcyclohexanone	583-60-8	Sensory irritation	IV.C.3
1271	Methylcyclopentadienyl MN tricarbonyl	12108-13-3	Analogy	IV.C.11
1273	4,4'-Methylene bis (2-chloro-aniline) (MBOCA)	101-14-4	Systemic toxicity	IV.C.8
1272	Methylene bis (4-cyclo-hexyli-socyanate)	5124-30-1	Respiratory effects	IV.C.6
1275	Metribuzin	21087-64-9	Noael	IV.C.9
1276	Mica	12001-26-2	Respiratory effects	IV.C.6
1277	Mineral wool fiber	None	Respiratory effects	IV.C.6
2111	Molybdenum (soluble compounds)	7439-98-7	Systemic toxicity	IV.C.8
1278	Molybdenum (insoluble compounds)	7439-98-7	Physical irritation	IV.C.10
1279	Monocrotophos (azodrin)	6923-22-4	Analogy	IV.C.11
1280	Monomethyl aniline	100-61-8	Biochemical/metabolic effects	IV.C.12
2112	Monomethyl hydrazine	60-34-4	Systemic toxicity	IV.C.8
1281	Morpholine	110-91-8	Analogy	IV.C.11
2113	Naphtha (coal tar)	8030-30-6	Narcosis	IV.C.2
1282	Naphthalene	91-20-3	Ocular effects	IV.C.5
2114	Nickel (metal)	7440-02-0	Systemic toxicity	IV.C.8
1283	Nickel (soluble compounds)	Varies	Respiratory effects	IV.C.6
2115	Nicotine	54-11-5	Systemic toxicity	IV.C.8
1286	Nitric acid	7697-37-2	Analogy	IV.C.11
2116	Nitric oxide	10102-43-9	Biochemical/metabolic effects	IV.C.12
1287	P-Nitroaniline	100-01-6	Analogy	IV.C.11
2117	Nitrobenzene	98-95-3	Biochemical/metabolic effects	IV.C.12
1288	P-Nitrochlorobenzene	100-00-5	Biochemical/metabolic effects	IV.C.12
2118	Nitroethane	79-24-3	Liver and kidney effects	IV.C.4
1289	Nitrogen dioxide	10102-44-0	Respiratory effects	IV.C.6
2119	Nitrogen trifluoride	7783-54-2	Liver and kidney effects	IV.C.4
1290	Nitroglycerin	55-63-0	Cardiovascular effects	IV.C.7

TABLE I.G.—INDEX TO PREAMBLE DISCUSSION OF INDIVIDUAL SUBSTANCES—Continued

H.S. No.	Substance name	CAS No.	Primary basis for limits	Preamble section
2120	Nitromethane	75-52-5	Analogy	IV.C.11
2121	1-Nitropropane	108-03-2	Noael	IV.C.9
1291	2-Nitropropane	79-46-9	Cancer	IV.C.14
1292	Nitrotoluene (all isomers)	99-08-1; 99-99-0; 88-72-2	Analogy	IV.C.11
1293	Nonane	111-84-2	Analogy	IV.C.11
1295	Octachloronaphthalene	2234-13-1	Liver and kidney effects	IV.C.4
1296	Octane	111-65-9	Narcosis	IV.C.2
1297	Oil mist (mineral)	8012-95-1	Noael	IV.C.9
1298	Osmium tetroxide	20816-12-0	Sensory irritation	IV.C.3
1299	Oxalic acid	144-62-7	Analogy	IV.C.11
1300	Oxygen difluoride	7783-41-7	Respiratory effects	IV.C.6
1301	Ozone	10028-15-6	Respiratory effects	IV.C.6
1302	Paraffin wax fume	8002-74-2	Sensory irritation	IV.C.3
1303	Paraquat, respirable dust	4685-14-7	Respiratory effects	IV.C.6
2122	Parathion	56-38-2	Biochemical/metabolic effects	IV.C.12
1294	Particulates not otherwise regulated	None	Physical irritation	IV.C.10
1304	Pentaborane	19624-22-7	Neuropathy	IV.C.1
2123	Pentachloronaphthalene	1321-64-8	Liver and kidney effects	IV.C.4
2124	Pentachlorophenol	87-86-5	Cardiovascular effects	IV.C.7
1305	Pentaerythritol, total dust	115-77-5	Physical irritation	IV.C.10
1306	Pentane	109-66-0	Narcosis	IV.C.2
1307	2-pentanone (methyl propyl ketone)	107-87-9	Narcosis	IV.C.2
1308	Perchloroethylene	127-18-4	Cancer	IV.C.14
2125	Perchloromethyl mercaptan	594-42-3	Systemic toxicity	IV.C.8
1309	Perchloryl fluoride	7616-94-6	Analogy	IV.C.11
1310	Perlite	93763-70-3	Physical irritation	IV.C.10
1312	Petroleum distillates (naphtha)	None	Noael	IV.C.9
2126	Phenol	108-95-2	Systemic toxicity	IV.C.8
1313	Phenothiazine	92-84-2	Sensitization effects	IV.C.13
2128	P-phenylene diamine	106-50-3	Sensitization effects	IV.C.13
1314	Phenyl ether (vapor)	101-84-8	Sensory irritation	IV.C.3
2127	Phenyl ether-biphenyl mixture	None	Sensory irritation	IV.C.3
1315	Phenyl glycidyl ether	122-60-1	Sensitization effects	IV.C.13
1316	Phenyl mercaptan	108-98-5	Neuropathy	IV.C.1
1317	Phenylhydrazine	100-63-0	Systemic toxicity	IV.C.8
1318	Phenylphosphine	638-21-1	Systemic toxicity	IV.C.8
1319	Phorate (thimet)	298-02-2	Biochemical/metabolic effects	IV.C.12
2129	Phosgene	75-44-5	Sensory irritation	IV.C.3
1320	Phosdrin (mevinphos)	7786-34-7	Analogy	IV.C.11
1321	Phosphine	7803-51-2	Systemic toxicity	IV.C.8
1322	Phosphoric acid	7664-38-2	Sensory irritation	IV.C.3
2130	Phosphorus (yellow)	7723-14-0	Systemic toxicity	IV.C.8
1323	Phosphorus oxychloride	10025-87-3	Analogy	IV.C.11
2131	Phosphorus pentachloride	10026-13-8	Sensory irritation	IV.C.3
1324	Phosphorus pentasulfide	1314-80-3	Analogy	IV.C.11
1325	Phosphorus trichloride	7719-12-2	Sensory irritation	IV.C.3
1326	Phthalic anhydride	85-44-9	Analogy	IV.C.11
1327	M-phthalodinitrile	626-17-5	Noael	IV.C.9
1328	Picloram (tordom)	1918-02-1	Physical irritation	IV.C.10
1329	Picric acid	88-89-1	Sensitization effects	IV.C.13
1330	Piperazine dihydrochloride	142-64-3	Systemic toxicity	IV.C.8
2132	Pindone	83-26-1	Analogy	IV.C.11
1331	Plaster of paris, total dust	7778-18-9	Physical irritation	IV.C.10
1332	Platinum, metal	7440-06-4	Noael	IV.C.9
2133	Platinum (soluble salts)	7440-06-4	Sensitization effects	IV.C.13
1333	Portland cement	65997-15-1	Physical irritation	IV.C.10
1334	Potassium hydroxide	1310-58-3	Sensory irritation	IV.C.3
2134	Propane	74-98-6	Systemic toxicity	IV.C.8
1335	Propargyl alcohol	107-19-7	Analogy	IV.C.11
1336	Propionic acid	79-09-4	Analogy	IV.C.11
1337	Propoxur (baygon)	114-26-1	Biochemical/metabolic effects	IV.C.12
1338	N-propyl acetate	109-60-4	Analogy	IV.C.11
1339	Propyl alcohol	71-23-8	Analogy	IV.C.11
1340	N-propyl nitrate	627-13-4	Systemic toxicity	IV.C.8
1341	Propylene dichloride	78-87-5	Liver and kidney effects	IV.C.4
1342	1,2-propylene glycol dinitrate	6423-43-4	Neuropathy	IV.C.1
1343	Propylene glycol monomethyl ether	107-98-2	Sensory irritation	IV.C.3
2135	Propyleneimine	75-55-8	Analogy	IV.C.11
1344	Propylene oxide	75-56-9	Analogy	IV.C.11
2136	Pyrethrum	8003-34-7	Systemic toxicity	IV.C.8
2137	Pyridine	110-86-1	Systemic toxicity	IV.C.8
2138	Quinone (P-benzoquinone)	106-51-4	Ocular effects	IV.C.5
1346	Resorcinol	108-46-3	Noael	IV.C.9
1347	Rhodium (metal, fume & insoluble compounds)	7440-16-6	Sensitization effects	IV.C.13
1348	Rhodium (soluble salts)	Varies	Sensitization effects	IV.C.13
1349	Ronnel	299-84-3	Biochemical/metabolic effects	IV.C.12
1350	Rosin core solder pyrolysis product (as hcho)	None	Sensory irritation	IV.C.3

TABLE I.G.—INDEX TO PREAMBLE DISCUSSION OF INDIVIDUAL SUBSTANCES—Continued

H.S. No.	Substance name	CAS No.	Primary basis for limits	Preamble section
2139	Rotenone (commercial)	83-79-4	Noael	IV.C.9
1351	Rouge, total dust	None	Physical irritation	IV.C.10
2140	Selenium and compounds	7782-49-2	Systemic toxicity	IV.C.8
2141	Selenium hexafluoride	7783-79-1	Analogy	IV.C.11
1352	Silica, amorphous, diatomaceous earth	61790-53-2	Respiratory effects	IV.C.6
1353	Silica, amorphous, precipitated and gel	None	Respiratory effects	IV.C.6
1354	Silica, crystalline-cristobalite	14464-46-1	Respiratory effects	IV.C.6
1355	Silica, crystalline quartz, respirable	14808-60-7	Respiratory effects	IV.C.6
2142	Silica, crystalline quartz, total dust	14808-60-7	Respiratory effects	IV.C.6
1356	Silica, crystalline tridymite	15468-32-3	Respiratory effects	IV.C.6
1357	Silica, crystalline tripoli (as quartz dust)	1317-95-9	Respiratory effects	IV.C.6
1358	Silica, fused	60676-86-0	Respiratory effects	IV.C.6
1359	Silicon	7440-21-3	Physical irritation	IV.C.10
1360	Silicon carbide	409-21-2	Physical irritation	IV.C.10
1361	Silicon tetrahydride	7803-62-5	Analogy	IV.C.11
1362	Silver, metal and soluble compounds	7440-22-4	Systemic toxicity	IV.C.8
1363	Soapstone, total dust	None	Respiratory effects	IV.C.6
1363A	Soapstone, respirable dust	None	Respiratory effects	IV.C.6
1364	Sodium azide (as HN3), (as NaN3)	26628-22-8	Cardiovascular effects	IV.C.7
1365	Sodium bisulfite	7631-90-5	Sensory irritation	IV.C.3
1366	Sodium fluoroacetate	62-74-8	Systemic toxicity	IV.C.8
1367	Sodium hydroxide	1310-73-2	Sensory irritation	IV.C.3
1368	Sodium metabisulfite	7681-57-4	Sensory irritation	IV.C.3
1369	Starch, total dust	9005-25-8	Physical irritation	IV.C.10
2144	Stibine	7803-52-3	Analogy	IV.C.11
1371	Stoddard solvent	8052-41-3	Narcosis	IV.C.2
2145	Strychnine	57-24-9	Systemic toxicity	IV.C.8
1372	Styrene (phenylethylene)	100-42-5	Narcosis	IV.C.2
1373	Subtilisins (proteolytic enzymes)	1395-21-7	Sensitization effects	IV.C.13
1374	Sucrose, total dust	57-50-1	Physical irritation	IV.C.10
1375	Sulfur dioxide	7446-09-5	Respiratory effects	IV.C.6
2146	Sulfur hexafluoride	2551-62-4	Noael	IV.C.9
2147	Sulfuric acid	7664-93-9	Sensory irritation	IV.C.3
1376	Sulfur monochloride	10025-67-9	Sensory irritation	IV.C.3
1377	Sulfur pentafluoride	5714-22-7	Sensory irritation	IV.C.3
1378	Sulfur tetrafluoride	7783-60-0	Respiratory effects	IV.C.6
1379	Sulfuryl fluoride	2699-79-8	Analogy	IV.C.11
1380	Sulprofos	35400-43-2	Biochemical/metabolic effects	IV.C.12
2148	2,4,5-T	93-76-5	Physical irritation	IV.C.10
1381	Talc (containing no asbestos)	14807-96-6	Respiratory effects	IV.C.6
1382	Tantalum	7440-25-7	Noael	IV.C.9
2149	TEDP (sulfotep)	3689-24-5	Analogy	IV.C.11
2150	Tellurium	13494-80-9	Systemic toxicity	IV.C.8
2151	Tellurium hexafluoride	7783-80-4	Analogy	IV.C.11
1383	Temephos	3383-96-8	Physical irritation	IV.C.10
2152	TEPP	107-49-3	Analogy	IV.C.11
1384	Terphenyls	26140-60-3	Biochemical/metabolic effects	IV.C.12
2153	1,1,2,2-Tetrachloro-1,2-difluoroethane	76-12-0	Noael	IV.C.9
2154	1,1,1,2-Tetrachloro-2,2-difluoroethane	76-11-9	Noael	IV.C.9
1385	1,1,2,2-Tetrachloroethane	79-34-5	Liver and kidney effects	IV.C.4
2155	Tetrachloronaphthalene	1335-88-2	Liver and kidney effects	IV.C.4
1386	Tetraethyl lead	78-00-2	Systemic toxicity	IV.C.8
1387	Tetrahydrofuran	109-99-9	Sensory irritation	IV.C.3
1388	Tetramethyl lead	75-74-1	Systemic toxicity	IV.C.8
2156	Tetramethyl succinonitrile	3333-52-6	Systemic toxicity	IV.C.8
2157	Tetranitromethane	509-14-8	Sensory irritation	IV.C.3
1389	Tetrasodium pyrophosphate	7722-88-5	Sensory irritation	IV.C.3
2158	Tetryl	479-45-8	Systemic toxicity	IV.C.8
2159	Thallium (soluble compounds)	7440-28-0	Analogy	IV.C.11
1391	4,4'-Thiobis (6-tert-butyl-m-cresol)	96-69-5	Physical irritation	IV.C.10
1392	Thioglycolic acid	68-11-1	Sensory irritation	IV.C.3
1393	Thionyl chloride	7719-09-7	Analogy	IV.C.11
2160	Thiram	137-26-8	Systemic toxicity	IV.C.8
2161	Tin (metal and inorganic compounds)	7440-31-5	Systemic toxicity	IV.C.8
1394	Tin (organic compounds)	Varies	Change in skin designation only	IV.C.16
1395	Tin oxide	21651-19-4	Respiratory effects	IV.C.6
1396	Titanium dioxide	13463-67-7	Physical irritation	IV.C.10
1397	Toluene	108-88-3	Narcosis	IV.C.2
1398	Toluene-2,4-diisocyanate	584-84-9	Sensitization effects	IV.C.13
1399	O-Toluidine	95-53-4	Cancer	IV.C.14
1400	P-Toluidine	106-49-0	Cancer	IV.C.14
1401	M-Toluidine	108-44-1	Biochemical/metabolic effects	IV.C.12
1402	Tributyl phosphate	126-73-8	Analogy	IV.C.11
1403	1,1,2-Trichloro-1,2, 2-Trifluoro-ethane	76-13-1	Cardiovascular effects	IV.C.7
1404	Trichloroacetic acid	76-03-9	Analogy	IV.C.11
1405	1,2,4-Trichlorobenzene	120-82-1	Sensory irritation	IV.C.3
2162	1,1,2-Trichloroethane	79-00-5	Analogy	IV.C.11
1406	Trichloroethylene	79-01-6	Narcosis	IV.C.2
2163	Trichloronaphthalene	1321-65-9	Liver and kidney effects	IV.C.4

TABLE I.G.—INDEX TO PREAMBLE DISCUSSION OF INDIVIDUAL SUBSTANCES—Continued

H.S. No.	Substance name	CAS No.	Primary basis for limits	Preamble section
1407	1,2,3-Trichloropropane	96-18-4	Liver and kidney effects	IV.C.4
1408	Triethylamine	121-44-8	Sensory irritation	IV.C.3
2164	Trifluoromonobromomethane	75-63-8	Noael	IV.C.9
1409	Trimellitic anhydride	552-30-7	Respiratory effects	IV.C.6
1410	Trimethyl phosphite	121-45-9	Noael	IV.C.9
1411	Trimethylamine	75-50-3	Analogy	IV.C.11
1412	Trimethylbenzene	25551-13-7	Systemic toxicity	IV.C.8
1413	2,4,6-Trinitrotoluene (TNT)	118-96-7	Biochemical/metabolic effects	IV.C.12
1414	Triorthocresyl phosphate	78-30-8	Change in skin designation only	IV.C.16
1415	Triphenyl amine	603-34-9	Noael	IV.C.9
2165	Triphenyl phosphate	115-86-6	Noael	IV.C.9
1416	Tungsten & compounds (insoluble)	7440-33-7	Systemic toxicity	IV.C.8
1417	Tungsten & compounds (soluble)	7440-33-7	Systemic toxicity	IV.C.8
2166	Turpentine	8006-64-2	Sensory irritation	IV.C.3
1418	Uranium (insoluble compounds)	7440-61-1	Noael	IV.C.9
1419	Uranium (soluble compounds)	7440-61-1	Liver and kidney effects	IV.C.4
1420	N-Valeraldehyde	110-62-3	Analogy	IV.C.11
1421	Vanadium (v205, dust)	1314-62-1	Sensory irritation	IV.C.3
1422	Vanadium (v205, fume)	1314-62-1	Sensory irritation	IV.C.3
1423	Vegetable oil mist	None	Physical irritation	IV.C.10
1424	Vinyl acetate	108-05-4	Sensory irritation	IV.C.3
1425	Vinyl bromide	593-60-2	Cancer	IV.C.14
2167	Vinyl chloride	75-01-4	Cancer	IV.C.14
1426	Vinyl cyclohexene dioxide	106-87-6	Cancer	IV.C.14
1427	Vinyl toluene	25013-15-4	Sensory irritation	IV.C.3
1428	Vinylidene chloride	75-35-4	Systemic toxicity	IV.C.8
1429	VM & P naphtha	8032-32-4	Sensory irritation	IV.C.3
2168	Warfarin	81-81-2	Biochemical/metabolic effects	IV.C.12
1430	Welding fumes (total particulate)	None	Systemic toxicity	IV.C.8
1430a	Wood dust, hard wood	None	Respiratory effects	IV.C.6
1430b	Wood dust, soft wood	None	Respiratory effects	IV.C.6
1430c	Wood dust, western red cedar	None	Respiratory effects	IV.C.6
1431	Xylene (O,M,P-isomers)	1330-20-7	Sensory irritation	IV.C.3
1432	M-xylene-alpha,alpha'-diamine	1477-55-0	Analogy	IV.C.11
1433	Xylidine	1300-73-8	Analogy	IV.C.11
2169	Yttrium	7440-65-5	Respiratory effects	IV.C.6
1434	Zinc stearate	557-05-1	Physical irritation	IV.C.10
1435	Zinc chloride fume	7646-85-7	Sensory irritation	IV.C.3
1436	Zinc chromates (CrVI)	Varies	Cancer	IV.C.14
1437	Zinc oxide (fume)	1314-13-2	Systemic toxicity	IV.C.8
1438	Zinc oxide, total dust	1314-13-2	Physical irritation	IV.C.10
1439	Zirconium compounds	Varies	Systemic toxicity	IV.C.8

H. Mixture Formula

The current OSHA PELs for general industry, construction, and maritime are covered by a mixture or computation formula. It is located at 29 CFR 1910.1000(d) for general industry. In construction it is incorporated by cross reference to the 1970 ACGIH TLVs. Maritime includes the mixture formula by cross reference either to § 1910.1000 or to the 1970 ACGIH TLVs.

The purpose of the mixture formula is to protect the health of workers exposed to two or more toxic substances. If a worker is exposed to two or more toxic substances which affect the same organ or cause the same disease or material health impairment, being exposed at the maximum permissible exposure limit for each substance will often not protect the worker from material health impairment.

Consequently, in these circumstances the mixture formula keeps exposure below the maximum for each of the several substances the workers are exposed to. For example, if a worker is exposed to 50% of the PEL for a substance that causes cardiovascular disease, then that worker can be exposed to no more than 50% of the PEL of a second substance which causes cardiovascular disease. The mixture formula is only applied by OSHA when the substances affect the same organ, or cause the same disease or material health impairment.

OSHA has requested that the White House's Federal Coordination Council for Science, Engineering and Technology (FCCSET) give advice on the use of the mixture formula. The Council will hold a conference in December on the issue. Both EPA and FDA are interested in this issue and will participate. Depending on the Council's advice, OSHA may open the mixture formula for reconsideration in the first update of the PELs for all industry sectors. It would be premature to consider this issue before receiving the Council's advice and inefficient to consider it for one industry sector at a time since the biology, of course, is the same for all workers.

OSHA is proposing to include the mixture formula for agriculture because of the health reasons stated above, its long acceptance in occupational health practice, and for consistency among the sectors. OSHA is not opening the issue of the mixture formula for construction and maritime since it already applies and would be premature prior to the Council's recommendations.

II. Pertinent Legal Authority

The publication of this standard is authorized by sections 6 and 8 of the Occupational Safety and Health Act of 1970 (the Act), 29 U.S.C. 655 and 657. Section 6(b)(5) governs the issuance of occupational safety and health standards dealing with toxic materials or harmful physical agents.

It states:

> The Secretary in promulgating standards dealing with toxic materials or harmful physical agents under this subsection shall set the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee

will suffer material impairment of health or functional capacity even if such employee has regular exposure to the hazard dealt with by such standard for the period of his working life. Development of standards under this subsection shall be based upon research, demonstrations, experiments, and such other information as may be appropriate. In addition to the attainment of the highest degree of health and safety protection for the employee, other considerations shall be the latest available scientific data in the field, the feasibility of standards, and experience gained under this and other health and safety laws. Whenever practicable, the standard promulgated shall be expressed in terms of objective criteria and of the performance desired.

Section 3(8) defines an occupational safety and health standard as "a standard which requires conditions, or the adoption or use of one or more practice, means, methods, operations, or processes, reasonably necessary or appropriate to provide safe or healthful employment and places of employment."

The Supreme Court has held under the Act that the Secretary, before issuing any new standard, must determine that it is reasonably necessary and appropriate to remedy a significant risk of material health impairment. *Industrial Union Department* v. *American Petroleum Institute, (IUD* v. *API).* 488 U.S. 607 (1980). The Court stated that "before he can promulgate any permanent health or safety standard, the Secretary is required to make a threshold finding that a place of employment is unsafe in the sense that significant risks are present and can be eliminated or lessened by a change in practices" (448 U.S. at 642). The Court also stated "that the Act does limit the Secretary's power to require the elimination of significant risk" (488 U.S. 644, n. 49).

The Court indicated, however that the significant risk determination is "not a mathematical straitjacket," and that "OSHA is not required to support its finding that a significant risk exists with anything approaching scientific certainty." The Court ruled that "a reviewing court [is] to give OSHA some leeway where its findings must be made on the frontiers of scientific knowledge [and that] * * * the Agency is free to use conservative assumptions in interpreting the data with respect to carcinogens, risking error on the side of over protection rather than under protection" (448 U.S. at 655, 655).

The Court also stated that "while the Agency must support its finding that a certain level of risk exists with substantial evidence, we recognize that its determination that a particular level of risk is 'significant' will be based largely on policy consideration." (488 U.S. at 655, n. 62).

After OSHA determines that a significant risk exists and that such risk can be reduced or eliminated by the proposed standard, it must set a standard which is technologically and economically feasible. In *American Textile Manufacturers Institute* v. *Donovan,* 542 U.S. 490, 531, n. 32 (1981) the Supreme Court held that "cost-benefit analysis is not required by statue because feasibility analysis is." The aim is to set the lowest feasible level necessary to eliminate significant risk.

Congress intended the OSH Act to be very broad in coverage. Section (2)(b) of the act states:

"The Congress declares it to be its purpose and policy * * * to assure so far as possible every working man and woman in the nation safe and healthful working conditions * * *"

Clearly this indicates that OSHA should cover working men and women in all sections of the economy—general industry, construction, maritime and agriculture, and indeed OSHA has set standards for all those sectors.

When OSHA proposed and issued its updated exposure limits for general industry it stated that it would expand the new limits to cover the construction, maritime and agriculture sectors. See 53 FR 20979; June 7, 1988 and 54 FR 2383; January 19, 1989. The reason that OSHA did not propose to immediately cover those sectors was the time it would take to consult with those sectors and to perform feasibility analyses for these sectors.

Shortly after the completion of the updated PEL's for general industry OSHA commenced those activities. It consulted with the Construction Advisory Committee (CACOSH) and the Shipyard Advisory Committee. It held a series of meetings with the agricultural community. Also feasibility analyses were commenced for these sectors. These activities are discussed above and in the feasibility section of the preamble and the consultations and information have been taken into account in this proposal.

Construction

Federal regulation of construction activities was covered prior to OSHA by the Construction Safety Act (Pub. L. 91–54, August 23, 1969, 33 U.S.C. 941). That Act gives the Secretary of Labor authority to issue and enforce regulation to protect the health and safety of employees who worked for contractors performing construction work for the U.S. government. It also provided for the creation of an advisory committee on such regulations.

The Department of Labor issued regulations under the Construction Safety Act on April 17, 1971 at 36 FR 7340. Those regulations included at 29 CFR 1518.55 requirements that the workers of construction contractors for the U.S. government not be exposed over the Threshold Limit Values for 1970 of the ACGIH. That requirement became an occupational safety and health standard for all construction workers under the OSH Act pursuant to Section 4(b)(2) on April 28, 1971. It was later recodified at 29 CFR 1926.55 on June 24, 1974 at 39 FR 22801. The large majority of exposure limits applicable to construction employers are the 1970 TLV's.

Many of the single substance standards which OSHA has issued have been made applicable to the construction industry. See 29 CFR 1910.19 and the individual standards.

OSHA's procedural rules for issuing standards under the OSH Act state that OSHA shall consult with the Construction Advisory Committee. See 29 CFR 1911.10. OSHA has done so in this case and the Committee has advised OSHA to proceed. See the discussion above under History and need for the Standard in Construction.

Maritime

The Department of Labor has had some authority for many years for the maritime industry under the Longshore and Harbor Workers Compensation Act (33 U.S.C. 901 *et seq.*). Specific authority was granted prior to the OSH Act under Public Law 89–742, August 22, 1958, 33 U.S.C. 941 for the Secretary of Labor to issue regulations to protect the health and safety of longshoremen, marine terminal workers, ship repairers, shipbuilders and ship breakers (See also 33 U.S.C. 902). Pursuant to section 4(b)(2) of the OSH Act, standards issued pursuant to 33 U.S.C. 941 became OSH standards on May 29, 1971 at 36 FR 10400.

At that time the Shipyard standards were in three parts of 29 CFR, part 1915 for ship repairing, part 1916 for shipbuilding and part 1917 for shipbreaking. These were recodified with the same basic organization on October 19, 1977 at 37 FR 22458.

On April 20, 1982 at 47 FR 16984, parts 1915, 1916 and 1917 were consolidated into a new part 1915, Shipyards, covering shipbuilding, ship repairing and shipbreaking. As a consequence of their history, the permissible exposure limits applicable to the new part 1915, Shipyards are complex. The shipbreaking and ship repairing subsectors and the use of toxic solvents

and removers in the shipbuilding and ship repair subsectors are covered by the 1970 TLV's of the ACGIH. See §§ 1915.5, 1915.11, 1915.12, 1915.32 and 1915.33. For shipbuilding operations not involving toxic solvents and removers, the 1971 OSHA PEL's apply. No specific exposure limits are specified for shipbuilding outside those 2 circumstances. When no standard is specified for a subsector, and there is a standard for general industry, then the general industry standard applies to the subsector—See 29 CFR 1910.5(c). The OSHA general industry standards for air contaminants are the Z Tables in 29 CFR 1910.1000. OSHA did not make the updated (1989) PELs applicable to maritime awaiting the completion of this rulemaking. See 29 CFR 1910.1000(f)(3)(ii). In addition a number of OSHA single substance standards (§§ 1910.1001–1048) are applicable to the maritime sectors. See the individual standards and 29 CFR 1910.19.

Clearly it is confusing both to the shipyard industry, its employees and for OSHA enforcement for slightly different standards to apply to different operations in the same work place. The 1970 TLV's and the 1968 PEL's do differ for some substances though usually not by significant amounts. Consequently for shipyards, OSHA feels the existence of different standards for different subsectors should be eliminated as soon as possible. Accordingly OSHA is proposing that the new PELs take effect for all of shipyards, as soon as the effective date of the standard, to be achieved by any reasonable means of compliance. The preference for engineering controls in the transition period will apply to the 1970 TLV's for all subsectors of shipyards.

Pursuant to the Longshoremen and Harbor Worker Compensation Acts 1958 amendments (33 U.S.C. 941), OSHA in 1960 issued regulations protecting longshore employees (25 FR 1569). These regulations also covered marine terminals employees. They were adopted as OSHA standards on May 29, 1971 at 36 FR 10466. They were recodified as 29 CFR part 1918 on May 19, 1977 at 37 FR 22530.

On July 5, 1983 (48 FR 30886), OSHA issued a final standard specifically covering marine terminals separately from longshoring. The Marine Terminals Standard was designated 29 CFR part 1917. (It should be recalled that the earlier part 1917 covering shipbreaking had been recodified as part of part 1915—Shipyards.) The Marine Terminal Standard basically provides that employees not be exposed to air contaminants over the limit set in the 1971 Z tables of CFR 1910.1000. See §§ 1917.2(p), 1917.22, 23, 25.

Longshoring operations continue to be regulated by 29 CFR part 1918. OSHA has consistently interpreted that the air contaminant exposure limits set forth in 1910.1000 are applicable pursuant to § 1910.5(c) to longshoring because no quantitative exposure limits are set forth for most air contaminants. Section 1918.93 only sets forth specific numerical limits for carbon monoxide. It is OSHA's intention to clarify in this rulemaking that all the air contaminant exposure limits in the 1971 Z Tables in § 1910.1000 are applicable and will remain applicable to longshoring during the transition period. Any comments to the contrary should be submitted in this rulemaking.

Agriculture

OSHA issued its first standards for agriculture May 29, 1971 (36 FR 10466, 10699) pursuant to section 6(a) of the OSH Act. The specific requirements were specified in 29 CFR 1910.267. That section stated:

§ 1910.267 Agricultural operations
(a)(1) The standards referenced in the remaining subparagraphs of this paragraph apply to the indicated operations, whether or not they include as a part of, agricultural operations.
(2) Sanitation in temporary labor camps—§ 1910.142.
(3) Storage and handling of anhydrous ammonia—§ 1910.111 (a) and (b).
(4) Pulpwood logging—§ 1910.226.
(5) Slow-moving vehicles—§ 1910.145.
(b) Except to the extent specified in paragraph (a) of this section, the standards contained in subparts B through S of this part do not apply to agricultural operations.
(c) The development of standards having more general application to agricultural operations will be the subject of study under section 6 of the Act.

OSHA issued a standard to protect farm workers from pesticides by specifying field reentry times, protective equipment and sanitation codified at § 1910.267a (1974). That standard was vacated by the Court of Appeals on the basis that pursuant to section 4(b)(1) of the OSH Act, OSHA was preempted from regulating pesticides regulated by EPA under FIFRA where EPA exercised statutory authority to prescribe or enforce standards or regulations enforcing occupational safety or health.

In 1975 OSHA recodified the agriculture standard as a new 29 CFR part 1928. (40 FR 18254, April 25, 1979). OSHA issued various standards applicable to the agriculture sector but because of § 1910.267(b) as amended, the exposure limits in what came to be § 1910.1000 Tables Z–1, Z–2 and Z–3 never became applicable to agriculture. When OSHA issued substance specific standards it also did not make them applicable to agriculture. Consequently none of OSHA's exposure limits are currently applicable to agriculture.

On September 30, 1976 (Pub. L. 94–439, 90 Stat. 1418) Congress included a rider on the Department of Labor appropriations act for fiscal 1977 stating that OSHA should not expend any funds to issue or enforce any standard applicable to any person engaged in a farming operation employing 10 or fewer employees.

That rider has been reenacted each year subsequently. The rider for fiscal 1991 (Pub. L. 101–517, 104 Stat. 2194) states

> That none of the funds appropriated under this paragraph shall be obligated or expended to prescribe, issue, administer, or enforce any standard, rule, regulation, or order under the Occupational Safety and Health Act of 1970 which is applicable to any person who is engaged in a farming operation which does not maintain a temporary labor camp and employs ten or fewer employees:

Accordingly OSHA is only proposing to apply the Air Contaminants standard to farming operations which maintain a temporary labor camp or which employ more than 10 employees. For this exemption to apply there can not be more than 10 employees on any day in the year. In addition, employees who technically work for a labor only subcontractor but work under the direction of the farm operation are considered employees of the farm for purposes of determining whether it has more than 10 employees. In practice this rider means that only quite large farms will be covered by this regulation. See the discussion under the History and Need Section.

As discussed above pursuant to section 4(b)(1) of the OSH Act and the above cited cases, use of an EPA labelled pesticide on a farm as a pesticide pursuant to the label where that label includes employee protection provisions is not covered by the proposed air contaminant standard. Approximately 100 of the chemicals that OSHA is proposing in 29 CFR 1928.1000 Table Z may fit that category.

These EPA-labeled pesticides are still listed in Table Z and the exposure limit will be applicable in circumstances when those substances are not used as pesticides pursuant to the FIFRA label. Examples of these situations include when the pesticide is used for a non pesticide use or for a pesticide use not authorized by the EPA label. It should be noted that a number of common chemicals with non pesticide uses are

also labelled pesticides. In addition a farmer may make and use a pesticide which he could have purchased already labelled. In these circumstances there is no requirement for EPA labelling, and OSHA would have jurisdiction. The agricultural employee would then be covered by the OSHA PEL. OSHA has come across situations like this and believes that it is important not to allow loopholes to develop. In addition OSHA believes that publishing exposure limits for pesticides even when not enforceable provides useful information. Employer and employees may find the information useful to assist in protecting themselves from the pesticide's toxic effects.

In addition it should be noted OSHA is applicable only to employment relationships. If the farmer is a sole proprietor the farmer and generally the farmer's immediate family are not covered.

OSHA has concluded that updating the Air Contaminant levels for construction, maritime and agriculture workers to reflect recent information is among the highest priorities for the Agency. This will reduce exposure limits for approximately 212 substances currently regulated and add exposure limits for approximately 164 substances which are currently unregulated for the construction and maritime sectors. These new limits and 220 others which currently protect other employees will protect agriculture employees. The health literature indicates this must be accomplished to improve worker health; it is one of Congress's current goals and will greatly increase occupational health protection for a very large number of workers.

In order to accomplish this high priority task in a reasonable time in the light of limited administrative resources, it is necessary to narrow somewhat the issues to be faced by the Agency in this proceeding and to build on previous efforts of the agency. Consequently, it is necessary to delay other worthwhile goals and concurrent Congressional purposes.

This approach is consistent with the general principles of administrative law. An Agency may set priorities within the framework of its statutory authority. Secondly, an Agency may take substantial steps towards its statutory goals, without having to achieve them completely, when Agency resources are not sufficient to complete all aspects initially.

Congress recognized that the Secretary could not address all occupational safety and health problems simultaneously. It therefore gave the Secretary discretion to set priorities in exercising the Secretary's rulemaking authority. As section 6(g) of the Act, 29 U.S.C. 655(g), states:

> In determining the priority for establishing standards under this section, the Secretary shall give due regard to the urgency of the need for mandatory safety and health standards for particular industries, trades, crafts, occupations, businesses, workplaces or work environments.

In proposing this addition to the Act, Senator Javits explained that its purpose was "to relieve the Secretary of the necessity for waiting to promulgate whatever standards he wishes to promulgate across the board but rather, allowing him to yield to more urgent demands before he tries to meet others." Leg. Hist 505. Thus, the Act has "built in flexibilities" that the Secretary may use, such as establishing "the priorities between the various occupations that may require standards." *National Congress of Hispanic American Citizens* v. *Usery*, 554 F. 2d 1196, 1199 (D.C. Cir. 1977); see also National Congress v. Marshall, 656 F. 2d 882 (D.C. Cir. 1979). The flexibility expressed in the statute and legislative history is consistent with the well-established principle that an administrator may adopt a "rational, 'one step at a time' approach to rulemaking." *National Roofing Contractors Ass'n* v. *Brennan*, 495 F. 2d 1294, 1299 (7th Cir.), cert denied, 419 U.S. 1105 (1974) (OSHA roofing standard); cf., *Industrial Union Dept.* v. *American Petroleum Institute*, 488 U.S. 607, 663, (1980) (Burger, concurring) (OSHA can act in its legislative capacity "to focus on only one aspect of a larger problem"); *United Steelworkers* v. *Auchter*, 763 F. 2d 728, 738 (3rd Cir. 1985) (Although OSHA's decision to exclude workers in some industries from a standard requires explanation, "[s]ection 6(g) clearly permits the Secretary set priorities for the use of the agency's resources and to promulgate standards sequentially."); *IUD* v. *Hodgson*, 499 F. 2d 467, 480 n. 31 (D.C. Cir 1974) ("The [OSHA] statutory scheme is generally calculated to give the Secretary broad responsibility for determining when standards are required and what those standards should be.").

OSHA has concluded that setting exposure limits for the large number of substances involved in this rulemaking for construction, maritime and agriculture employees has priority over exploring the need for accompanying medical surveillance, exposure monitoring and industrial hygiene provisions for a much smaller number of substances at this stage. Section 6(b)(7) of the Act, of course, indicates that "where appropriate" such provisions are to be included. Although regulating these provisions is a concurrent goal of Congress, so was lowering exposure for the many unregulated or inadequately regulated substances where scientific data indicate lower exposures are needed. However, OSHA has inadequate resources to accomplish both goals at this time. Lowering exposures is a higher priority because it is more effective in reducing diseases and material impairments of health.

OSHA has already addressed some of section 6(b)(7)'s goals, as they relate to labels and warnings, in the generic Hazard Communication Standard, 29 CFR 1910.1200. It is working on a standard to improve respirator use for all chemicals (47 FR 20803). It is considering generic regulation for exposure monitoring (53 FR 32591–32595) and medical surveillance (53 FR 32595–32598). OSHA does not have the resources to conclude this rulemaking in any reasonable time, and also consider these issues.

OSHA does have legal authority not to address ancillary provisions in this rulemaking and determinations about the appropriateness of ancillary provisions have not been made. That is a rational use of its priority setting authority in the light of OSHA's limited resources. The language of section 6(b)(7) is not an absolute requirement to include such provisions and this is an "appropriate" circumstance not to include them. The actions already initiated by OSHA indicate it is facing the issue of ancillary provisions in a responsible and reasonable manner.

OSHA, in the air contaminants rulemaking process, has rationally and efficiently used the priority process to maximize employee protection while meeting all legal requirements. OSHA proceeded first with general industry so as not to hold up the general industry rulemaking while it was consulting with the construction, maritime and agriculture sectors and so it would have the resources to carefully study feasibility in the construction, maritime and agriculture sectors.

In the general industry rulemaking OSHA carefully considered what would be the appropriate universe of substances to consider for change. (See 53 FR 20966–87, 20977–79). Then OSHA proposed changing or requested comments on the appropriateness of changing the exposure limits for 428 substances. After reviewing comments and analysis of the studies, OSHA concluded that changes were appropriate for 376 substances.

OSHA believes that reconsidering the universe of substances to be considered

in this rulemaking would substantially delay issuing more protective limits for many substances for maritime, agriculture and construction workers. Similarly, proposing new limits for substances for which OSHA concluded new limits were not necessary or would involve excessively lengthy analysis in the general industry rulemaking, would also result in substantial delay in issuing more protective limits for many substances in construction, maritime and agriculture. In addition it would be a waste of OSHA's resources to reconsider decisions it just recently made based on substantial evidence and public comment on these matters. Resources used for such reconsideration could not be utilized on other necessary improvements in worker health and safety.

Accordingly, based on its priority setting authority, OSHA is proposing to change in this rulemaking only the 376 substances for which it determined change was appropriate for general industry and is opening the record for maritime and construction only for those 376 substances. In addition to proposing these 376 substances for agriculture, OSHA is proposing to adopt for agriculture the PEL's for the approximately 220 substances which currently cover construction and maritime and which OSHA is not reopening for consideration in construction and maritime. These include the approximately 160 Z-table substances OSHA did not consider in the general industry rulemaking, approximately 10 single standard substances and the approximately 50 substances, OSHA considered, but decided not to change for general industry.

Lead is being covered in this rulemaking. The general industry lead standard of 50 ug/m^3 plus ancillary provisions in 29 CFR 1910.1029 is applicable to the maritime industry but does not apply to construction. Currently all that is applicable is a 200 ug/m^3 exposure limit. Several recent studies have indicated that cases of lead poisoning are occurring in construction workers. The Construction Advisory Committee recommended that OSHA lower the exposure limit for construction workers as soon as possible. OSHA agrees with the recommendation and is accordingly proposing the 50 ug/m^3 limit for construction (and agriculture) through the PEL's rulemaking as the most efficient way of setting a more protective exposure limit for lead in these sectors.

OSHA has also commenced consideration of ancillary provisions for lead exposure in construction. However, this process will take longer because of issues like medical removal protection, which require extensive consideration because of the different characteristics of the construction industry.

In the general industry rulemaking for air contaminants OSHA extensively considered the health effects of the substances regulated. The initial analysis was published in the proposal, extensive public comments were received, and OSHA analyzed more than 2000 studies and 1500 comments in reaching its final decisions as discussed in the final preamble. Those final conclusions are based on the science current through 1987. In consequence OSHA has a high degree of confidence in the underlying scientific merit of its final exposure limits. (In a very few cases OSHA did indicate that more extensive analysis would be appropriate.)

The proposed exposure limits for agriculture, construction and maritime are based on the final levels and analysis for general industry. That information is both current and has been based on a recent notice and comment rulemaking. OSHA has incorporated into the analysis the "latest available scientific data in the field" (section 6(b)(5)). OSHA has made an extensive search of the 1987 and later scientific literature which may not have been available in the general industry rulemaking and has incorporated the most recent studies into the health analyses for each substance.

OSHA will issue final standards meeting legal requirements and good science. If the new studies or new comments demonstrate that the proposed limits are not legally or scientifically supportable OSHA will make appropriate changes.

However OSHA's intent is not to make adjustments in the proposed level in this rulemaking because on balance the new studies or comments may suggest but do not compel some minor adjustment in the limit. OSHA's belief is that employees in all of the sectors it regulates should be equally well protected based on the health science information. (There may be some differences based on feasibility). Obviously people are biologically the same no matter what industry they may be employed. In addition a certain unfairness is created if employees in one sector are better protected than employees in another sector (absent compelling feasibility reasons).

If OSHA made minor changes in the proposed exposure limit based on a slight tilting of the weight of the evidence, the result would be that workers in general industry would be covered by a different level than workers in construction, agriculture and maritime. Consequently consistency in approach and protection would be lost. In addition it would seriously waste OSHA's scientific resources to fine tune levels for construction, agriculture and maritime in this rulemaking, and then have to reconsider that same fine tuning for general industry in a subsequent rulemaking.

OSHA believes that adjustments to the new air contaminants levels are best done in the first PEL update, rather than in this rulemaking. OSHA intends to regularly update the Z Table levels based on new scientific data and to add new substances to the Z–Tables. In each such update OSHA intends that the additions and changes will apply to all sectors; general industry, construction, maritime and agriculture. OSHA has publicly announced its intention to carry out these periodic updates. See the Regulatory Agenda at 56 FR 17566 (April 22, 1991). This approach is biologically sound, fairer to workers and is a more efficient use of OSHA's scientific resources. Consequently OSHA believes that this two prong approach, using this rulemaking to expand the new PEL's to construction, maritime and agriculture, and the first update to be the principle rulemaking to change any of the new PEL's across the board, is a sensible and permissible use of OSHA priority setting authority.

OSHA wishes to make it absolutely clear, that it is not reopening exposure limits for general industry in this rulemaking. It will not consider, and will not consider as a part of the rulemaking record, comments or information intended to change the exposure limits for general industry. Any such comments will be considered requests under section 6(b)(1), that OSHA consider such substance for review in the first PEL update rulemaking. That rulemaking is likely to commence in 1993.

There are 3 substances only, which OSHA will be considering for general industry, as well as construction, maritime and agriculture. These are asphalt fumes, mineral wool and fiberglass (which includes refractory ceramic fibers or RCF's). OSHA stated in the general industry final rule that it needed to further study these substances before reaching final conditions. (See 54 FR 2510, 2515, 2679). In this rulemaking, OSHA is reopening the rulemaking record for comment on all issues (health and feasibility) relating to these substances for general industry

and construction, maritime and agriculture.

OSHA through this rulemaking is proposing that the exposure limits for all substances it regulates (basically) will apply to all workers in all sector within OSHA jurisdiction. Consequently exposure limits for some substances will apply to sectors where OSHA is not aware of exposure. OSHA is following this approach because it believes that it represents sound policy and meets OSHA legal requirements.

OSHA has at various times carved out exemptions from its standards based on the belief that there were no exposures or only very low exposures. However in OSHA's experience it often turns out that there are indeed exposures which OSHA is not aware of at the time, or indeed the exemption becomes a loophole where an employer attempts to organize its business operations so as to come within the exempt sector though there are significant exposures.

For example the coke oven standard did not apply to the construction sector. So some employer's designated work on coke ovens, construction work, and hired construction contractors to perform it. It was then argued that though these workers had significant exposures, they were not covered because it was "construction work" they were doing. OSHA also exempted gas station attendants from the benzene standard because studies showed exposures were very low in relation to the PEL because of the low levels of benzene in gasoline. Subsequently oil companies greatly increased the level of benzene in gasoline and employee exposures are now much closer to the PEL. It also was argued that the wording of the exemption covered mechanics, though the exposure patterns are different and could be higher. A wood preserver argued to EPA that since it made the arsenical preservative it used, it was not covered by EPA labeling requirements, and to OSHA that since the chemical it used was an EPA regulated preservative, it was not covered by OSHA regulations. (As stated above, OSHA believes this argument without merit).

OSHA believes that making it easier to create loopholes will tend to reduce worker protection. Secondly the few workers who might be exposed in sectors where OSHA is not aware of exposure are entitled to protection. They are at just as much health risk per unit of exposure as are workers in other sectors.

In addition this policy does not create any unnecessary economic burdens. If there are no workers exposed, there will be no costs. If there are few workers exposed the costs will be low.

In addition the time taken to analyze in depth whether or not there are exposures in a sector where there may be just a few, are a waste of regulatory resources. That information adds nothing to worker protection or for that matter to economic efficiency.

This approach meets OSHA legal requirements. A worker in a sector where there are few workers exposed (some of which OSHA may not know about) is at significant risk of material impairment of health, if over the exposure limit to the same degree as the worker in a sector where many workers are exposed. The cost is going to be insignificant in relation to industry resources and none, if indeed there is no exposure.

As previously noted OSHA is required under its statutory authority to "set the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposure to the hazard dealt with by such standard for the period of his working life" under the provisions of section 6(b)(5) of the OSH Act. "Material impairment" is therefore a term which needs to be considered when issuing standards.

In this rulemaking, OSHA is considering establishing new or revised PELs for approximately 370 separate substances for construction and maritime and 600 for agriculture. The health effects for these substances cover a wide spectrum of severity including: life threatening effects; disabling effects; various diseases; irritation to different organs or tissues; and changes in organ functions indicative of future health decrements.

The statutory requirements in sections 3(8) and 6(b)(5) are quoted above. Other statutory criteria are set forth in section (2)(b) which states:

> The Congress declares it to be its purposes and policy, through the exercise of its powers to regulate commerce among the several States and with foreign nations and to provide for the general welfare, to assure so far as possible every working man and woman in the Nation safe and healthful working conditions and to preserve our human resources.

One of the earlier Senate drafts of the OSHA bill did not include the word "material" before the word "impairment." That word was added by an amendment of Senator Dominick. The Senator stated with respect to that amendment:

> What we were trying to do in the bill—unfortunately, we did not have the proper wording or the proper drafting—was to say that when we are dealing with toxic agents or physical agents, we ought to take such steps as are feasible and practical to provide an atmosphere within which a person's health or safety would not be affected. Unfortunately, we had language providing that anyone would be assured that no one would have a hazard, or at least, we would require the Secretary to set standards so stating, and that in the HEW standard there would be a requirement to proceed on that basis so that no one would have any problem for the rest of his working life. It was an unrealistic standard. As modified, we would be approaching the problem by looking at the problem and setting a standard or criterion which would not result in harm (Legislative History p. 502).

The D.C. Circuit Court considered the concept of material impairment and reviewed the Legislative History in the Lead Case.

It stated:

> The essential question under section 6(b)(5) for this case is whether OSHA acted within the limits of this mandate to establish "material" impairment of health when it set a standard designed to protect workers from the subclinical effects of lead. As a statutory matter, after examining precedent and legislative history, we hold that section 6(b)(5) empowers OSHA to set a PEL that prevents the subclinical effects of lead that lie on a continuum shared with covert lead disease. (*United Steelworkers* v. *Marshall*, 647 F. 2d at 1248–49. See also the more extended discussion there).

The legislative history and judicial analysis indicate that OSHA is to take a balanced but protective approach. Some impairments are so slight a discomfort that they are not material and do not provide a basis for regulation. A complaint of minor discomfort, in and of itself, is not material impairment. However, the OSH Act is designed to be protective of workers and is to protect against impairment with less impact than severe impairment.

These health effects are related to two different types of exposure: acute and chronic. Because of the difference between the health effects associated with these two types of exposure, OSHA must consider different types of PELs (TWA, STEL, or ceiling) to protect against material impairment.

OSHA asked for comment on the subject of "material impairment" in the general industry rulemaking, especially with regard to whether sensory irritation should be considered to be material impairment. There was a relatively limited amount of comment. The most complete response was provided by NIOSH (Ex. 8–47) which stated:

The recognition of sensory irritation as potentially being "material impairment of health" is consistent with the current scientific consensus related to health effects of environmental agents.

Mucous membrane irritants can cause increased blink frequency and tearing; nasal discharge, congestion, and discomfort, wheezing, chest tightness, and dyspnea. Work environments often require levels of physical and mental performance considerably greater than encountered in daily living. Even in the absence of any permanent impairment, the symptoms listed can interfere with job performance and safety.

Mucous membrane irritation can result in inflammation, which may lead to increase susceptibility to nonspecific irritants and infectious agents. For example, experimental ozone exposure in humans results in increased airway reactivity. Also, studies of exposure to environmental tobacco smoke have shown irritative symptoms and evidence of increased frequency of respiratory tract illnesses in young children and decreased pulmonary function in adults

* * *

Mucous membrane irritation is associated with respiratory illnesses, depending on the composition of specific exposure and on the dose, duration, and frequency of exposure. No universally applicable conclusion can be drawn at this time regarding the association between irritative symptoms and permanent injury or dysfunction. Where certain individuals show no measurable impairment after an exposure, even when experiencing irritative symptoms, others may develop identifiable dysfunction.

Aside from the effects of irritations, mucous membrane exposure may result in absorption of a substance, with resultant systemic toxicity. An inflamed mucous membrane may be an even more effective route of absorption, either for the irritant or for other substances. Furthermore, injury to bronchopulmonary membranes can impair removal of particulates from the respiratory system.

Thus, according to NIOSH, sensory irritants interfere with job performance and safety, cause inflammation, may increase the victim's susceptibility to other irritants and infectious agents, lead to permanent injury or dysfunction, or permit greater absorption of hazardous substances. In sum, NIOSH and most other respondents, in the general industry rulemaking agreed that sensory irritation caused by occupational exposure to the irritant substances included in this proposal constitutes a material impairment of health.

Of course, irritation also covers a spectrum of effects, some serious and some trivial. Hence, a complaint of minor irritation would not in and of itself constitute material impairment. In addition, OSHA would weight irritation with physical manifestations more heavily than irritation with purely subjective responses. This does not mean that purely subjective responses would not constitute material impairment. That judgment would depend on the magnitude of the irritation.

OSHA also believes that clinical, tissue or organ changes, or properly documented pain, chest tightness, migraine headache or similar reactions reflected in responses by persons, may also represent material impairment of health. Each of these are considered on a case-by-case basis in this Rulemaking. OSHA believes that its approach is consistent with the Act and Congressional intent regarding material impairment of health.

All of the revised or new PELs in this proposal represent in OSHA's views situations involving material impairment of health.

OSHA's analyses regarding material impairment of health, as applied in this proposal, are provided in three separate ways. First, they are included in the introductory discussion for health effects. Second, the discussion of each of the substances contains health effect information for all new or revised PELs. Third, this discussion provides examples, and general guidance regarding the OSHA decision process, and accounts for the severity spectrum of health effects, and the separate cases involving chronic and acute exposures.

As previously indicated, health effects cover a wide range of severity levels. A precise delineation between material impairment and non-material impairment is not possible since a variety of factors must be considered, such as the composite health effect and frequency and duration of the effect, to determine if a substance represents, a material impairment of health. However, a small reduction in nerve conductivity may not constitute material impairment. Moreover, an occupational, transitory, non-progressive and/or non-intensive coughing reaction may represent non-material impairment. Major intensification of some of these factors could result in a health effect which represents material impairment of health. Consequently, general considerations can be stated but they must be applied on a case-by-case basis taking into account the scientific evidence, public comments and agency expertise.

For the substances OSHA is regulating as carcinogens, it has met all requirements of *IUD* v. *API* and has acted consistently with 29 CFR 1990. Specifically, OSHA reviewed all data to determine whether there were studies of sufficient merit to determine that the substance was qualitatively a carcinogen.

Secondly, appropriate risk assessments were performed where the scientific data permitted. For most of the carcinogens, OSHA contracted with Dr. Nathan J. Karch, President of Karch & Associates, Inc., consultant experts in risk assessment, to analyze the available studies for two purposes; first to determine whether there was sufficient evidence to perform a quantitative risk assessment or to state the reasons why there was not; second, to perform a quantitative risk assessment where the studies permitted, using techniques generally accepted by the scientific community. The risk assessments are presented in this preamble.

The risk assessment for asphalt fumes was performed by OSHA scientists. The risk assessment for perchloroethylene was performed by Dr. Dale Hattis. All these assessments are discussed in this preamble.

For other substances, significant risk determinations were made directly from the studies themselves. They indicate the levels where significant risk exists and where it is eliminated. Appropriate safety factors are incorporated.

Accordingly OSHA believes that the type of significant risk analysis undertaken in this rulemaking both for carcinogens and non carcinogens is most consistent with the studies generally available and is a valid scientific approach. OSHA concludes it is fully consistent with the requirements of the Supreme Court in *IUD* v. *API*.

OSHA is utilizing its priority setting authority in another respect. There are a few substances (both carcinogens and non carcinogens) where more detailed analysis of the evidence might in the future lead to the conclusion that there is remaining significant risk. If that were the case in a single substance rulemaking, OSHA would explore that issue in great depth and do much more extended economic analysis of several different exposure levels to determine what the lowest feasible level might be.

Past experience has shown this to be a major undertaking both from a health and economic point of view. OSHA, for example, spends an average of $500,000 and takes one year of study to determine the lowest feasible level for a single substance. OSHA does not have the resources to engage in that kind of analysis for more than a few substances. The attempt to do so in this rulemaking would significantly reduce the chances of it ever being completed and would result in far more workers being exposed to significant risk in exchange

for the incremental risk reduction attained by further lowering the PEL for a small group of substances in this Rulemaking.

In this rulemaking OSHA's priority is to extend coverage of more protective PEL's for hundreds of substances to all workers in construction, maritime and agriculture. That will achieve far greater overall health benefits for workers than fine tuning exposure limits for a few substances. OSHA, where appropriate, will consider such adjustments in the light of legal requirements in the first update of the PEL's as discussed above.

OSHA concludes this is both rational priority setting and a reasonable integration of its priority setting authority and relevant case law. Clearly multi-issue and multi-substances rulemaking require a different balancing than single substance or single issue rulemaking.

OSHA feasibility determinations are based on both the statute and on a consistent and extensive body of case law extending over its entire history. In addition to *ATMI* (supra) see for example: *AFL–CIO* v. *Hodgson*, 499 F. 2d 467 (D.C. Cir., 1974); *Society of Plastics Industries* v. *OSHA* (SOCMA), 509 F. 2d 1301 (2d Cir., 1975); *American Iron and Steel Inst.* v. *OSHA* (AISI), 577 F. 2d 825 (3rd. Cir., 1977); *United Steelworkers* v. *Marshall*, 647 F. 2d 1189 (D.C. Cir., 1980); *ASARCO* v. *OSHA*, 746 F. 2d 483 (9th Cir., 1984) and others.

Standards may be expensive and still be feasible if necessary to protect occupational health.

> Standards may be economically feasible, though from the standpoint of employers, they are financially burdensome and affect profit margins adversely. Nor does the concept of economic feasibility necessarily guarantee the continued existence of individual employers. It would appear to be consistent with the purpose of the Act to envision the economic demise of an employer who has lagged behind the rest of industry in protecting the health and safety of employees * * * (*Hodgson*; 499 F. 2d at p. 478)

An OSHA standard may be technology forcing. OSHA may demonstrate feasibility by showing that only a few plants are now in compliance. Moreover, a standard is still feasible even though some respirator use is needed to achieve compliance.

> [T]he Secretary is not restricted by the status quo. He may set standards which require improvements in existing technologies or the development of new technology * * * (SOCMA, 509 F. 2d at p. 1309).
>
> The experience at (2) batteries provides a sufficient basis for the Secretary's reasoned belief that the 0.15 mg/m³ limit could be met (for the entire industry) (AISI, 577 F. 2d at p. 834; See also SOCMA, p. 1309).
>
> The limited respirator use that the standard requires does not in any way render the standard infeasible * * * (ASARCO, 746 F. 2d at p. 483. See also ATMI generally and SOCMA 509 F. 2d at p. 1310, etc.)

OSHA must show a general presumption of feasibility in most operations in an industry with engineering controls to place the burden of proof in enforcement action on the industry to show that compliance with engineering controls cannot be attained in a particular circumstance. But a showing by a particular industry that compliance requires respirators in certain operations or an admission by OSHA that is the case does not make a standard infeasible. Rather it "will reduce the strength of the presumption a firm will have to overcome on justifying its use of respirators" in an enforcement or variance action. *United Steelworkers*, pp. 1272–73. See also *Building and Construction Trades* (Supra). OSHA has indicated in the feasibility analysis various operations where respirator protection may be the appropriate method of protection in some circumstances. When OSHA has stated that, OSHA would not site for failure to use engineering controls unless in the specific circumstances there was a simple engineering control available which was widely known in the industry.

Finally the standards themselves require the use of engineering controls when feasible. If they are not feasible, the employer may use respirators. Consequently, this standard does not become infeasible simply because engineering controls may not achieve the PEL in a specific operation.

In this rulemaking, OSHA concludes that it has demonstrated feasibility without taking that concept to the full limits of its legal authority. Many of the substances regulated constitute acute hazards with apparent thresholds; the limit set to protect health does not approach the limits that could feasibly be achieved. Since OSHA's feasibility analyses are based on what industry is already achieving or what could be achieved with standard "off-the-shelf" technology, there are few if any cases where OSHA is attempting to force technology.

As discussed in the economic feasibility section below, the cost of this standard is low in relation to the sales and profits of each subsector. Consequently OSHA believes that this standard meets the test for economic feasibility as discussed in much greater length below.

To clarify, OSHA is opening the record for general industry only for asphalt fumes, mineral wool and fiberglass (including RCF's). For construction and maritime OSHA is only opening the record for those substances which it is proposing to change the PEL or add a new PEL for the first time. This is approximately 380 substances. The number varies slightly among the sectors depending upon whether the prior standards were the 1970 TLV's or the 1971 PEL's. It includes those substances for which OSHA changed PEL's in the 1989 final standard for general industry and those single substance standards in §§ 1910.1001–1048 which had not been made applicable to construction or maritime but had PEL's. (The 13 carcinogens (§ 1910.1002–1016) are not included because they do not have exposure limits). For those single substance standards which do not already apply to construction or maritime, only the exposure limit is being made applicable—not ancillary provisions. This rulemaking for reasons stated above, only covers exposure limits—not ancillary provisions. OSHA is not opening the record for the appropriate 200 substance already applicable to construction and maritime where OSHA is not proposing to change limits. This includes not opening the record for substances which OSHA proposed changing for general industry but determined that change was inappropriate during the air contaminants rulemaking.

For agriculture, OSHA is opening the record on every substance since no limits are now applicable. There accordingly are health write-ups for 160 substances which will be made applicable to agriculture for which OSHA did not reopen the record for general industry, and for which OSHA is not opening the record for construction and maritime. There are also write-ups for the substances which were considered and not changed for general industry, and which have not been opened for maritime or construction but which OSHA is proposing to cover for agriculture. Finally there are brief health write-ups for the substances listed in 1910.1001–1048 which are not applicable to agriculture, but for which OSHA is proposing that the PEL's cover agriculture. For each of these, OSHA is putting in the docket the final preamble for those substances which OSHA incorporates by reference as part of its health analysis.

OSHA is proposing in this standard to make the Z table cotton dust limit of 500 $\mu g/m^3$ vertical elutriator or 1 mg/m^3 personal sampler applicable to agriculture and to cotton gins. OSHA's 1978 cotton dust standard for cotton gins

(29 CFR 1910.1046 (1974)) was vacated by the Fifth Circuit in *Texas Ind. Ginners Assoc.* v. *Marshall,* 630 F. 2d 398 (1980). That standard specified work practices and medical surveillance, but did not specify an exposure limit. OSHA never appealed.

The Fifth Circuit's decision was prior to the Supreme Court's decision upholding the cotton dust standard for the textile industry in *ATMI* v. *Donovan* (supra). The *Tex. Ind. Ginners* decision was based in principle part on OSHA's failure to perform a cost benefit analysis and on a finding that acute reductions in lung function were not material impairments of health. The Supreme Court in *ATMI* held that OSHA was not to use cost benefit analysis to determine whether to issue health standards and that the acute effects of cotton dust were material impairments of health. Accordingly OSHA concludes it has authority to propose this cotton dust standard covering agriculture and gins.

The OSHA Air Contaminants standard for general industry is being challenged in the case of *AFL–CIO* v. *OSHA,* Nos. 89–7185 et al, before the Eleventh Circuit Court of Appeals. Currently pending are nine suits by industry challenging as too low the exposure limits for seven chemicals (perchlorothylene, nickel carbonyl, carbon monoxide, sulfur dioxide, welding fumes and nitrogen dioxide). The AFL–CIO in the tenth suit is challenging as too high exposure limits for fifteen chemicals (methyl bromide, methyl chloride, TDI, crystalline silica, mineral oil, chloroform, wood dust, m-butyl glycidal ether, m-hexanone, perchoroethylene, sulfur dioxide, carbon tetrachloride, vinyl bromide, trichloroethylene and gasoline). The petitioners also challenge the standard on several more general aspects. Eighteen other law suits were filed. Fifteen were settled and withdrawn, three were voluntarily withdrawn.

OSHA believes its decisions and approach are correct and has so argued to the Eleventh Circuit. If during the course of this rulemaking, the Eleventh Circuit remands any of the challenged substances to OSHA for further consideration on health issues, OSHA will probably withdraw those substances from this rulemaking and combine their consideration with the remand consideration for general industry. OSHA, if the Eleventh Circuit permits, would prefer that consideration to take place in the first update proceeding discussed above.

OSHA has committed to reconsider the carbon tetrachloride, vinyl bromide, trichloroethylene and gasoline PEL's for general industry in the first update. It will at that time, reconsider them for construction, maritime and agriculture as well. OSHA believes in this rulemaking, its priority is to concentrate on issuing more protective limits for these sectors, and refinements should await the first update. OSHA believes that the AFL–CIO challenges to OSHA's failure to issue final asphalt, fiberglass and mineral wool standards in the general industry rulemaking have been made moot by OSHA's proposing levels for those substances for all sectors in this rulemaking.

III. Glossary

The following terms and acronyms appear in the standard and the preamble supporting it. This glossary is provided as a convenience to the reader.

ACGIH—The American Conference of Governmental Industrial Hygienists is a professional society devoted to the development of administrative and technical aspects of worker health protection. Membership is limited to professional personnel in governmental agencies or educational institutions engaged in occupational safety and health programs. The ACGIH issues guidelines and recommendations in the form of Threshold Limit Values (TLV®s) which are published annually.

CAS—The Chemical Abstracts Service (CAS) Registry Number is a numeric designation assigned by the American Chemical Society's Chemical Abstracts Service which uniquely identifies a specific chemical compound. This entry allows one to conclusively identify a substance regardless of the name or naming system used.

CHRIS—The Chemical Hazards Response Information System was developed by the U.S. Coast Guard in cooperation with the National Academy of Sciences to provide information on the handling and disposal of toxic substances. CHRIS consists primarily of the Hazardous Chemical Data Manual which contains chemical, physical and health hazard data on approximately 600 hazardous chemicals and substances; and a Hazard Assessment Computer System in an extensive data base of the information contained in the Hazardous Chemical Data Manual.

HSDB—The Hazardous Substances Data Bank, a part of the National Library of Medicine System, will soon be available on OSHA's Computerized Information System (OCIS). This data bank, currently available through TOXNET, contains health and safety profiles for over 4100 chemicals. It includes 144 data elements in 10 categories including use information, substance identification, animal and human toxicity, environmental fate, standards, personal protective equipment, fire, physical and chemical properties.

IARC—The International Agency for Research on Cancer (IARC) is a research organization authorized by the World Health Organization in 1965. IARC's mission is to study the causes of cancer in the human environment. IARC has published (and continues to update) a series of monographs on a substantial number of toxic chemicals and substances in which the carcinogenic risk of these chemicals is evaluated.

ILO—The International Labour Organization (ILO) is a specialized agency associated with the United Nations. Established in 1919 as part of the Versailles Peace Treaty, the ILO serves to band together governments, employers, and workers of 145 nations in an international effort to improve overall working conditions and to protect the life and health of workers.

IMIS—The Integrated Management Information System (IMIS) is a data base developed by OSHA in 1979 with sampling information on more than 100,000 individual measurements. The IMIS contains exposure measurements obtained by OSHA compliance officers during thousands of health inspections; it is the most extensive data base of its kind.

Material—The term "material" is used in the original standard whereas "substance" is used in the revision. The meaning is the same.

MSDS—The Material Safety Data Sheet (MSDS) is a compilation of data and information on individual hazardous chemicals produced by the manufacturers and importers of that chemical, as required by OSHA's Hazard Communication Standard, 29 CFR 1910.1200. An MSDS contains data on chemical identification, current exposure limits, chemical reactivity, fire and explosion limits, and information on health hazards and emergency procedures, spill, leak, and disposal procedures, and any needed special protection or precautions.

NIOSH—The National Institute for Occupational Safety and Health (NIOSH) was created by the Occupational Safety and Health Act of 1970. NIOSH is part of the Centers for Disease Control under the Department of Health and Human Services. Its mandate includes conducting research in developing criteria and/or recommendations to be used in setting occupational exposure standards, identifying and evaluating workplace hazards, measurement techniques, and control technologies, and providing

professional education as well as health and safety information.

NOES—The National Occupational Exposure Survey (NOES) is a data base completed in 1982 by NIOSH. NOES is the successor to the first such data base, completed by NIOSH in 1974, and known as the National Occupational Hazard Survey (NOHS). The NOES data base contains a sample of the number of persons exposed by substance and industry from approximately 4500 businesses in 98 geographic areas in the U.S. These surveys provide national estimates of potential exposure to workplace hazards, by industry and occupational group.

OCIS—The OSHA Computerized Information System is a comprehensive data base that contains information and data on standards interpretation, chemical information, hazardous waste activity, 5(a)(1) citations, a health hazard evaluation index, training materials, and other information compiled by OSHA on subjects related to occupational safety and health.

OSHA HS Number—A Health Standard (HS) number is a 4-digit code assigned, for ease in reference, to each of the hazardous substances or chemicals considered for change of PEL in this rulemaking.

PEL—Permissible Exposure Limits (PELs) are limits developed by OSHA to indicate the maximum airborne concentration of a contaminant to which an employee may be exposed over the duration specified by the type of PEL assigned to that contaminant.

REL—Recommended Exposure Limits (RELs) are issued by NIOSH to aid in controlling hazards in the workplace. These limits are generally expressed as 8- or 10-hour TWAs for a 40-hour workweek and/or ceiling levels with time limits ranging from instantaneous to 120 minutes. RELs are published in a variety of NIOSH documents.

RTECS—The Registry of Toxic Effects of Chemical Substances (RTECS) is a data base that lists an identification number, synonyms, Department of Transportation (DOT) hazard label information, EPA Toxic Substances Control Act (TSCA) information, OSHA and Mine Safety and Health Administration (MSHA) air exposure limits, and animal and human toxicologic data.

Substance—The term "substance" is used in the revised standard whereas "material" is used in the original. The meaning is the same.

TLV—The Threshold Limit Value (TLV) is a registered trademark for an exposure limit developed by the American Conference of Governmental Industrial Hygienists (ACGIH). A listing of TLVs may be found in the ACGIH's "Documentation of the Threshold Limit Values and Biological Exposure Indices for 1988–1989." TLVs may be stated as a time-weighted average (TLV®–TWA), a Short-Term Exposure Limit (TLV®–STEL), or a Threshold Limit Value Ceiling (TLV®–C). OSHA utilized the 1987–88 TLVs as a starting point for this rulemaking.

TSCA—The Toxic Substances Control Act (TSCA), administered by the Environmental Protection Agency (EPA), was passed by Congress to protect human health and the environment by requiring testing and necessary use restrictions to regulate the commerce of certain chemical substances.

WHO—The World Health Organization (WHO) is part of the United Nations. WHO's programs in occupational health include development of an occupational health information system, criteria for early detection of health impairment, and the development of internationally recommended health-based permissible exposure limits for occupational exposure to toxic substances.

IV. Health Effects Discussion and Determination of Proposed PELs

A. General Principles of Toxicology and Dose Response Introduction

As long ago as the 16th century, people recognized that there is no such thing as an absolutely safe chemical. The Swiss physician Paracelsus, who lived from 1493 to 1541, said:

> All substances are poisons; there is none which is not a poison. The right dose differentiates a poison and a remedy.

On the other hand, methods have been devised to permit any chemical, no matter how poisonous, to be handled safely; this is done either by limiting the dose or controlling the exposure. However, before the necessary degree of control can be determined for a particular exposure or situation, the toxicity of the substance in question must be known. The paragraphs that follow describe the methods used by scientists to measure the relative toxicity of substances and to select exposure limits that will prevent exposed individuals from suffering adverse effects from such exposures. As this discussion demonstrates, methods of choosing exposure limits must, because of the lack or inadequacy of dose-response information for many chemicals, rely on experience in the use of these substances and on scientific and professional judgment.*

Chemicals range in inherent toxicity from those that are relatively harmless even after large doses have been administered to others that cause death if encountered even in small quantities. Toxicologists rank chemicals by categories that range from practically nontoxic (an adult human would have to consume a quart) to supertoxic (fewer than 7 drops would be lethal for most people).

In the occupational setting, it is the risk associated with a particular use of a chemical rather than its inherent toxicity that is important. Risk can be defined as the probability that a substance will produce harm under certain conditions of use. The converse of risk is safety, which is the probability that no harm will occur under specific circumstances.

The degree of hazard associated with exposure to a specific substance depends on the manner in which it is handled in a particular situation: a supertoxic chemical that is processed in a closed, isolated system may be less hazardous in actual use than a low-toxicity compound handled in an open batch process. Another factor affecting the ability of a chemical to elicit a toxic response is the susceptibility of the biological system or individual. To identify the relative degree of hazard in a particular instance requires knowledge about the chemical agent, the exposure situation, and the exposed subject. In addition, the route of administration and the duration and frequency of exposure must be known.

Route of Exposure

There are four principal routes of exposure by which toxic substances can invade humans or animals. These are inhalation, ingestion, dermal absorption, and parenteral administration (i.e., administration through routes other than the intestinal canal, e.g., by subcutaneous, intramuscular, intravenous, or intratesticular injection). The route of administration of a toxin also affects the relative toxicity of the agent. For example, a chemical that can be detoxified in the liver will be less toxic if it is administered orally than if it is given systemically (i.e., inhaled). Studies that provide information about the relative toxicity of an agent via different routes of exposure can provide a considerable amount of information about the absorbability of the agent. For example, if exposure to a certain dose of a chemical via all routes of administration causes death within the same time period, it can be assumed

*The material in this section derives principally from the following sources: Klaasen, Amdur, and Doull 1986; National Research Council 1988; Cohen 1988a,b; and Tardiff and Rodricks 1987.

that the substance in question is easily and rapidly absorbed. On the other hand, if the dermal dose of a chemical that is required to kill a subject is much higher than the dose required to produce the same effect when the chemical is ingested, one can deduce that the skin provides, to some degree, a barrier against that agent's toxicity. Other, less important, elements affecting the response to a toxic substance include the relative concentration of the substance, the volume of the vehicle used to administer the chemical, the chemical and physical properties of the vehicle, and the dose rate (i.e., the period of time over which the dose is administered).

Duration and Frequency of Exposure

Scientists conduct animal experiments that involve four different types of exposure: acute, subacute, subchronic, and chronic. Acute exposures are limited to periods of less than 24 hours and can involve either single or repeated exposures within that period. Subacute exposures are repeated exposures that last for one month or less, while subchronic exposures have a duration of one to three months. When a research project having a chronic regimen is conducted, the test animals are dosed repeatedly for a period lasting more than three months. Animals exposed acutely can have both immediate and delayed-onset responses. Similarly, chronic exposures can cause immediate reactions as well as long-term effects.

The frequency of dosing also has an important influence on the magnitude of the toxic effect: A large single dose of an acute toxin will usually have more than three times the effect of one-third the dose given at three different times, and the same dose administered in 10 or 15 applications might have no effect whatsoever. The pattern of dosing is important because it is possible for some of the substance to be excreted between successive administrations or because the lesion caused by the toxin has a chance to be partially or completely repaired between applications. Thus a chronic effect is said to occur: (1) If a toxic substance accumulates in the system of an exposed person or animal because the dose absorbed is greater than the body's ability to transform or eliminate the substance; (2) if it produces adverse effects that are not reversible; or (3) if it is administered in a manner that permits inadequate time for repair or recovery.

Variation in Response

Responses to toxic insults vary in a number of ways. For example, some toxicants have immediate effects, while others are associated with delayed symptom onset. The latency period for carcinogenic agents may be as long as 40 years for some types of cancer, and even some acute agents, such as some chemicals that have adverse ocular effects, may not cause overt symptoms until hours after exposure.

Another difference in type of response concerns the reversibility or irreversibility of the effect. Reversibility depends on the site of action as well as the magnitude of the insult. That is, some tissues of the body, such as the liver, have considerable ability to regenerate; others, like the kidney or central nervous system, do not.

The site of action associated with toxic substances also varies widely. Local effects are those lesions caused at the site of first contact between the agent and the organisms. Examples of localized effects are skin burns caused by contact with a caustic substance and site-of-contact tumors that develop at the locus of the injection of the carcinogen.

In contrast to localized effects, systemic effects involve the absorption and distribution of the toxic agent from the point of entry to a distant site; the toxic response is manifested at this distant point. An example of a systemic poison is mercury, which produces its toxic effect on the central nervous system. Often, the site of deposition for a chemical is not the organ system most affected by the toxin. For example, although lead is deposited and concentrated in the bone, it affects the central nervous system. Any sites that are adversely affected by the toxic effects of exposure to a substance, whether they are sites of contact or distal sites, are called the target organs of toxicity.

In cases of systemic poisoning, the system most often affected is the central nervous system (CNS); it is common for the CNS to be involved even when another organ, such as the liver, is the primary target organ of toxicity. In descending order of frequency, the systems or organs most often involved in cases of systemic poisoning are the central nervous system, the circulatory system, the blood and hematopoietic system, the visceral organs (liver, kidneys, lungs), and the skin.

Dose-Response

The relationship that associates the dose of a chemical with the effects it causes is called the dose-response relationship. A single data point relating a dose to a response is sufficient to establish a dose-response relationship. As additional data become available, it is possible to expand our understanding of the dose-response relationship to cover a range of doses or exposures. Dose-response is an important principle in toxicology, and an understanding of dose-response is important in establishing occupational or other exposure limits. Knowing how toxic substances act makes it easier to predict the potential effects of exposure. It is generally true that lowering dose reduces response; however, although data are often available to demonstrate that lower doses reduce responses on the grossly observable level, data showing that more subtle responses (e.g., those at the subcellular level) have been reduced are rarely available.

To apply dose-response relationships, it is helpful if several types of data are available. First, it must be possible to relate a response to a particular chemical. Although basic data pointing toward causality may be available, it is often difficult to refine the dose-response relationship further. For example, epidemiological studies often identify an association between a disease and one or more causative agents. However, since information on the precise identity of the etiologic agent, the actual dose received, and the true site of the response is usually not available, it is often impossible to use data from epidemiological studies to establish a precise dose-response relation between a specific dose of a toxin and an effect.

The second condition to be met before dose-response can be established is that it must be possible to relate the response to the dose. It is relatively easy to determine that a large dose causes an obvious response. Refining the relationship, however, involves three other requirements: (1) That there be a receptor site; (2) that the response and the intensity of the response be related to the concentration of the toxin at the receptor site; and (3) that the concentration of the toxin at the site be related to the dose given.

The third principle underlying the concept of dose-response is that there must be a quantifiable means of measuring the toxicity of a substance and a method of expressing this measured toxicity. Although lethality in test animals is often used to measure toxicity, the best form of measurement would involve quantification of the sequence of molecular events occurring during the toxic response. In the absence of such endpoints, other good methods are available. For example, it is common to measure an effect believed to be related to the substance in question. The level of activity of an enzyme in the blood is often used as a

measure of effect, e.g., serum glutamic-oxaloacetic transaminase (SGOT) levels are used to measure liver damage. Many different endpoints can be used to measure toxic effects, such as changes in muscle tone, heart rate, blood pressure, electrical activity of the brain, motor functioning, and behavior.

The most widely used endpoint, especially when a new substance is involved, is lethality in an animal test system. Lethality studies allow scientists to make comparative assessments of a chemical's toxicity as it relates to that of many other substances. Research of this type also permits the gathering of essential information on dose, duration, route of administration, site of action, and the target organ of toxicity.

Form of the Response

The classic form of dose-response is sigmoidal (Figure 1). This form characterizes the relationship between the amount of a toxin administered and the degree of response to that dose. The response is measured on the ordinate, and the dose is represented on the abscissa.

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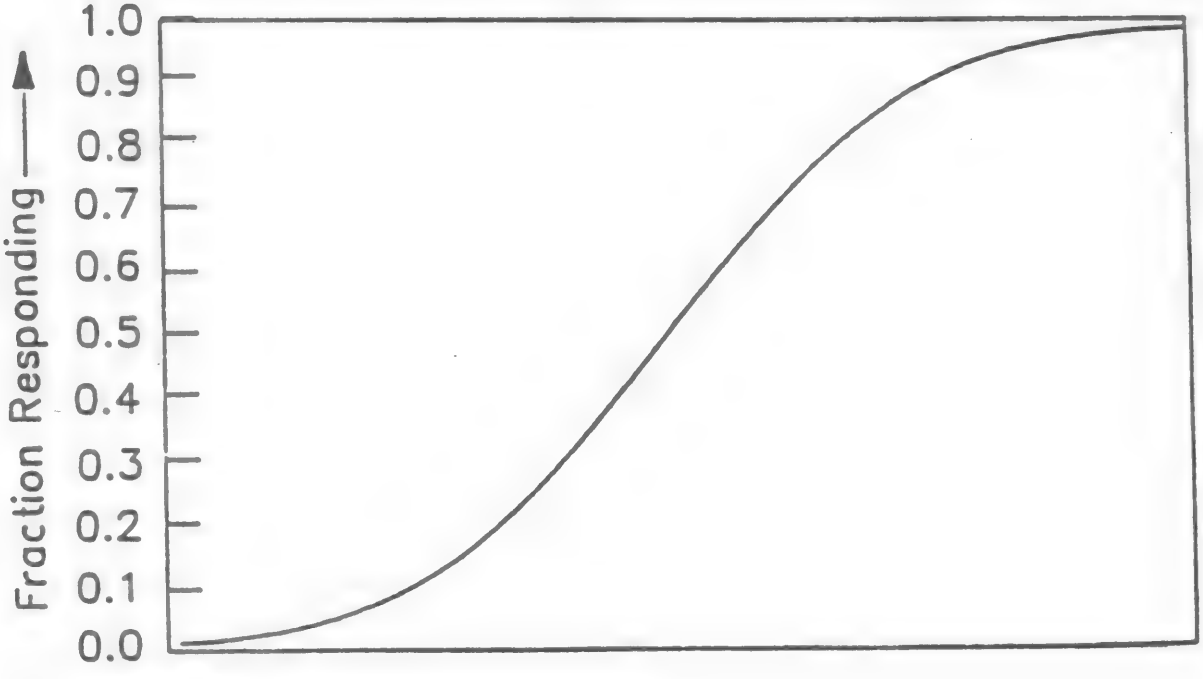
FIGURE 1. DIAGRAM OF THE DOSE-RESPONSE RELATIONSHIP

Figure 1

Diagram of Dose-Response Relationship



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Dose-response can be thought of in two ways:

- As exposure increases, the proportion of the population that manifests the response increases (quantal response); and
- As exposure increases, the intensity of an individual's response increases (graded response).

A relatively flat dose-response curve means that a large change in dose is required before there is a significant change in response. A steep curve, on the other hand, means that a small change in dose will elicit a large increase in response. Although it is sometimes possible to generate a curve of the type shown in Figure 1, it is not necessary to do so to demonstrate that exposure at a given level is associated with a particular response. That is, it is not necessary to have sufficient data to define, in mathematical terms, the dose-response relationship to know that exposure at a given level is associated with adverse consequences.

In the regulatory context, it is most common to express dose-response relations in terms of the percentage of the population responding. However, before this information can be evaluated, the endpoint being considered must be known. For every substance, there are several dose-response relationships, depending on endpoint: a substance that produces irritation at low doses may cause more severe symptoms or even death at high doses and in other conditions. For example, many substances that are mucosal irritants at low doses will produce pulmonary edema and nervous system effects at high doses.

Plotting the cumulative percentage of individuals responding against dose produces the typical sigmoid curve. Such a curve reflects the fact that at the lowest dose, zero percent of the population responds, while 100 percent of the population will respond at the highest dose. However, if the percentage responding is plotted against incremental rather than total dose, the curve produced is a normal distribution (Figure 2). This curve says that a relatively small percentage of the population will manifest the response at the lowest dose and that a similarly small percentage of the population will exhibit the effect only at the highest dose. What this normal distribution of response reflects is individual and species variation in exposed populations. A wide degree of variation occurs even in inbred, homogeneous laboratory animals, and such variability increases dramatically when a heterogeneous population, such as workers, is involved. Individuals responding at the left end of the curve shown in Figure 2 are sometimes called hypersusceptible, while those at the right end could be termed resistant. Because the relationship between dose and response is sigmoidal, response approaches zero as dose approaches zero. However, because of the mathematical form used to express this relationship, a true zero response can never be achieved. In the strictest sense, therefore, a true threshold dose level (i.e., the dose with which a zero response is associated) can never be established on the basis of experimental research. Instead, scientists attempt to define the minimum dose associated with a specific endpoint, which is customarily termed the "threshold" dose for that particular endpoint. However, unless a specific endpoint (such as respiratory irritation, cholinesterase inhibition, the development of a tumor, or death) is specified, the concept of a threshold is essentially meaningless. In fact, a separate threshold could be said to exist for each of these endpoints.

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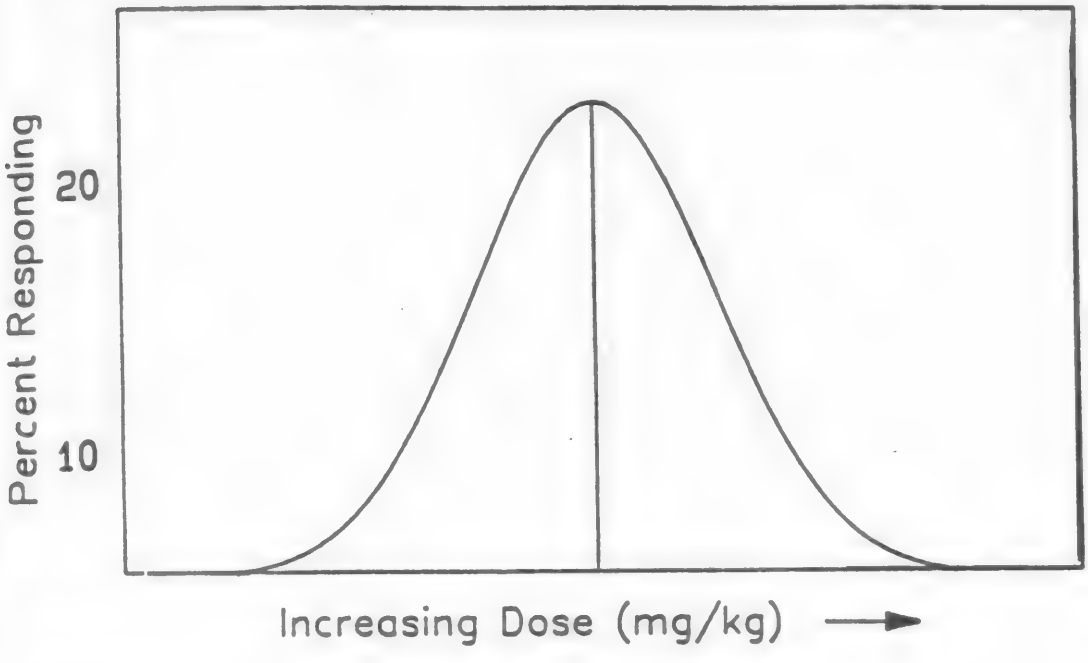
FIGURE 2. DIAGRAM OF QUANTAL DOSE-RESPONSE RELATIONSHIP

FIGURE 2

Diagram of Quantal Dose-Response Relationship



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The extent to which an experimentally derived "threshold" actually reflects the true threshold for a substance (i.e., the level above which a response will occur and below which no response will occur) depends on several factors, such as the number of animals used to determine the experimental threshold, the number of dose levels tested, and the degree of variation represented in the test subjects. For example, to determine an LD_{50} (the lethal dose that will kill 50 percent of the animals tested) with a high degree of precision requires the use of a minimum of 50 test animals and five dose groups (10 animals in each group). Other factors that can influence the magnitude of the median lethal dose include the sources involved, the sex and age of the animals, the environmental conditions prevailing during the test conditions, diet, the health status of the subjects being tested, and the subjects' past exposure to other toxic substances.

In toxicological research, the experimentally observed threshold dose is called the lowest-observed-effect level (LOEL) or the lowest-observed-adverse-effect level (LOAEL). Alternatively, the threshold may be expressed as the highest no-observed-effect level (NOEL), i.e., the highest dose administered and found not to produce a given response. Determination of an accurate NOEL requires both a careful interpretation of the toxicological data and the use of an adequate number of test animals. The National Academy of Sciences (1985) has concluded that the chance of finding a no-adverse-effect level (that is, of missing an adverse effect) at a given dose is statistically greater in experiments having a small number of animals than in studies involving a large number of animals. Thus, the degree of confidence one has that a NOEL actually represents a "safe" dose, rather than a research design artifact, increases with the number of animals tested. The greatest degree of confidence is associated with studies involving a large number of animals that were tested at several doses that were administered at close intervals.

In a recent publication (Tardiff and Rodricks 1987), David W. Gaylor of the National Center for Toxicological Research explained that experimentally derived thresholds represent statistical limitations in study design rather than biological characteristics:

> The existence of dose-response relationships might lead one to assume incorrectly the existence of threshold doses below which no toxic effects could occur. As dosage is decreased, the prevalence of an observable toxic effect * * * diminishes to zero. Eventually, a dosage is reached below which the experiment has essentially no resolving power to distinguish between the spontaneous background rate and small induced toxic effects * * *.
>
> If no toxic effects are detected at a specified dosage, this dosage is called the no-effect, or more correctly the no-observed-effect dosage. Because of the limitations of any given experiment, the no-observed-effect dosage is not a precise estimate of a true no-effect level. Lack of statistical significance is not equivalent to no toxic effect. It may or may not be, and further experimentation would be required to resolve this equivocal issue.* * * The no-observed-effect level is not a biological property, but, rather, a statistical property or operational threshold that is highly dependent on sample size.

The scientific issues surrounding the concept of no-observed-effect levels or experimentally derived thresholds have important implications for their use in establishing protective occupational exposure limits. Because the no-observed-effect level cannot represent the "true" threshold for an adverse effect, given the design of most toxicologic studies, regulators and others have used the concept of safety factors (also known as uncertainty factors) to aid them in setting permissible exposure limits; that is, the exposure limit is established at some interval below the no-observed-effect level to provide additional assurance that populations exposed at the level permitted are not likely to suffer harm.

The size of the interval between the permissible exposure limit and the no-observed-effect level depends on a professional judgment as to whether the no-observed-effect level is likely to represent a level that is not harmful to humans. Thus, if the available data include a NOEL derived from a well-conducted human study, a smaller safety factor might be used to establish an exposure limit than would be used if the data to be used to establish the limit consisted of a NOEL from an animal study; in the latter case, there is greater uncertainty regarding the relationship between the animal NOEL and human NOEL. Safety factors have also been used to recognize the fact that the human population is heterogeneous and that there may be a wide variation in individual responses to toxic substances (the wide range in the odor thresholds reported for some substances is a good illustration of individual variability in response).

The use of NOELs, LOAELs, and safety factors to develop permissible exposure limits is not a recent development:

> For more than half a century, evaluation of the safe use of chemicals has been focused mainly on the development of toxicity data and on the application of professional judgment to the ad hoc interpretation of such data to derive acceptable levels of exposure for humans. Generally, this practice has taken the form of identifying from studies in laboratory animals the no-observed-effect level and dividing it by a safety factor (usually 100 for NOELs derived from chronic studies) reflecting the uncertainties of relating data to humans under their conditions of exposure and the quality and appropriateness of the data base * * *.
>
> Safety factors are usually chosen prospectively to address the uncertainties of interspecies extrapolation. Although safety factors as small as 2 and as large as 2000 have been used * * * the safety factor of 100 is used most commonly, at least for NOELs derived from chronic toxicity studies, and incorporates adjustments for interspecies variability (usually 10) and intra-human variability (usually 10) * * *. The resulting value is equivalent to a NOEL in humans (Tardiff and Rodricks 1987, pp. 391, 421).

Many toxicologists urge that safety factors be used with care and interpreted with caution; they note that these factors "often create the [erroneous] impression that human population thresholds have been identified and that there is virtually no risk below that level of exposure" (Tardiff and Rodricks 1987, p. 421).

Although safety factors have traditionally been used to establish exposure limits for chronic or lifetime exposure situations, they have also been applied to establish limits for acute effects resulting from short-term exposure. For example, the National Academy of Sciences' Committee on Toxicology has been using a safety-factor approach to establish emergency exposure guidance levels (EEGLs), which are exposure levels judged to be acceptable for military personnel operating under emergency conditions. Exposure at an EEGL concentration would not be considered safe for routine or normal operations, but such exposures would be considered acceptable for performing tasks that would prevent even greater risks (e.g., death or injury caused by fires or explosions). When developing EEGLs, safety factors are used to account for uncertainties in the use of animal data and when extrapolating between different dose routes. The NAS also develops short-term public emergency exposure guidance levels (SPEGLs) that estimate acceptable levels of exposure for the general public during airborne chemical releases; SPEGLs are generally set at levels of one-tenth or one-half the EEGL (i.e., at levels incorporating an additional safety factor of from 2 to 10) (Criteria and Methods for Preparing Emergency Exposure Guidance Level (EEGL), Short-Term Public Emergency

Guidance Level (SPEGL), and Continuous Exposure Guidance Level (CEGL) Documents. Washington, DC: National Academy Press, National Academy of Sciences 1986).

In the prior air contaminants rulemaking, the use of the safety factor approach in establishing occupational exposure limits was addressed by many commenters (Exs. 3–744, 3–1095, 8–16, 8–47, 116, and 144; Tr. 1–221, Tr. 2–163 to 2–164). NIOSH (Ex. 8–47) pointed out that, since safety factors cannot be used to estimate human risk, they should not be interpreted as relating to the magnitude or significance of a risk; instead, as NIOSH notes, safety factors are intended only to reflect uncertainty in the available data. This comment echoes the concern expressed above, i.e., that safety factors should not be misinterpreted as identifying a human population threshold. NIOSH (Ex. 8–47) also endorsed OSHA's use of safety factors in generic rulemaking, noting that this approach is a "pragmatic method" of developing standards (except when a nonthreshold process, such as the induction of cancer, is the outcome of concern).

Dr. Marcus Key, Professor of Occupational Medicine at the University of Texas School of Public Health, also testified in the earlier rulemaking on the appropriateness of using safety factors to establish occupational exposure limits:

> We seldom, if ever, know with any precision where a significant risk level begins or ends; hence, the need for safety factors. Safety factors depend on several considerations, * * * mainly on toxicity and the nature of the health effects, but also on the availability of scientific evidence of effects at lower levels.
>
> Professional judgment must be relied on in selecting safety factors, with one to three orders of magnitude being commonly used for serious effects, and 50 percent, or [a] safety factor of 2, [being used] for acute, less harmful effects (Tr. 1–221).

Both Dr. Key (Tr. 1–221) and Dr. Ernest Mastromatteo, Chairman of the ACGIH TLV Committee (Tr. 2–163 to 2–164), testified that safety factors are frequently used by the American Conference of Governmental Industrial Hygienists to develop recommended exposure limits.

Other commenters in the earlier rulemaking (Exs. 8–16, 116, and 144; Tr. 7–121) urged OSHA to adopt a uniform system of assigning safety factors to establish permissible exposure limits. These participants suggested that OSHA apply the Reference Dose approach used by the Agency for Toxic Substances and Disease Registry or by the Environmental Protection Agency to calculate PELs for the Z-table substances. In response to these commenters, OSHA stated (54 FR 2399) that the Agency's approach has involved an evaluation of the efficacy of the proposed rule's limits on a case-by-case basis. OSHA believes that, at this time, this case-by-case assessment is the best way to establish new and revised limits for the numerous substances addressed in this proposed rulemaking.

Types of Toxicological Evidence

The evidence available to scientists who wish to evaluate the toxicity of a substance can be derived from studies in laboratory animals, *in-vitro* studies in cell or tissue systems, reports of clinical observations, studies of exposed human populations, or intervention studies conducted with human volunteers. The preceding paragraphs have described animal studies (or "bioassays"). The following section discusses the two most common types of human evidence: data derived from clinical observations and information from epidemiological studies.

Clinical Observations

Much of the data on the toxic effects associated with human exposures has come from industrial accidents, fatal poisonings, or other such tragedies. This information is generally more useful in delineating broad categories of pathological effects than in refining a specific dose-response relationship, because the exposure levels causing the accident are known to be high but cannot be quantified with precision.

Epidemiological Studies

Studies conducted by epidemiologists are designed to reveal the patterns of disease or mortality prevailing in certain groups of people (usually workers) exposed to a single toxin or to a group of substances. One of the advantages of epidemiological studies is that they involve humans and their responses to actual situations. The interpretation of the results of epidemiological studies is complicated by the inevitable presence of confounding variables that occur whenever human populations are involved. Ideally, the populations being studied (i.e., the study population and the control population) should be fully comparable with regard to every variable except the single characteristic under study. Because it is rarely possible to achieve this degree of comparability, statistical techniques are often used to attempt to adjust for this lack of comparability. In addition, if the measured effect is relatively large, it is unlikely that confounding factors will obscure the true picture.

Broadly speaking, epidemiological studies can have two possible outcomes: They can report an effect or they can report no effect; in the former case, the study is termed a positive study, and in the latter, a negative one. Within each of these categories, it is possible for the study to be correct (that is, to give a true-positive or true-negative result) or to be incorrect (that is, to give a false-positive or a false-negative result). A false-positive result reports that there is an increased risk when in fact there is not, and a false-negative study reports that there is no increased risk when in fact there is.

The probability that a study will detect a statistically significant effect if that effect is actually present is called the power of the study. As the power of a study increases, the likelihood of producing a false-negative error decreases. Power is dependent on two factors: the level of relative risk being evaluated and the number of cases of the effect (i.e., disease) that are expected in the population being studied. The number of expected cases depends both on the sample size and the expected disease frequency in the comparison population. For example, a study involving a small population and a common disease can have the same power as a study of a rare disease in a large population. Consequently, studies of larger samples have sufficient power to detect smaller increases in risk, and studies of smaller samples will be able only to detect large increases in relative risk.

Because epidemiological studies have limitations, it is essential that the power of such studies, particularly of negative studies, be examined to ensure that their sample sizes are adequate to detect the absence of increased risk with validity. When the power of a study is not adequate, negative studies cannot be said either to contradict or to support the conclusion that increased risk exists. Essentially, a negative epidemiologic study identifies a NOAEL, which, as discussed above, reflects the statistical limitations of a study more than the "true" population threshold for an effect. However, a study with a positive result may indicate a relationship if the excess risk is high, even if the study's sample size is small and the effects of some factors are not controlled for.

Quality of Evidence

Dose-response models have often been used in the quantitative assessment of the risks associated with exposures to carcinogenic substances. However, less scientific effort has been devoted to models to be used with non-

carcinogenic substances. Mathematically precise methods to establish the true no-effect level or to define the dose-response curve have not been developed for most of the more than 400 substances involved in this rulemaking.

Most of the scientific work that has been done was designed to identify lowest-observed-effect or no-effect levels for a variety of acute effects. As described above, experts in industrial hygiene and occupational health have developed factors to be used to offset, at least to some extent, the insensitivity of NOELs and LOELs to such factors as subcellular effects, sensitive individuals, and chronic exposure. It is possible to use these data, combined with professional judgment and OSHA's expertise and experience, to determine that significant risk exists at current levels of exposure and that a reduction in these levels will substantially reduce this risk of material impairment of health. OSHA is also confident that it is not attempting in this rulemaking to reduce exposures to insignificant levels. However, additional analysis may well reveal that the levels being proposed can be refined further in the future.

B. Historical Development of Occupational Exposure Limits

Early Limits

Until the development of occupational health standards, the occurrence of adverse health effects resulting from exposures to hazardous substances or conditions in the workplace could only be determined *ex post facto*—after material impairment had already occurred to the health and welfare of exposed employees. For example, in her 1910 studies of lead poisoning, Dr. Alice Hamilton was forced to rely on "personal observations of working conditions and the illness and deaths of workers to demonstrate the existence of harmful exposures" (Paull 1984/Ex. 1–255). The concept of occupational exposure limits thus represents a dramatic breakthrough in the battle against occupational disease, and these limits remain "one of the most useful and indispensable tools yet devised for safeguarding the health and well-being of industrial workers" (Thomas 1979/Ex. 1–96). Occupational exposure limits are air quality values that apply in workplaces, and they are derived by studying the correlation between the amount of a toxic substance absorbed by the body and its effects on health. Within the context of occupational exposure, knowledge of this relationship permits quantification of the etiology "of a large number of occupational health impairments, [evaluation of] the risk of such impairments and, if necessary, [consideration of] the effectiveness of preventive measures" (Parmeggiani 1973/Ex. 1–229). More specifically, an understanding of the levels at which disease or other adverse health effects occur can be used to establish limits for occupational exposure that reflect concentrations below which health hazards are unlikely to occur in most workers.

The historical development of occupational exposure limits began with the published reports of a German scientist whose investigations in 1883 into the effects on experimental animals (and on himself) of exposure to carbon monoxide at known air concentrations caused him to conclude that "the boundary of injurious action of carbon monoxide lies at a concentration in all probability of 500 parts per million, but certainly [not less than] 200 parts per million" (Cook 1987/Ex. 1–187). Shortly after the appearance of this first documented dose-response value, another German researcher, K. N. Lehmann, published a series of reports on a number of chemical substances under the title "Experimental Studies on the Effect of Technically and Hygienically Important Gases and Vapors on the Organism." This series culminated in 1936 in a comprehensive paper on chlorinated hydrocarbons, published as Volume 116 of *Archiv für Hygiene.*

In 1912, Rudolf Kobert published a table of exposure limits, based on animal studies, for 20 compounds. One of the first tables of hazardous air concentrations to originate in the United States was a technical paper published in 1921 by the U.S. Bureau of Mines. The 33 substances included in this table were those frequently encountered in the workplace. In addition to limits based on the avoidance of acute toxic effects, this table provided some information on the least detectable odor concentration and the lowest airborne concentration required to cause irritation (Paull 1984/Ex. 1–255; Cook 1987/Ex. 1–187).

Throughout the 1920s and 1930s, data became available that correlated concentrations of harmful substances with observed effects on worker health for such materials as lead and mercury compounds, benzene, and granite dusts. These early occupational health studies, which were based on animal experiments and on findings in exposed workers, provided the kind of data needed to link human exposures "to concentrations that were capable of producing not only acute, but chronic health effects" (Paull 1984/Ex. 1–255).

After 1935, the emphasis of researchers had shifted, for the most part, from the reporting of a series of values for a range of acute effects to results that yielded a single limit based on studies of repeated exposures. Over the years, a sizable amount of data about the levels of exposure that would not produce injurious effects had been amassed for a considerable number of substances. "By the early 1940s, control of the occupational environment to prevent the harmful absorption of toxic materials was becoming an accepted principle, and the practical problem of defining what was 'harmful' was beginning to be met by employing maximum allowable concentrations" (Paull 1984/Ex. 1–255). In 1943, Sterner (Ex. 1–806) explained the meaning of the term maximum allowable concentrations as "the upper limit of concen-tration of an atmospheric contaminant which will not cause injury to an individual exposed continuously during his working day and for indefinite periods of time" (Paull 1984/Ex. 1–255).

The first lists of maximum allowable concentrations of airborne toxic substances were issued between 1933 and 1938. The Union of Soviet Socialist Republics (U.S.S.R.) was the first country to make occupational exposure limits a statutory obligation; in 1933 the U.S.S.R. published a list that included 14 substances. The first American list was published four years later by the State of Massachusetts, and in 1938 Germany issued occupational health standards for a number of organic solvents (Holmberg and Winell 1977/Ex. 1–141). Additionally, the United States "imposed limited occupational safety and health requirements on certain contractors with the Federal government" when the Walsh-Healey Act was passed in 1936 (Mintz 1984/Ex. 1–840).

Standards Developed by Professional Organizations

During the 1940s, American organizations led in the development of occupational health standards, beginning with the American Standards Association (now the American National Standards Institute, or ANSI) list of "maximum acceptable concentrations" (MACs), which appeared in 1941. This list represented a consensus of opinion by the ASA and a number of industrial hygienists who had formed the American Conference of Governmental Industrial Hygienists (ACGIH) in 1938 (Baetjer 1980/Ex. 1–

223). Originally conceived of as a time-weighted concentration to be maintained as an average over an entire work shift, the MAC was redefined in 1957 to mean an upper level (i.e., a ceiling concentration) that should never be exceeded (Turner 1976/Ex. 1–79).

An important contribution to occupational health standard-setting was made in 1945 by Warren Cook (Ex. 1–726), who published a list of maximum allowable concentrations for 132 industrial atmospheric contaminants. These limits had been developed by six states, the U.S. Public Health Service, and the American Standards Association, and included Cook's own list of "accepted or tentative values" based on industrial experience, animal experimentation, human sensory response, or a combination of these factors. This table was followed by

> Documentation supported by 187 specific references, indicating the basis and reliability of each value. Cook was the first investigator to codify all of the available data on MAC's and present it in one publication. His list of recommended values was incorporated, practically without changes, by the ACGIH in establishing the TLVs. In support of Cook's inferences, it should be noted that 50 of the * * * values that he recommended in 1945 were subsequently adopted as federal standards, and are still in use today (Paull 1984/Ex. 1–255).

The American Conference of Governmental Industrial Hygienists' Subcommittee on Threshold Limits presented its second report at the Eighth Annual Meeting of the ACGIH in 1946. The report included values for 131 gases, vapors, dusts, fumes, mists, and 13 mineral dusts "compiled from the list reported by this subcommittee * * * in 1942, from the list published by Warren Cook in * * * 1945, and from published values of the Z-37 Committee of the American Standards Association" (Cook 1987/Ex. 1–87). The Committee's report noted that:

> Considerable difficulty attends the fixing of satisfactory values for maximal allowable concentrations of chemicals in respirable atmospheres because of the lack of a uniform definition of the maximum allowable concentration concept. One concept is that the M.A.C. value should represent as accurately as possible that concentration at which a worker exposed for a sufficient period of time will just escape physiological or organic injury and occupational disease.
>
> A second concept is that the M.A.C. should represent some fraction of that concentration which will injure the worker in order to allow a margin of safety in the design of protective equipment and guard against possible synergistic effects in the case of multiple exposures. A third concept is that the M.A.C. should perform the functions of the former concepts and in addition provide a work environment free of objectionable but non-injurious concentrations of smokes, dusts, irritants and odors. Obviously all of these concepts cannot be fulfilled with the establishment of a single value. M.A.C. values in use at the present time represent examples of all of these concepts. The committee feels that the establishment of dual lists or a single definition is not possible at the present time (ACGIH 1946).

The report concluded by stressing that the 1946 list of MAC values was presented "with the definite understanding that it be subject to annual revision" (ACGIH 1946).

Papers presented at both the Ninth International Congress on Industrial Medicine in London (1948) and at the Fifteenth International Congress of Occupational Health in Vienna (1966) also dealt with maximum acceptable concentrations. The first of these proposed that zones of toxicity be set up to facilitate an understanding of the relative hazards of substances, "since the boundaries of MAC values were not sharp lines of demarcation" (Cook 1987/Ex. 1–87). At the 1966 meeting, discussion took place on the advantages of the concept of a "peak level" of exposure—an extension of the "ceiling level" notion inherent in the definition of a MAC since 1957. A "peak level" was defined as one "that can be applied to certain substances for brief designated periods and for a strictly limited number of times during the work shift, with a designated time interval between peaks. The 'peak' concept places a limit on the intermittent higher exposures that occur in many industrial operations. The time-weighted average exposure limit is of course to be observed [even when a peak has also been assigned to a substance]" (Cook 1987/Ex. 1–87).

Terminology and definitions throughout this early period were ambiguous and imprecise, reflecting uncertainty as to exactly what needed to be and could be done in the realm of occupational health standard setting. Initially, the ACGIH designated its recommended limits as "maximum allowable concentrations," although this term was often used interchangeably with "threshold limit values." Confusion about the meaning, interpretation, and relative significance of the terms being employed during this embryonic period was common. After 1953, the ACGIH defined the concept of threshold limit value in the preface to its annual published list of occupational health standards as "maximum average atmospheric concentrations * * * for an eight-hour day." Documentation for the 238 substances included in the TLV® list for 1956 was provided by Smyth (Ex. 1–759) in a separate paper in which the author

> Recommended that the TLVs include references to the underlying data, and that the concepts represented by the values be restated in more realistic toxicological terms. In his analysis of the TLVs, he [Smyth] concluded that nine categories of objectionable action were guarded against: chronic toxicity, acute toxicity, narcosis, irritation, asphyxiation, fume fever, eye pigmentation, allergic response, and cancer (Paull 1984/Ex. 1–255).

At about the same time, Stokinger stated that, in his opinion, the Threshold Limits Committee had avoided grappling with the issue of developing a method for establishing limits for industrial carcinogens and noted that, with the exception of nickel carbonyl, limits had not been assigned for potential carcinogens (Paull 1984/Ex. 1–255). In 1962, however, the TLV Committee added three carcinogens to the TLV list, although these were listed separately in an appendix and did not have assigned numerical values.

Despite the fact that the ACGIH had stressed early on that TLVs® were intended as guides and not as rigidly enforceable limits, the American Standards Association's MAC values (or, where none was available, the TLV®) were included as mandatory limits in the Safety and Health Standards for Federal Supply Contracts, which were published in 1960 under the Walsh-Healey Act. Following this action, the ACGIH issued a statement on the definitions and interpretations of TLVs® and MACs (Stokinger 1962/Ex. 1–998). At the same time, the ACGIH announced the production of the first edition of the Documentation for Threshold Limit Values (ACGIH 1962); this was followed by another paper in which the work and intentions of the Threshold Limits Committee were reviewed. Turner states that

> [A]t this time the concept of ceiling values and excursion factors around the time-weighted average values was introduced in order to reduce conflict or confusion with the "maximal" values in the American [ANSI] Standards. A "C" (ceiling value) listing was to be given to those fast-acting substances thought likely to be injurious if the concentration exceeded the limit value by more than a designated factor for a relatively short period (about 15 min.). The factor varied between 3 and 1.25, depending inversely upon the magnitude of the TLV. A corollary was that the factor would also indicate the limit of permissive excursion of the concentration above the TLV for a substance not given a "C" listing, always provided that the time-weighted average concentration did not exceed the TLV. This rule of thumb approach to limiting exposure is no doubt appropriate to certain substances

when they are used routinely throughout the working day. It seems to have little relevance in other instances where exposure is irregular or where the basis for fixing the TLV is on grounds other than toxicity (Turner 1976/Ex. 1–79).

Permissible Exposure Limits in the Era of OSHA

Shortly after the Occupational Safety and Health (OSH) Act became effective, OSHA issued standards that would provide the Agency with a basis for promptly commencing its enforcement program. Congress gave OSHA the authority under section 6(a) of the Act to promulgate certain standards without rulemaking, i.e., without the delays inherent in public comment proceedings. These so-called "start-up" standards derived from national consensus standards and established Federal standards, i.e., ANSI, the National Fire Protection Association (NFPA), the Walsh-Healey Public Contracts Act, the Longshoremen's and Harbor Workers' Compensation Act (LHWCA), and the Contract Work Hours and Safety Standards Act of 1969 (also known as the Construction Safety Act).

The startup standards adopted by OSHA for general industry in this early period derived from the ACGIH 1968 list of TLVs® that had been incorporated into the Walsh-Healey Act, and those adopted for construction derived from the 1970 ACGIH TLVs® that had been incorporated into the Construction Safety Act. (There are thus a few differences in the PELs OSHA applies in general industry and construction, since the ACGIH had updated its TLVs® for a few substances when it issued its 1970 list.) In agriculture, however, OSHA did not adopt startup standards and, in the intervening years, the Agency has not, until now, corrected this deficiency (see the discussion in the History and Need for Revision of the PELs section of this preamble). In the interval since the establishment of OSHA and the adoption of the 1970 ACGIH TLVs® and a few ANSI limits for construction and maritime, the ACGIH has continued to revise, update, and document its annual list of TLVs®; since 1988, this process has been an annual one. During this time, the ACGIH TLVs® have been "accepted on an international basis as the best available guides for providing healthful occupational environments, and at least 18 countries, including the United States, have either adopted them as legal standards or as guides to legal action, thus verifying their efficacy in accomplishing this purpose" (Paull 1984/Ex. 1–255).

In 1989, in the largest rulemaking ever undertaken by the Agency, OSHA updated and supplemented its list of permissible exposure limits by considering new or more protective exposure limits for 428 toxic substances; these new and revised PELs apply in general industry workplaces. The action OSHA takes today initiates the process of extending these new and revised limits to workplaces in the construction, maritime, and agriculture industries. That the limits being proposed for revision and updating are seriously out of date is attested to by the fact that the ACGIH has found it necessary to revise or to add nearly 400 limits to its list of TLVs® in the 20 years since the limits adopted by OSHA in 1971 and still in force in these sectors were initially published. Recognition that OSHA's permissible exposure limits in construction and maritime need updating to reflect recent developments in toxicology and new data on the health effects associated with exposure to these substances is nearly universal and is evidenced by the widespread support for the recent air contaminants rulemaking that updated and revised OSHA's PELs for general industry.

The Agency believes that promulgation of the proposed limits will address a broad range of significant risks now prevalent in the construction, maritime, and agriculture industries. As many industrial hygienists and occupational safety and health professionals have noted, the use of permissible exposure limits continues to be the single most effective way of protecting the health, functional capacity, and well-being of the American worker.

C. Description of the Substances for Which Limits Are Being Proposed

In this rulemaking, OSHA is proposing to revise or to add PELs for approximately 588 toxic substances and to apply these limits to workplaces in the construction, maritime, and agriculture industries. OSHA recently completed this action for general industry (see 54 *FR* 2332 et seq.). This section of the preamble identifies the PELs being proposed for construction, maritime, and agricultural workplaces, describes the available toxicological data, and explains the Agency's rationale for selecting the permissible exposure limits proposed for these substances.

The universe of substances included in this rulemaking is bounded by the substances that OSHA recently considered in the general industry Air Contaminants rulemaking that amended 29 CFR 1910.1000. That is, OSHA is not at this time proposing exposure limits for any hazardous substance that was not considered in that rulemaking, with the exception of a group of approximately 160 substances. For these 160 substances, OSHA's current limits in general industry, construction, and maritime are, with very few exceptions, identical and are believed by the Agency already to be at adequately protective levels. However, since OSHA has never had PELs that applied in agricultural workplaces, the Agency is proposing to apply its PELs for these 160 substances, as well as those for the other substances described above, to agriculture for the first time. In addition, OSHA's interpretation is that the exposure limits included in Tables Z–1, Z–2, and Z–3 apply in longshoring. However, the language at 29 CFR 1918.93 (e) and (f) is somewhat ambiguous. Accordingly, OSHA intends in the present rulemaking to eliminate this ambiguity and to make all such limits applicable to the longshoring sector.

For all of the substances addressed in this rulemaking, OSHA has already evaluated in great detail the studies, public comments, and evidence in the prior rulemaking record for general industry. The levels proposed in this rulemaking are the same as those that OSHA determined to be justified in the general industry rulemaking. Therefore, the limits being proposed today represent, in the Agency's professional judgment, those levels found to be most consistent with the best available toxicological data, OSHA's mandate, and the case law that has subsequently developed to interpret that mandate. (For a discussion of the relevant legislative and judicial principles, see the sections of this preamble entitled Pertinent Legal Authority, History and Need for Revision of the PELs, and Approach).

For ease of analysis and presentation, the substances included in the scope of this rulemaking have been grouped into 16 separate sub-sections. In general, these groupings (sections IV.C.1 through IV.C.14) reflect the primary toxicological basis underlying the ACGIH or NIOSH recommended limits for these substances. In addition, two additional sections (Sections IV.C.15 and 16) cover substances for which OSHA is proposing to add short-term limits and those for which the Agency is proposing to add skin notations.

The following sections are included:

1. Substances for which Proposed Limits Are Based on Avoidance of Neuropathic Effects
2. Substances for which Proposed Limits Are Based on Avoidance of Narcotic Effects

3. Substances for which Proposed Limits Are Based on Avoidance of Sensory Irritation
4. Substances for which Proposed Limits Are Based on Avoidance of Liver or Kidney Effects
5. Substances for which Proposed Limits Are Based on Avoidance of Ocular Effects
6. Substances for which Proposed Limits Are Based on Avoidance of Respiratory Effects
7. Substances for which Proposed Limits Are Based on Avoidance of Cardiovascular Effects
8. Substances for which Proposed Limits Are Based on Avoidance of Systemic Toxicity
9. Substances for which Proposed Limits Are Based on Observed-No-Adverse-Effect Levels
10. Substances for which Proposed Limits Are Based on Avoidance of Physical Irritation and Other Effects
11. Substances for which Proposed Limits Are Based on Analogy to Related Substances
12. Substances for which Proposed Limits Are Based on Avoidance of Biochemical/Metabolic Effects
13. Substances for which Proposed Limits Are Based on Avoidance of Sensitization Effects
14. Substances for which Proposed Limits Are Based on Avoidance of Cancer
15. Substances for Which 1987–1988 ACGIH TLV® Are Less Stringent Than Existing OSHA PELs
16. Substances for Which OSHA Is Proposing Short-Term Exposure Limits
17. Substances for Which OSHA Is Proposing to Add Skin Notations.

A list of the references that OSHA is relying on in evaluating the toxicological evidence pertaining to these chemicals appears in section IV–D.

1. Substances for Which Proposed Limits Are Based on Avoidance of Neuropathic Effects

Introduction. Many industrial chemicals have been shown to cause severe neurological effects in exposed workers, and these effects are often irreversible. The proposed limits for 25 substances have been set at levels established to avoid neuropathic effects in exposed workers. Table C1–1 lists these neuropathic agents, along with their CAS numbers, HS numbers, 1987–1988 ACGIH TLV®s, and NIOSH RELs. In addition, Table C1–1 shows OSHA's current PELs for these substances in construction, shipyards, marine terminals, and longshoring operations. OSHA currently has no PELs for these substances in agriculture. The right-hand column in Table C1–1 shows the current PEL for each substance in general industry; these are the limits being proposed today for these substances in construction, maritime, and agricultural workplaces. Promulgation of these limits will make OSHA's PELs for these substances consistent across all regulated sectors.

TABLE C1–1.—SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON AVOIDANCE OF NEUROPATHIC EFFECTS

H.S. No./chemical name	CAS No.	Current OSHA PEL in construction, shipyards, marine terminals, and longshoring *	1987–1988 ACGIH TLV **	NIOSH REL †	Proposed OSHA PEL in construction, shipyards, marine terminals, longshoring, and agriculture*
2019 Biphenyl (Diphenyl)	92-52-4	0.2 ppm TWA	0.2 ppm TWA		0.2 ppm TWA.
1051 n-Butyl alcohol	71-36-3	100 ppm TWA	50 ppm Ceiling, Skin		50 ppm Ceiling, Skin.
1078 Chlorinated camphene	8001-35-2	0.5 mg/m³ TWA, Skin.	0.5 mg/m³ TWA, 1 mg/m³ STEL, Skin.		0.5 mg/m³ TWA, 1 mg/m³ STEL, Skin.
1114 Decaborane	17702-41-9	0.05 ppm TWA, Skin.	0.05 ppm TWA, 0.15 ppm STEL, Skin.		0.05 ppm TWA, 0.15 ppm STEL, Skin.
2054 Diborane	19287-45-7	0.1 ppm TWA	0.1 ppm TWA		0.1 ppm TWA
1116 Di-sec-octyl-phthalate (Di-ethylhexyl-phthalate).	117-81-7	5 mg/m³ TWA	5 mg/m³ TWA, 10 mg/m³ STEL	(††)	5 mg/m³ TWA, 10 mg/m³ STEL.
1123 Dichloroacetylene	7572-29-4	0.1 ppm Ceiling	0.1 ppm Ceiling		0.1 ppm Ceiling.
1149 Dipropylene glycol methyl ether.	34590-94-8	100 ppm TWA, Skin.	100 ppm TWA, 150 ppm STEL, Skin.		100 ppm TWA, 150 ppm STEL, Skin.
1200 n-Hexane	110-54-3	500 ppm TWA	50 ppm TWA	100 ppm TWA, 510 ppm, (15-min) Ceiling.	50 ppm TWA.
1202 2-Hexanone (Methyl n-butyl ketone).	591-78-6	100 ppm TWA	5 ppm TWA	1 ppm TWA	5 ppm TWA.
1216 Iron pentacarbonyl (as Fe)	13463-40-6		0.1 ppm TWA, 0.2 ppm STEL		0.1 ppm TWA, 0.2 ppm STEL.
2100 Lindane	58-89-9	0.5 mg/m³ TWA, Skin.	0.5 mg/m³ TWA, Skin		0.5 mg/m³ TWA, Skin.
2103 Manganese, dust and compounds.	7439-96-5	5 mg/m³ Ceiling	5 mg/m³ Ceiling		5 mg/m³ Ceiling.
1236A Manganese, fume (as Mn)	7439-96-5	5 mg/m³ Ceiling	1 mg/m³ TWA, 3 mg/m³ STEL		1 mg/m³ TWA, 3 mg/m³ STEL.
1237 Manganese cyclopentadienyl tricarbonyl (as Mn).	12079-65-1		0.1 mg/m³ TWA, Skin		0.1 mg/m³ TWA, Skin.
1238 Manganese tetroxide (as Mn).	1317-35-7		1 mg/m³ TWA		1 mg/m³ TWA.
1240 Mercury (aryl and inorganic compounds) (as Hg).	7439-97-6	0.1 mg/m³ TWA, Skin.	0.1 mg/m³ TWA, Skin*	0.05 mg/m³ TWA	0.1 mg/m³ Ceiling, Skin.
1241 Mercury, vapor (as Hg)	7439-97-6	0.1 mg/m³ TWA, Skin.	0.05 mg/m³ TWA, Skin	0.05 mg/m³ TWA	0.05 mg/m³ TWA, Skin.
1242 Mercury, (organo) alkyl compounds (as Hg).	7439-97-6	0.01 mg/m³ TWA, Skin.	0.01 mg/m³ TWA, 0.03 mg/m³ STEL, Skin.		0.01 mg/m³ TWA, 0.03 mg/m³ STEL, Skin.
2104 Methyl acetylene	74-99-7	1000 ppm TWA	1000 ppm TWA, 1250 ppm STEL		1000 ppm TWA.
1251 Methylacrylonitrile	126-98-7		1 ppm TWA, Skin		1 ppm TWA, Skin.
1253 Methyl bromide	74-83-9	20 ppm Ceiling, skin.	5 ppm TWA, Skin	(††)	5 ppm TWA, Skin.
1304 Pentaborane	19624-22-7	0.005 ppm TWA	0.005 ppm TWA, 0.015 ppm STEL.		0.005 ppm TWA, 0.015 ppm STEL.
1316 Phenyl mercaptan	108-98-5		0.5 ppm TWA		0.5 ppm TWA.

TABLE C1-1.—SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON AVOIDANCE OF NEUROPATHIC EFFECTS—Continued

H.S. No./chemical name	CAS No.	Current OSHA PEL in construction, shipyards, marine terminals, and longshoring *	1987-1988 ACGIH TLV **	NIOSH REL †	Proposed OSHA PEL in construction, shipyards, marine terminals, longshoring, and agriculture*
1342 - 1,2-Propylene glycol dinitrate.	[illegible]	.....	0.05 ppm TWA, Skin	.....	0.05 ppm TWA.

* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time; OSHA's PELs do not currently apply in Agriculture.

** The ACGIH TLV®-TWA is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

† NIOSH TWA limits are for 10 hour/day, 40 hour/week exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

‡ NIOSH considers this substance a potential occupational carcinogen and recommends that exposures be reduced to the lowest feasible concentration.

* The OSHA limit for the aryl and inorganic compounds of mercury derived from an American National Standards Institute (ANSI) standard rather than the ACGIH TLV®.

Description of the health effects. The human nervous system comprises the central nervous system (CNS) and the peripheral nervous system (PNS). The CNS is made up of the brain and spinal cord, while the PNS consists of a network throughout the body of nerves that communicate with the CNS via connections to the spinal cord. The brain and spinal cord are bathed in cerebrospinal fluid, which supplies nutrients to the CNS and also acts as a barrier against the entry of some foreign substances. This barrier protects the central nervous system. In general, fat-soluble substances readily diffuse across this barrier and water-soluble substances do not.

Chemicals that affect the central nervous system can manifest their toxic effects peripherally. An example of this is the tremor associated with elemental and organic mercury poisoning. Exposure to some neurotoxic chemicals (for example, to n-hexane) is associated with axonal degeneration of the nerves in both the central and peripheral nervous systems. Baker (1983/Ex. 1-230) refers to this dual-system effect as central-peripheral distal axonopathy.

Nervous system toxicants can affect motor function, sensory function, or integrative processes, and they can also cause changes in the behavior of exposed persons. Substances that cause demyelination or neuronal damage can produce motor dysfunction that is expressed as muscular weakness or unsteadiness of gait, while exposures to chemicals that are associated with loss of sensory function may result in alterations in touch, pain, or temperature sensation or damage to sight or hearing. Other neuropathic chemicals affect the way in which information is processed in the brain and can interfere with learning and memory. OSHA believes that all of the health effects described above constitute material impairments of health within the meaning of the Act.

Although mature neurons cannot divide and be replaced, the nervous system has considerable ability to restore function that is lost as a result of exposure to toxic chemicals. This capability to restore function even after neurons have been killed is achieved by two mechanisms: Plasticity of organization and redundancy of function; that is, when some neurons die, other cells that perform the same function may be able to maintain an adequate level of functioning, or other neurons may be able to "learn" how to perform the lost function. However, even when one of these mechanisms comes into play to compensate for neuronal damage, the overall reserve capacity of the nervous system will have been diminished. The loss of this reserve could be critical in a situation in which additional demands are placed on the nervous system. Thus, even so-called reversible neuropathic effects should be seen as toxic effects causing alterations in and material impairment of the normal functioning of the nervous system.

The neurological effects potentially associated with chemical exposures are numerous, and it is not always easy to identify the precise target site. However, recent medical advances have made tests available that can detect neurological damage that was not detectable several years ago. For example, electrophysiological methods have been developed to measure damage to the visual pathway caused by such exposures. Because of the variation in individual responses to chemical exposures, exposure limits should be set with a view toward this range of susceptibility and the avoidance of any neuropathic effects.

Peripheral nervous system effects. The pathological mechanisms associated with peripheral neuropathies result from segmental demyelination or axonal degeneration. Segmental demyelination destroys the myelin sheath but leaves the axon intact; this causes a slowing in nerve conduction velocity. Muscle weakness is often the first sign of such segmental demyelination, and this effect can progress to loss of motor function or paralysis. Although remyelination may occur within weeks after injury, even a temporary loss in motor or sensory function may place the affected worker (and his or her co-workers) at risk of injury.

Axonal degeneration is a more serious effect because recovery is often slow or incomplete. This type of degeneration causes demyelination secondary to the degeneration of the distal portion of the nerve. This effect occurs when a chemical interferes with the physiologic dynamics of the nerve, e.g., when it decreases the transport of nutrients to the nerve; in such cases, the axon degenerates (dies back) sufficiently to accommodate the cell's capacity to supply it with nutrients. Axonal degeneration can also occur as a result of biochemical or metabolic derangement of the central nervous system. Alkyl mercury and elemental mercury are examples of neurotoxic chemicals causing this type of effect (Cavanaugh 1977/Ex. 1-202).

Central nervous system effects. The mechanism of action of central nervous system toxins is not well understood but is believed to be associated with neurochemical alteration in the brain. Seizures, Parkinsonism, intellectual impairment, narcosis, dementia, cranial neuropathy, and visual disturbances are all examples of effects that can occur after overexposures to neuropathic chemicals. The more serious CNS effects, such as Parkinsonism, dementia, intellectual impairment, and cranial neuropathy, are generally not reversible (Baker 1983/Ex. 1-230). Before these

exposure-related effects are manifested, subtle changes in behavior may occur; if these subtle signs are interpreted correctly, exposure can be stopped before irreversible damage occurs.

Dose-response relationships and neuropathic effects. The development of chemically induced neurological effects is believed to follow a dose-response pattern. At an exposure intensity or duration below the no-effect level, detectable effects are unlikely to be evident. As exposure intensity/duration increases to and beyond this level, the toxin begins to interfere with the normal cellular processes of the neurological system. At this early stage, transient signs and symptoms may appear. Overt effects become more severe as exposure continues and finally progress to serious loss of neurological function and possible permanent damage to neural tissue. Increases in our ability to detect neurological changes at lower levels of exposure have shown that neurobehavioral changes or impairment may occur at levels previously thought to be innocuous. These early effects can be important indicators of potential functional impairment at exposure levels below those that produce either transient or permanent damage. Heavy metals, solvents, and pesticides are examples of substances that can cause symptoms and conditions such as nausea, sensory and motor function impairments, depression, sleep disturbances, cognitive impairment, and sexual dysfunction. The limits being proposed for the substances in this group of neurotoxins are designed to maintain worker exposures below the level associated with such adverse health effects. OSHA believes that this approach will ensure that employees will not be likely to suffer these material impairments of health and will provide a margin of safety against the risk of more severe or permanent neurological impairment.

The following discussions describe OSHA's preliminary findings for all of the substances in this group and illustrate the material impairments of health potentially faced by workers exposed to these toxicants.

BIPHENYL (DIPHENYL)

CAS: 92–52–4; Chemical Formula: $C_6H_5C_6H_5$

H.S. No. 2019

In general industry, construction, and maritime, OSHA's current permissible exposure limit for biphenyl (also called biphenyl) is 0.2 ppm as an 8-hour TWA. There is no current limit for this substance in agriculture. The ACGIH has a TLV®–TWA of 0.2 ppm for biphenyl. NIOSH has no REL but concurs with the PEL being proposed. OSHA is proposing an 8-hour TWA PEL in agriculture of 0.2 ppm for biphenyl. This is the limit recently established for this substance in general industry.

Biphenyl is a solid substance that is crystalline in form and ranges in color from colorless to light yellow (Hawley's 1987, p. 426; AIHA 1964). Biphenyl has a distinct aromatic odor and occurs most commonly in the form of a white, scaly solid (ACGIH 1986, p. 58; AIHA 1964). Biphenyl is used as a fungistat in the packaging of citrus fruits, for plant disease control, in organic synthesis, as a heat-transfer agent, and as a dyeing assistant for polyesters (Hawley's 1987, p. 426; ACGIH 1986, p. 58). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Biphenyl is an irritant of the eyes and mucous membranes in both animals and humans; at high concentrations, this substance causes peripheral and central nervous system damage, liver and kidney damage, and bronchopulmonary lesions. The oral LD_{50} in the rat is 3280 mg/kg (RTECS 1991). Rats who have inhaled diatomaceous earth impregnated with biphenyl at a 300-mg/m^3 concentration for 7 hours/day for 64 days showed signs of irritation of the nasal mucosa and labored breathing; bronchopulmonary lesions and mild toxic effects on the liver and kidneys were seen at autopsy. Rabbits were unaffected by this exposure, but mice exposed at only 5 mg/m^3 for this period of time showed signs of respiratory difficulty (Deichmann, Kitzmiller, Dievker et al. 1947). Among 33 workers in one plant who had had prolonged exposure to biphenyl at concentrations as high as 123 mg/m^3, the most commonly described symptoms were headache, numbness and aching of the limbs, general fatigue, and gastrointestinal symptoms (pain, nausea, and indigestion). Examinations of 22 of these workers for neurophysiological changes found that 19 had alterations representative of central and/or peripheral nervous system damage. In one fatal case in this plant, exposure was estimated to have been high (not further specified) for 11 years. Autopsy showed widespread liver necrosis with some cirrhotic areas, nephrotic changes, heart muscle degeneration, and edematous brain tissue (Hakkinen, Hernberg, Karli, and Vikkula 1973).

Based on this evidence, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing the material health impairments potentially associated with exposure to biphenyl; these include central and peripheral nervous system injury, irritation of the eyes and mucous membranes, bronchopulmonary lesions, and liver and kidney damage. The Agency believes that establishing an 8-hour TWA PEL of 0.2 ppm will substantially reduce these significant occupational risks in agricultural establishments. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

n-BUTYL ALCOHOL

CAS: 71–36–3; Chemical Formula: $CH_3CH_2CH_2CH_2OH$

H.S. No. 1051

In construction, shipyards, marine terminals, and longshoring operations, OSHA's current PEL for n-butyl alcohol is a 100-ppm 8-hour TWA; the ACGIH TLV® is a 50-ppm ceiling, with a skin notation. OSHA has no PEL for this substance in agriculture. NIOSH has no REL for n-butyl alcohol but concurs (Ex. 8–47, Table N1) that the 50-ppm ceiling PEL and skin notation OSHA is proposing for this substance in construction, maritime, and agriculture are appropriate. These were the limits recently established for n-butyl alcohol in general industry.

n-Butyl alcohol is a colorless, highly refractive liquid with a mild wine-like odor. n-Butyl alcohol is widely used as a solvent in shellacs, varnishes, resins, waxes, oils, dyes, and surface coatings, and as a chemical intermediate. It is also used in veterinary medicine as a bactericide (HSDB 1985). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

n-Butyl alcohol is a moderate irritant of the eyes, nose, and throat in humans and animals and has caused hearing loss in experimental animals and exposed workers. The oral LD_{50} in rats is 790 mg/kg, and the LC_{50} in the same species is 8000 ppm for 4 hours (RTECS 1990). The dermal LD_{50} in rabbits is 3400 mg/kg (RTECS 1990). In contact with rabbit eyes, n-butyl alcohol causes moderate but reversible injury (graded 7 on an ascending severity scale of 1 to 10) (Grant 1986, p. 162). Applied to the skin of rabbits, n-butyl alcohol caused mild-to-moderate irritation (RTECS 1990).

In humans, however, n-butyl alcohol is suspected of causing vacuolar keratopathy, i.e., many transparent vacuoles in the epithelial layers of the

cornea (Cogan and Grant 1945, 1948; Jaeger 1953; Kruger 1932). Exposure to butyl alcohol-containing solvents is believed to have caused this effect, which can, in severe cases, result in lacrimation and pain, especially on first arising in the morning (Cogan and Grant 1945). Contact dermatitis of the hands and fingers of exposed workers has also been reported (Clayton and Clayton 1981, p. 4577). Systemic effects in the form of vestibular and auditory nerve injuries have occurred in n-butyl-alcohol-exposed workers in France and Mexico (Seitz 1972; Velasquez 1964; Velasquez, Escobar, and Almaraz 1969/Ex. 1–1174). Based on data describing the rate of n-butyl alcohol uptake through the skin of dogs, DiVincenzo and Hamilton (1979) suggested that direct contact of unprotected human hands with liquid n-butyl alcohol for one hour would result in an absorbed dose that is four times that resulting from the inhalation of 50 ppm for one hour.

The current OSHA 8-hour TWA limit of 100 ppm in construction and maritime is based on the studies of Tabershaw, Fahy, and Skinner (1944) and of Smyth (1956/Ex. 1–759). These studies indicated that workers experienced no narcotic or systemic effects at n-butyl alcohol concentrations below 100 ppm. However, eye, nose, and upper respiratory tract irritation did occur in humans exposed to 24 ppm; this irritation became uncomfortable and was followed by headaches when the concentration reached 50 ppm (Nelson, Enge, Ross et al. 1943/Ex. 1–66; RTECS 1990).

Other data reported by Seitz (1972), Velasquez (1964), and Velasquez, Escobar, and Almaraz (1969/Ex. 1–1174) indicate serious exposure-related hearing loss (hypoacusia) and long-term systemic effects on the auditory nerves of exposed workers; the magnitude of the hearing loss was related to length of exposure to n-butyl alcohol. Nine of 11 workers exposed without hearing protection to an n-butyl alcohol concentration of 80 ppm for periods of from 3 to 11 years displayed impaired hearing, and this phenomenon was particularly evident in younger workers (Velasquez 1964; Velasquez, Escobar, and Almaraz 1969/Ex. 1–1174).

A commenter in the prior Air Contaminants rulemaking (Tr. p. 3–237) questioned whether n-butyl alcohol affects the hearing of exposed individuals. In response to this comment, OSHA noted many studies have shown a relationship between exposure to n-butyl alcohol and damage the auditory nerve (54 FR 2408).

Based on this evidence, OSHA preliminarily finds that the current 8-hour TWA PEL of 100 ppm is not sufficiently protective against the acute effects associated with exposure to n-butyl alcohol; in addition, the possibility that auditory nerve damage may be caused by exposure to concentrations of this substance below the 100-ppm level clearly suggests that the current PEL is inadequate. A skin notation is necessary because data in animals (DiVincenzo and Hamilton 1979) suggest that percutaneous absorption may contribute more to overall exposure than inhalation. OSHA is therefore proposing to establish a permissible exposure limit of 50 ppm as a ceiling, with a skin notation, for n-butyl alcohol. OSHA preliminarily concludes that this limit will protect workers in construction, maritime, and agriculture against the significant risks of potential vestibular and auditory nerve injury as well as of headaches, irritation, and dermatitis, which constitute material impairments of health and are associated with exposure to this substance in the workplace. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

CHLORINATED CAMPHENE (60 Percent)
CAS: 8001–35–2; Chemical Formula: $C_{10}H_{10}C_{18}$
H.S. No. 1078

In construction, shipyards, marine terminals, and longshoring operations, OSHA currently has an 8-hour TWA limit of 0.5 mg/m³, with a skin notation, for chlorinated camphene. The Agency has no PEL for this substance in agriculture. The ACGIH has a TLV®–TWA limit of 0.5 mg/m³ and a TLV®–STEL of 1 mg/m³ for chlorinated camphene (60 percent), with a skin notation; NIOSH has no REL for this substance. OSHA is proposing to add a 1-mg/m³ STEL to the 0.5-mg/m³ 8-hour TWA PEL for chlorinated camphene in construction and maritime, to retain the skin notation, and to extend these limits and the skin notation to workplaces in agriculture.

Chlorinated camphene (also called toxaphene) is a waxy amber solid with a pleasant, pine-like odor. This substance was formerly used as an insecticide on cotton and food crops and in agriculture as a sheep and cattle dip against scabies (HSDB 1987). In 1982, all major uses of this substance were banned by the EPA, and current use is consequently believed to be very low (EPA 1987).

Chlorinated camphene is a convulsant, a liver and kidney toxin, and a carcinogen in experimental animals (EPA 1987). The oral LD_{50} in rats is 55 mg/kg, and the dermal LD_{50} in rabbits is 1025 mg/kg (RTECS 1990). Before death, acutely poisoned animals show varied central nervous system effects, including nausea, muscle spasms, confusion, and convulsions (Hayes 1963/Ex. 1–982). Monkeys tolerate daily feeding at 10 ppm but show toxic symptoms after two weeks' feeding at the 60-ppm level (Sosnierz, Szczurek, Knapek, and Kolodziejczyk 1972/Ex. 1–760). Dogs chronically exposed to this substance by gavage convulsed and subsequently died of respiratory failure. At autopsy, kidney damage was seen (EPA 1987). Rats exposed to chlorinated camphene in lifetime feeding studies showed increases in liver weight and, at autopsy, fatty degeneration and/or necrosis of the liver (EPA 1987). In mice fed average doses of 98 or 198 ppm chlorinated camphene in the diet for 80 weeks, there was a dose-related and statistically significant increase in hepatocellular carcinomas (IARC 1979, Vol. 20, p. 339; EPA 1987). In rats fed average doses of 556 or 1112 ppm (males) or 540 or 1080 ppm (females) for 80 weeks, there was a statistically significant increase in thyroid follicular cell adenomas and carcinomas in the high-dose group in both sexes (EPA 1987; IARC 1979, Vol. 20, pp. 335–336). Based on this evidence, both NIOSH (Ex. 8–47, Table N6B) and the International Agency for Research on Cancer (IARC) have concluded that chlorinated camphene is a potential human carcinogen (IARC 1979, Vol. 20, p. 339). (As discussed earlier in this preamble, OSHA has placed this substance into the neurotoxin category based on the ACGIH's rationale for the TLV® for this substance; placement in a category does not mean that a substance does not also have other health effects.)

In humans, the acute lethal dose of chlorinated camphene by ingestion has been estimated at 60 mg/kg (EPA 1987), and a dose of 10 mg/kg has caused nonfatal convulsions in some exposed individuals. The ACGIH (1986/Ex. 1–3, p. 115) concludes that the acute toxicity of chlorinated camphene is equivalent to that of chlordane, for which the fatal human dose is estimated to be around 6 grams; the ACGIH TLV®–TWA for chlordane is 0.5 mg/m³. One study of 25 human volunteers failed to reveal toxic responses to daily 30-minute exposures to a 500 mg/m³ concentration of chlorinated camphene for 10 consecutive days, followed by similar exposures for three consecutive days three weeks later (ACGIH 1986, Ex. 1–3, p. 115). Two cases of acute aplastic anemia have been reported in workers dermally exposed to chlorinated camphene and lindane, and

one of these workers subsequently died (EPA 1987, p. 4).

OSHA is proposing to add a 15-minute STEL of 1.0 mg/m³ to the current 8-hour TWA PEL of 0.5 mg/m³ and the existing skin notation for chlorinated camphene, and to extend these limits to agriculture. OSHA preliminarily concludes that both a TWA limit and a STEL are required to protect exposed workers in construction, maritime, and agriculture against the significant risks of convulsions and other systemic effects associated with exposure to this substance. OSHA believes that these adverse health effects constitute material impairments of health and that the proposed TWA PEL and STEL will ensure that worker exposures are maintained under good industrial hygiene control. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

DECABORANE

CAS: 17702–41–9; Chemical Formula: $B_{10}H_{14}$

H.S. No. 1114

In construction and maritime, OSHA's current limit for decaborane is 0.05 ppm as an 8-hour TWA, with a skin notation. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 0.05 ppm and a TLV®–STEL of 0.15 ppm, also with a skin notation. NIOSH has no REL for decaborane but concurs (Ex. 8–47, Table N1) that the limits OSHA is proposing are appropriate. OSHA is proposing to add a 15-minute STEL of 0.15 ppm to the 8-hour TWA PEL of 0.05 ppm for decaborane, and to retain the skin notation, in the construction and maritime industries and to extend these limits to agriculture. These are the limits recently established for decaborane in general industry.

Decaborane forms colorless crystals that are stable at ordinary temperatures; this substance has a pungent odor. Decaborane is used as a rocket propellant and as a catalyst in olefin polymerization. It is also used to coat metals to impart corrosion resistance, in mothproofing and dye-stripping, and as a reducing and fluxing agent.

The acute toxicity of decaborane is extremely high for laboratory animals. The 4-hour LC_{50}s for rats and mice are 46 and 12 ppm, respectively (Schechter 1958/Ex. 1–963). Both acute and chronic exposure of several species of laboratory animals has consistently resulted in neurologic signs, including restlessness, depressed breathing, incoordination, weakness, and convulsions (Roush 1959). The ability of decaborane to penetrate the skin is particularly notable: the dermal LD_{50}s for rabbits and rats are 71 and 740 mg/kg, respectively (Svirbely 1954a/Ex. 1–385).

Neurotoxic signs and symptoms occurring among exposed humans have also been reported; these have included headache, lethargy, nausea, dizziness, convulsions, and muscle tremors (Lowe and Freeman 1957). Decaborane also produces marked irritation of the skin and mucous membranes; contact with the eyes can result in serious keratoconjunctivitis, with ulceration and necrotic changes (HSDB 1985).

OSHA is retaining its 8-hour TWA PEL of 0.05 ppm and the skin notation for decaborane in construction and maritime, and is proposing to add a 15-minute STEL of 0.15 ppm for this substance in these two sectors; OSHA also is proposing to extend the 8-hour TWA, the 15-minute STEL, and the skin notation to agriculture. The Agency preliminarily concludes that these limits will provide protection against the significant risks of material health impairment in the form of neuropathy and irritation. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

DIBORANE

CAS: 19287–45–7; Chemical Formula: B_2H_6

H.S. No. 2054

In general industry, construction, and maritime, OSHA's current permissible exposure limit for diborane is 0.1 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.1 ppm for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing to establish an 8-hour TWA PEL of 0.1 ppm for diborane in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Diborane is a colorless, flammable gas with a repulsive, sweet odor. Diborane is used in the synthesis of boron compounds and metal borohydrides, as a polymerization catalyst for ethylene, as a fuel for engines and rockets, as a reducing agent, and as a doping agent (NIOSH/OSHA Occupational Health Guideline 1981, p. 1; Hawley's 1987, p. 367).

Diborane causes pulmonary irritation and may also affect the nervous system in humans and animals. In rats and mice, LC_{50} values are 40 ppm and 29 ppm for 4 hours, respectively (RTECS 1990). The lowest lethal concentration in hamsters is 50 ppm for 8 hours; in dogs, this value is 125 ppm for 2 hours (RTECS 1990). Acutely poisoned animals died of pulmonary edema (Merritt 1965). Dogs repeatedly exposed to 5 ppm diborane for 6 hours/day died after 10 to 20 such exposures, but one of two dogs survived repeated exposures to concentrations of 1 to 2 ppm for 6 months (Comstock et al. 1954). Repeated exposure to an unspecified concentration of diborane caused spinal cord lesions in a dog (Jacobson, Murtha, Weir et al. 1960).

The inhalation dose that is lethal to humans is 159 ppm for 15 minutes (Braker and Mossman 1980, p. 219). Acute diborane poisoning via inhalation causes signs and symptoms similar to those of metal fume fever, including tightness, heaviness, and burning in the chest, coughing, shortness of breath, pericardial pain, nausea, and drowsiness. These symptoms may not onset for 24 hours after exposure and may persist for 1 to 3 days or more (Parmeggiani 1983, p. 318). Exposure to high concentrations (not further specified) of diborane causes eye irritation (Proctor, Hughes, and Fischman 1988, p. 181). Prolonged exposure to low levels of diborane causes headache, lightheadedness, vertigo, chills, and fever; fatigue or weakness may also occur and persist for several hours (Cordasco, Cooper, Murphy, and Anderson 1962; Lowe and Freeman 1957). Recurrent diborane exposure caused chronic respiratory distress in two patients (Cordasco et al. 1962).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to diborane causes eye irritation, respiratory injury, and central nervous system effects. The Agency believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse exposure-related effects. OSHA believes that establishing an 8-hour TWA PEL of 0.1 ppm for diborane in agriculture is necessary to substantially reduce the risks of these material health impairments. In addition, promulgation of this limit for diborane will make OSHA's PEL for this substance consistent across all OSHA-regulated sectors.

Di-sec-OCTYL PHTHALATE

CAS: 117–81–7; Chemical Formula: $C_{24}H_{38}O_4$

H.S. No. 1116

OSHA's current limit for di-sec-octyl phthalate in the construction and maritime industries is 5 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 5 mg/m³ and a TLV®–STEL of 10 mg/m³, and these are the limits OSHA is proposing for the construction, agriculture, and maritime industries.

NIOSH (Ex. 8–47, Table N6A) concurred with these limits.

Di-sec-octyl phthalate, also called di-2-ethylhexyl phthalate or DEHP, is a light-colored, viscous, odorless, combustible liquid that is used as a plasticizer for resins and elastomers (ACGIH 1986, p. 86/Ex. 1–3, p. 223). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

DEHP is not acutely toxic to small laboratory animals via the oral route of administration. The oral LD_{50} reported for mice is 26.3 g/kg; for rats, it is 33.8 g/kg (Krauskopf et al. 1973/Ex. 1–495). No skin irritation or sensitization potential has been demonstrated in either animals or humans, and the lethal dermal dose in rabbits is about 25 ml/kg (Singh, Lawrence, and Autian 1972/Ex. 1–436). Shaffer, Carpenter, and Smyth (1945/Ex. 1–369) and Lawrence (unpublished data, as cited in ACGIH 1986/Ex. 1–3, p. 223) have reported deaths in rats and chronic diffuse inflammation of the lung in mice exposed to DEHP at unspecified levels.

Long-term dietary toxicity studies in rats, guinea pigs, and dogs have established a no-effect dose level for DEHP of about 60 mg/kg/day, and no carcinogenic or histologic abnormalities were observed at this level (Gesler 1973/Ex. 1–481). Higher doses were associated with growth retardation and increased liver and kidney weights but not with histologic abnormalities. Metabolic studies have demonstrated that laboratory animals do not appreciably metabolize DEHP (Dillingham and Autian 1973/Ex. 1–477).

Di-sec-octyl phthalate has been shown to be carcinogenic to rats and mice fed higher doses; this evidence comes from a National Toxicology Program bioassay in which male and female Fischer 344 rats were fed 0, 6, or 12 g DEHP per kg diet and B6C3F1 mice were fed 0, 3, or 6 g DEHP per kg diet for 103 weeks (IARC 1982, Vol. 29, p. 269, corr. 42:261). There was a statistically significant and dose-related increase in the incidence of hepatocellular carcinomas in both sexes of both species of animals tested. Based on this evidence, IARC (1982) concluded that there was sufficient evidence of DEHP's carcinogenicity in animals, and NIOSH (Ex. 8–47, Table N6A) concurs with this assessment.

Several animal studies have reported fetotoxic, teratogenic, and reproductive effects associated with exposure to di-sec-octyl phthalate. Fetal resorptions, fetal deaths, and gross abnormalities were reported following treatment of pregnant rats with 5 or 10 g/kg DEHP by intraperitoneal injection on days 5, 10, and 15 of gestation (IARC, 1982, Vol. 29, p. 269). Administration of 0.05 to 1 percent DEHP in the diet of mice throughout gestation resulted in an increased frequency of fetal resorptions and fetal malformations, including neural tube defects and delayed ossification (Shiota and Nishimura 1982). Doses of 2 or 4 ml/kg administered intraperitoneally to pregnant rats on days 3, 6, and 9 of gestation prevented implantation and caused excessive bleeding, incomplete expulsion of fetuses, and maternal deaths (Peters and Cook 1973).

A study of workers exposed to a mixture of the vapors of diethyl phthalate, dibutyl phthalate, and di-2-ethylhexyl phthalate reported that exposures to 1 to 6 ppm caused no peripheral polyneuritis (Raleigh, as cited in ACGIH 1986/Ex. 1–3, p. 223). However, Russian investigators examined male and female workers exposed to between 1.7 and 66 mg/m³ of various combinations of airborne phthalates (including butyl phthalate, higher aryl phthalates, dioctyl phthalate and others) and noted complaints of pain, numbness, and spasms in the upper and lower extremities after six to seven years of exposure. Polyneuritis was observed in 32 percent of the workers studied, and 78 percent of these workers showed depression of vestibular receptors (Milkov, Aldyreva, Popova et al. 1973/Ex. 1–646).

OSHA is retaining the 8-hour TWA PEL of 5 mg/m³ in construction and maritime, proposing to add a 15-minute STEL of 10 mg/m³ for di-sec-octyl phthalate in these two industries, and proposing to extend both limits to the agricultural sector. The Agency preliminarily concludes that these limits together will protect workers from the significant risks of neuropathic and other systemic injuries that constitute material health impairments that are associated with exposure to this substance. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

DICHLOROACETYLENE

CAS: 7572–29–4; Chemical Formula:

ClC≡CCl

H.S. No. 1123

OSHA currently has no limit for dichloroacetylene in the agriculture, construction, or maritime industries. The ACGIH has a TLV®-ceiling of 0.1 ppm. NIOSH has no REL but concurs (Ex. 8–47, Table N6A) with the limit being proposed. OSHA is proposing a ceiling limit of 0.1 ppm for dichloroacetylene in construction, maritime, and agriculture. This is the limit recently established for dichloroacetylene in general industry.

Dichloroacetylene is not produced commercially but is formed from the thermal decomposition of trichloroethylene. This substance is a spontaneously combustible liquid.

In preliminary inhalation exposure studies, guinea pigs demonstrated a 4-hour LC_{50} of 20 ppm; death occurred in these animals 2 or 3 days after exposure and was caused by pulmonary edema. In rats, similar exposures to dichloroacetylene in the presence of a 330-ppm concentration of trichloroethylene resulted in an LC_{50} of 55 ppm (Siegel, Jones, Coon, and Lyon 1967). The LC_{50}s for 1- and 4-hour exposures in mice were 124 and 19 ppm, respectively; deaths were caused by kidney injury and, at autopsy, degenerative lesions were found in the brain (Reichert, Ewald, and Henschler 1975). Rats exposed for 6 hours/day, 5 days/week, for 6 weeks to a dichloroacetylene-trichloroethylene mixture exhibited weakness in the hind limbs, respiratory distress, self-inflicted bite wounds, and kidney injury; the concentrations of dichloroacetylene employed in this study were 2, 9, 9.8, and 15.5 ppm (Reichert, Henschler, and Bannasch 1978).

In humans, dichloroacetylene exposure causes headache, loss of appetite, extreme nausea, and vomiting; exposure affects the trigeminal nerve and facial muscles and exacerbates facial herpes. Disabling nausea was experienced by approximately 85 percent of individuals exposed for prolonged periods of time (not further specified) at concentrations ranging from 0.5 to 1 ppm (Saunders 1967/Ex. 1–361). Several occupational fatalities have been attributed to exposure to dichloroacetylene (Humphrey and McClelland 1944/Ex. 1–491; Firth and Stuckey 1945, as cited in ACGIH 1986/Ex. 1–3, p. 177). Humphrey and McClelland (1944/Ex. 1–491) reported 13 cases of cranial nerve palsy, nine of which had labial herpes, following exposure to dichloroacetylene. These patients also had symptoms of nausea, headache, jaw pain, and vomiting. Autopsies of two of these victims revealed edema at the base of the brain (Humphrey and McClelland 1944/Ex. 1–491).

In the prior air contaminants rulemaking, NIOSH indicated that, in its opinion, this substance should be designated as a potential occupational carcinogen (Ex. 8–47, Table N6A) based on findings of an increased incidence of kidney adenocarcinomas in male mice and an increased incidence of

lymphomas and benign liver and kidney tumors in rats administered dichloroacetylene by inhalation. The International Agency for Research on Cancer has concluded that there is limited evidence for the carcinogenicity of dichloroacetylene in animals (IARC 1986, Vol. 39, pp. 369–378). As discussed elsewhere in this preamble, OSHA notes that placement of this substance in the neurotoxin category for the purposes of this rulemaking in no way obviates these findings of dichloroacetylene's carcinogenicity in animals; placement in this category is intended for ease of classification only and simply reflects the rationale underlying the ACGIH limit for this substance.

Based on the neurotoxic effects described above, OSHA is proposing a ceiling limit of 0.1 ppm for dichloroacetylene in the agriculture, construction, and maritime industries. The Agency preliminarily concludes that this limit will substantially reduce the significant risks of disabling nausea and serious systemic effects posed to workers exposed to dichloroacetylene in these sectors at the levels currently permitted by the absence of an OSHA limit. OSHA finds that these health effects constitute material impairments of health. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

DIPROPYLENE GLYCOL METHYL ETHER

CAS: 34590–94–8; Chemical Formula: $CH_3OC_3H_6OC_3H_6OH$

H.S. No. 1149

For the construction and maritime industries, OSHA currently has an 8-hour TWA limit of 100 ppm for dipropylene glycol methyl ether (DPGME), with a skin notation. The ACGIH recommends a TLV®–TWA of 100 ppm and a TLV®–STEL of 150 ppm, with a skin notation. In construction and maritime, OSHA is retaining the 8-hour TWA permissible exposure limit of 100 ppm and the skin notation and is proposing to add a 150-ppm STEL for dipropylene glycol methyl ether; the Agency is also proposing to extend these limits to agriculture. NIOSH has no REL but concurred (Ex. 8–47, Table N1) that these limits are appropriate. These are the limits recently established in general industry.

DPGME is a colorless liquid with a mild, pleasant odor. It is used as a solvent in hard-surface liquid household cleaners and water-based surface coatings and is also a component of hydraulic brake fluid. DPGME is also used in the manufacture of various cosmetics (HSDB 1985).

DPGME is an irritant and central nervous system depressant in humans and animals. The oral LD_{50} in rats is 5.2 g/kg (Rowe et al. 1954/Ex. 1–435). Dogs receiving intravenous injections of DPGME exhibited central nervous system depression and died as a result of respiratory failure (Shideman and Procita 1951/Ex. 1–667). Four animal species, including the monkey, were exposed repeatedly to seven-hour daily inhalation exposures of between 300 and 400 ppm DPGME; the animals exhibited narcosis and, at autopsy, histological changes in the lung and liver (Rowe, McCollister, Spencer et al. 1954/Ex. 1–435).

Humans inhaling DPGME concentrations of 300 to 400 ppm judged this level to be very irritating and disagreeable, but 100 ppm was tolerable and, in the opinion of the authors, was unlikely to produce organic injury (Rowe, McCollister, Spencer et al. 1954/Ex. 1–435). Patch tests on the skin of 250 human subjects produced neither irritation nor sensitization (ACGIH 1986/Ex. 1–3, p. 221). Humans exposed to DPGME vapor concentrations at levels between 50 to 2000 ppm experienced eye, nose, and throat irritation before the onset of CNS impairment, which occurred at 1000 ppm in one of two subjects (Stewart, Baretta, Dodd, and Torkelson 1970/Ex. 1–379).

During the general industry rulemaking, NIOSH (Ex. 150) reported that it is developing a criteria document on the glycol ethers and submitted recent toxicity data on DPGME, including the following: rats and mice inhaling concentrations of 50, 140, or 330 ppm DPGME six hours/day for nine days showed increased liver weights (at 50 and 140 ppm for the rat and at 330 ppm for the mouse), but no effects were observed when rats inhaled 15, 50, or 200 ppm DPGME 6 hours/day, 5 days/week for 13 weeks (Landry and Yano 1984). A 1985 study also indicates that DPGME is metabolized via the same routes and to the same types of metabolites—propylene glycol, and sulfate and glucuronide conjugates of DPGME—as those previously identified for PGME (1-methoxy-2-propanol) (Miller, Hermann, Calhoun et al. 1985). The Landry and Yano study (1984) further indicated that, at the concentrations tested, DPGME exerted no teratogenic or reproductive effects. Even at the highest levels tested (19 g/kg), no single application of DPGME to the skin of rabbits was lethal, although some narcosis and transient weight loss did occur (Rowe et al. 1954/Ex. 1–435). However, a significant number of deaths occurred in a group of rabbits tested with 65 repeated dermal applications of 5 ml/kg (4.8 g/kg) or higher during a 90-day period (Rowe et al. 1954/Ex. 1–435).

In construction and maritime, OSHA is retaining its 8-hour TWA PEL of 100 ppm and the skin notation and is proposing to add a STEL of 150 ppm for dipropylene glycol methyl ether; in addition, OSHA is proposing to extend these limits to agriculture. The Agency preliminarily concludes that these limits will substantially reduce the significant risks of central nervous system effects and irritation, which constitute material health impairments, that exist when workers in construction, maritime, or agriculture are exposed to DPGME even for short periods. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

n-HEXANE

CAS: 110–54–3; Chemical Formula: $CH_3(CH_2)_4CH_3$

H.S. No. 1200

OSHA's current 8-hour PEL for n-hexane (normal hexane) is 500 ppm for the construction and maritime industries; there is no limit in agriculture. The ACGIH has a 50-ppm TLV®–TWA for this substance, and the NIOSH RELs are 100 ppm as a 10-hour TWA and 510 ppm as a 15-minute ceiling. OSHA is proposing an 8-hour TWA limit of 50 ppm for n-hexane for the agricultural, construction, and maritime industries; this was the limit recently established in general industry. NIOSH (Ex. 8–47, Table N1) concurs that an 8-hour TWA PEL of 50 ppm is appropriate for n-hexane.

Normal hexane is a clear, volatile liquid. It is used as a solvent for the extraction of soybean and various seed oils; in the manufacture of polyolefins, elastomers, and pharmaceuticals; and as a denaturant for alcohol.

The acute lethality of n-hexane is low; the lowest reported lethal concentration for mice is 33,330 ppm (Clayton and Clayton 198, p. 3186). However, it has been well established that chronic exposure to n-hexane produces distal axonopathy in both experimental animals and humans; it is metabolized to 2,5-hexanedione (2,5–HD), which is thought to be the causative agent of most of the adverse neurological effects observed after exposure to hexane (Schaumburg, Spencer, and Thomas 1983/Ex. 1–228).

The ACGIH established a TLV® of 50 ppm for this substance, based primarily on studies (Miyagaki 1967/Ex. 1–198; Inoue, Takeuchi, Takeuchi et al. 1970/Ex. 1–75) showing peripheral neuropathies in workers exposed to levels as low as 210 ppm. NIOSH based

its 100-ppm REL on the same studies as those cited by the ACGIH (Miyagaki 1967/Ex. 1–198; Inoue, Takeuchi, Takeuchi et al. 1970/Ex. 1–75). In the rulemaking for general industry, NIOSH reasoned as follows:

> The absence of definitive epidemiologic or toxicologic evidence makes it difficult to determine how much lower the environmental limit should be. Professional judgment suggests [that] a TWA concentration of 350 mg/m³ (100 ppm) offers a sufficient margin of safety to protect against the development of chronic nerve disorders in workers (NIOSH 1977a/Ex. 1–233, p. 74).

The adverse neurological effects of hexane exposure are manifested as both sensory and motor dysfunction. Initially, there is a symmetric sensory numbness of the hands and feet, with loss of pain, touch, and heat sensation. Motor weakness of the toes and fingers is often present; as the neuropathy becomes more severe, weakness of the muscles of the arms and legs may also be observed (Schaumburg, Spencer, and Thomas 1983/Ex. 1–228). Clinical findings may include footdrop, muscle atrophy, and paresthesias (tingling) in the arms and legs. Biopsies of peripheral nerves show swelling of the nerve and thinning of the myelin sheath (Proctor, Hughes, and Fischman 1988, p. 271). There are no known conditions that predispose an individual to hexane neurotoxicity (Schaumburg, Spencer, and Thomas 1983/Ex. 1–228). The onset of neurological symptoms may not be evident for several months to a year after the beginning of exposure. Recovery may be complete but may require 1 to 2 years, and severely exposed individuals often retain some degree of sensorimotor deficit (Proctor, Hughes, and Fischman 1988, p. 271).

The dose-response relationship for n-hexane exposure in humans is not well defined, although it is clear that the severity of the resulting neuropathy increases as the exposure level of n-hexane increases. A number of studies have shown a consistent relationship between exposure levels of 500 ppm (OSHA's current exposure limit in construction and maritime) to 2000 ppm and the development of characteristic peripheral neuropathies (Yamamura 1969, as cited in ACGIH 1986/Ex. 1–3, p. 305; Yamada 1967/Ex. 1–192). Neuropathic effects have also been shown to occur at levels between 210 and 500 ppm (Takeuchi, Maluchi, and Takagi 1975/Ex. 1–217).

Two recent studies (Wang, Chang, Kao et al. 1986; Iida 1982) suggest that an exposure limit of 100 ppm would not sufficiently protect against the neuropathic effects of n-hexane exposure. Iida (1982) found 21 cases of mild neuropathy among workers in a sandal factory where ventilation systems were installed to achieve a 100-ppm concentration of n-hexane. In a cross-sectional study, Wang et al. (1986) found that workers in a press-proofing factory showed a significant decrease in motor nerve conduction velocities, despite the fact that n-hexane exposures were being maintained below 100 ppm.

Chronic exposure studies of n-hexane conducted in animals show effects consistent with those reported in human studies. Peripheral neuropathy has been demonstrated in rats exposed to 400 to 600 ppm n-hexane for 45 days and in mice exposed to 250 ppm for over 7 months. Degeneration of myelin and axis cylinders have been found in rats exposed to 850 ppm for 143 days (Kurita 1974; Ashbury, Nieben, and Telfer 1974; Gonzales and Downey 1972).

Reports of effects occurring at levels ranging from below 100 to 500 ppm indicate that the current OSHA PEL of 500 ppm is not adequate to protect exposed workers in construction and maritime from adverse sensorimotor neuropathic effects, and exposure at this level thus represents a significant risk to workers. The decreased sensitivity to pain, touch, and temperature associated with n-hexane exposure can also make a worker more susceptible to injuries and accidents. Further, the delayed onset of a clinical response, which is typical of hexane exposure, increases the probability that exposure will continue until prolonged or irreversible effects occur.

Both the presence of peripheral neuropathies in workers exposed below 100 ppm and the delay in the onset of neurological symptoms indicate that workers exposed at levels above the proposed 50 ppm limit are at significant risk of developing these symptoms. OSHA therefore proposes to revise the PEL for n-hexane to 50 ppm as an 8-hour TWA for workplaces in construction and maritime and to extend this PEL to agriculture. The Agency preliminarily concludes that this PEL will substantially reduce the significant risk of peripheral neuropathies and other adverse neuropathic effects that constitute material impairments of health and are associated with the exposures to levels above the proposed limit. In addition, promulgation of these limits will make OSHA's PEL for this substance consistent across all regulated sectors.

2–HEXANONE (METHYL n-BUTYL KETONE)

CAS: 591–78–6; Chemical Formula: $CH_3CO—CH_2CH_2CH_2CH_3$

H.S. No. 1202

In construction, marine terminals, shipyards, and longshoring operations, OSHA's current PEL for 2-hexanone is 100 ppm as an 8-hour TWA. The NIOSH REL is a 1 ppm (10-hour) TWA, and the ACGIH recommends a TLV®–TWA of 5 ppm. In OSHA's recent Air Contaminants rulemaking for general industry, the final rule established a permissible exposure limit for this substance of 5 ppm as an 8-hour TWA; this is the limit being proposed for 2-hexanone in construction, maritime, and agriculture.

2–Hexanone (also called methyl butyl ketone) is a colorless, volatile liquid with a characteristic acetone-like odor that is more pungent than that of acetone. 2–Hexanone is used as a solvent for resins, oils, fats, waxes, and lacquers, and in lacquer and varnish removers (HSDB 1984).

2–Hexanone is an irritant and a neurotoxin both in humans and in animals. The oral LD_{50} in rats is 2590 mg/kg, and the LC_{50} in the same species is 8000 ppm for 4 hours (RTECS 1990). The dermal LD_{50} in rabbits is 4800 mg/kg (RTECS 1990). Animals exposed continuously to 100- or 600-ppm concentrations of 2-hexanone developed neuropathic signs within 4 to 8 weeks of the beginning of exposure; some animals showed axonal swelling and thinning of the myelin sheath at autopsy (Mendell et al. 1974). A metabolite of 2-hexanone, 2,5-hexanedione, appears to be responsible for the neural damage; this same metabolite is formed when n-hexane (discussed above) is metabolized. Animals of several species, including rats, cats, dogs, monkeys, hens, and guinea pigs, have developed 2-hexanone-related peripheral neuropathies (O'Donoghue 1985). Rats intermittently exposed over a period of 4 months to a 2-hexanone concentration of 1300 ppm displayed severe hind-limb weakness.

Vapor exposures of 1000 ppm 2-hexanone caused moderate eye and nose irritation in human volunteers in a few minutes (Clayton and Clayton 1982, pp. 4741–4747). In addition to the textile printing plant episode described below, three cases of peripheral neuropathy have been seen in a group of 26 spray painters involved in construction; 2-hexanone is believed to have been responsible for these cases (Rom 1983, pp. 524–525). Another group of workers exposed for 5 weeks or longer to 9- to 36-ppm concentrations of this substance developed distal polyneuropathy, complete with motor and sensory decrements (Gosselin, Smith, and Hodge 1984, p. II–185).

The NIOSH REL for 2-hexanone of 1 ppm (as a 10-hour TWA) is based on an epidemiologic study describing an outbreak of neurologic disease among workers in a plant that manufactures printed fabrics (Allen, Mendall, Billmaier et al. 1975/Ex. 1–80). This study reported that a screening of 1,157 exposed workers revealed 86 verified cases of distal neuropathy. 2-Hexanone was suspected of being the neurotoxicant because it had only recently been introduced into the process (Allen, Mendall, Billmaier et al. 1975/Ex. 1–80). When recommending its limit, NIOSH relied on an industrial hygiene survey of the plant conducted by Billmaier, Yee, Allen et al. (Ex. 1–76) in 1974, which showed that 2-hexanone concentrations near the textile printing machines ranged from 1 to 156 ppm (10-minute area samples). After reviewing this evidence, NIOSH concluded that 1 ppm could not be ruled out as an effect level for 2-hexanone-induced neuropathy (NIOSH Criteria Document). OSHA is not persuaded by NIOSH's reasoning in this instance, both because the samples taken in the plant were area rather than personal samples and because workers at the plant reportedly washed their hands in 2-hexanone. OSHA believes that the area samples were likely to have reflected higher concentrations than the workers would have experienced and that the outbreak described in the NIOSH Criteria Document resulted, at least in part, from percutaneous absorption of this substance.

Both human and animal studies show the development of neuropathic disease at exposure concentrations well below the current 100-ppm PEL; OSHA preliminarily concludes that a reduction in the PEL is needed to reduce this significant risk. Accordingly, OSHA is proposing a 5-ppm (8-hour TWA) PEL for 2-hexanone in construction, maritime, and agricultural workplaces. In addition, promulgation of these limits will make OSHA's PEL for this substance consistent across all regulated sectors.

IRON PENTACARBONYL

CAS: 13463–40–6; Chemical Formula: $Fe(CO)_5$

H.S. No. 1216

In construction, shipyards, marine terminals, and longshoring operations, OSHA currently has no exposure limit for iron pentacarbonyl. The ACGIH has a TLV®–TWA (measured as iron) of 0.1 ppm and a TLV®–STEL of 0.2 ppm for iron pentacarbonyl. NIOSH has no REL for iron pentacarbonyl but concurs (Ex. 8–47, Table N1) that these limits are appropriate. The Agency is proposing permissible exposure limits of 0.1 ppm (TWA) and 0.2 ppm (STEL) for iron pentacarbonyl in the construction, maritime, and agriculture sectors; this is the limit recently established in general industry.

Iron pentacarbonyl is used in iron production, as an anti-knock fuel additive, and as a catalyst and reagent in chemical synthesis (HSDB 1989). This substance is an oily liquid that is either colorless or yellow (HSDB 1985).

Iron pentacarbonyl is a lung and central nervous system toxin on acute exposure. The oral LD_{50} in rats is 40 mg/kg, and the LC_{50} in the same species is 44 mg/m³ (no duration specified) (RTECS 1989). The dermal LD_{50} in rabbits is 240 mg/kg (RTECS 1989). In 1970, Gage found that a 5.5-hour exposure to a 33-ppm concentration of iron pentacarbonyl caused fatalities in three of eight rats; four of eight animals died after two 5.5-hour exposures at 18 ppm. At 7 ppm, no ill effects were observed in rats exposed 18 times in 5.5 hours (Gage 1970/Ex. 1–318).

Immediate symptoms of acute exposure to high concentrations of iron pentacarbonyl include headache and dizziness, followed in 12 to 36 hours by fever, cyanosis, cough, and shortness of breath. Another clinical effect of overexposure to this substance is lung injury, and degenerative changes in the central nervous system have also been reported. Iron carbonyl is the least toxic of the metal carbonyls (Clayton and Clayton 1982, p. 1797).

Based on these findings in humans and animals, OSHA is proposing permissible exposure limits of 0.1 ppm (TWA) and 0.2 ppm (STEL) for iron pentacarbonyl. The Agency preliminarily concludes that these limits will protect workers in construction, maritime, and agriculture from the significant risks of material health impairment in the form of exposure-related headache, dizziness, fever, dyspnea, cyanosis, pulmonary injury, and central nervous system effects. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

LINDANE

CAS: 58–89–9; Chemical Formula: $C_6H_6Cl_6$

H.S. No. 2100

The current OSHA PEL for lindane in general industry, construction, and maritime is 0.5 mg/m³ as an 8-hour TWA, with a skin notation. There is no PEL for lindane in agriculture. The ACGIH TLV®–TWA for this substance is 0.5 mg/m³, with a skin notation; NIOSH has no REL for lindane but concurs with the limit being proposed (Ex. 8–47, Table N3A). OSHA is proposing a PEL of 0.5 mg/m³ as an 8-hour TWA, with a skin notation, for lindane in agriculture. Promulgation of this limit will make the PEL for lindane consistent across all OSHA-regulated sectors.

Lindane, also called benzene hydrochloride or BHC, is a colorless to white crystalline solid with a faint musty odor (HSDB 1986). Lindane is used as an insecticide for field crops, ornamentals, timber, soil and seed treatment, stored products, and in household use. It is used in human medicine as a scabicide and pediculicide, most often as a 1-percent lotion, cream, or shampoo. Lindane is also used in veterinary medicine (HSDB 1986; Grant 1986, p. 561). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Lindane is a convulsant in both animals and humans; it is an irritant of the eyes, nose, and upper respiratory tract and has been associated with the development of aplastic or hypoplastic anemia and leukemia in humans. This substance also has carcinogenic, reproductive, and embryotoxic effects in experimental animals. The oral LD_{50} in rats is 76 mg/kg, and the dermal LD_{50} in rabbits is 50 mg/kg (RTECS 1990). Following a single dose of lindane, the convulsion threshold in mice decreases; 2 days after administration, however, the threshold increases (Hulth et al. 1978). Lindane also has reproductive and embryotoxic effects in rats (IARC 1974, Vol 5; Petrescu et al. 1974, in Rev. Med. Chin. Soc. Nat. 1951 78(4):831–842). Rats gavaged daily with 32 mg/kg lindane for 6 months developed nervous symptoms and showed fatty degeneration of the liver and renal tubular epithelium and vacuolization of cerebral cells at autopsy (NRC 1977, p. 587). Minor lesions of the liver have been seen in rats dosed with lindane at 2.6 to 5 mg/kg/day and in mice dosed with 6 to 10 mg/kg/day. Rodents exposed orally to high doses developed degenerative changes in the kidney, pancreas, and testes (Hayes 1982, pp. 211–229). Oral administration of lindane has caused thyroid tumors in rats, and mice developed liver tumors, sometimes with lung metastases, when administered lindane orally (IARC 1987, Suppl. 7, p. 221; IARC 1979, Vol. 20, p. 189). Based on this evidence, the International Agency for Research on Cancer has concluded that the evidence of lindane's carcinogenicity in animals is sufficient (IARC 1987, Suppl. 7, p. 221).

Rats fed 1500 ppm in the diet for 90 days developed testicular atrophy, spermatogenic arrest, and apparent inhibition of androgenesis (Shivanandappa and Krishnakumari 1983). Lindane is mutagenic in mammalian test systems (RTECS 1987).

Acute exposure to lindane vapor at unspecified concentrations causes irritation of the eyes, nose, and upper respiratory tract in humans (AIHA 1972). Accidental ingestion of lindane has caused repeated, violent, and fatal convulsions in humans; respiratory difficulty and cyanosis were also seen in these cases. Nonlethal ingestions have been associated with convulsions, tremor, fever, cyanosis, loss of consciousness, dizziness, and nausea (Hayes 1963; Hayes 1982, p. 222). Neurological studies of 37 workers exposed to lindane for 2 years showed that these workers had serious electroencephalographic disturbances and that 14 workers had minor neurological symptoms (Czegledi-Janko and Avar 1970). Four cases of leukemia have been reported in men exposed to lindane with or without other chemical exposures, and agricultural workers exposed to lindane and other pesticides have shown an increase in lung cancer mortality; however, the International Agency for Research on Cancer (IARC) has concluded that the evidence of lindane's carcinogenicity in humans is inadequate (IARC 1987, Suppl. 7, p. 220).

Based on this evidence in humans and animals, OSHA is proposing a 0.5 mg/m³ 8-hour TWA PEL, with a skin notation, to protect workers in agriculture from the significant risk of adverse health effects caused by exposure to lindane. The Agency preliminarily concludes that this limit is necessary to substantially reduce a significant risk of material health impairment in exposed workers. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

MANGANESE compounds
CAS: 7439–96–5; Chemical Formula: Mn
H.S. No. 2103

In general industry, construction, and maritime, OSHA's current limit for manganese and its compounds is 5 mg/m³ (measured as manganese) as a ceiling. There is no limit in agriculture. The ACGIH has a TLV® of 5 mg/m³ as an 8-hour TWA, and NIOSH has a REL of 5 mg/m³ for the metal and its compounds, also as a ceiling. OSHA is proposing a 5-mg/m³ ceiling limit for manganese and its compounds in agriculture; this action will make the PELs for manganese and its compounds consistent across all regulated sectors.

Manganese is a reddish-gray or silvery powder or metal that has no odor. Some of the more important manganese compounds are manganese dioxide, manganese tetroxide, manganous carbonate, manganous chloride, manganous acetate, and potassium permanganate. Manganese is used in steelmaking as a component of ferrous alloys, in other operations to make nonferrous alloys, in the manufacture of dry cell batteries, and in the production of chemicals (Clayton and Clayton 1981, p. 1750).

Manganese is a central nervous system toxin on chronic exposure and causes metal fume fever on acute exposure. The oral LD_{50} in rats is 9 g/kg (RTECS 1990). Instilled into the eyes of rabbits, manganese caused a mild degree of irritation; contact with the skin of rabbits also caused mild irritation (RTECS 1990). Subcutaneous doses of 50 mg/kg manganese chloride caused death in mice, rabbits, and guinea pigs (Cervinka 1929, in Compte Rend. Soc. (France) 102:262). Injected intratracheally into the lungs of rats, both manganese oxide and manganese chloride at doses of 10 mg/kg immediately caused histological changes in the lung tissues; before death, the animals injected with the chloride developed pulmonary edema (Davies and Harding 1949, in Br. J. Ind. Med. 6:82). Rabbits exposed to manganese dust for 4 hours/day for 3 to 6 months at concentrations of 10 to 20 mg/m³ developed fibrotic lung changes but no pneumonitis; a depression in the number of red blood cells and hemoglobin also occurred (Carratala and Carboneschi in ACGIH 1986, p. 354(86)). Monkeys inhaling a manganese aerosol showed agitation, followed by torpor, nervousness, tremor, flexion and extension of the limbs, and cyanosis; these signs disappeared within 3 weeks of the exposure but returned in more severe form 5 months later (van Bogaert and Dallemagne 1945, in Mgsch. Psychiatr. Neurol. 111:60, as cited in Clayton and Clayton 1981, p. 1755).

In humans, chronic exposure to manganese or its compounds causes a form of poisoning known as manganism; the signs and symptoms of this condition strongly resemble those of parkinsonism. Workers with manganism experience languor, sleepiness, limb weakness, and spastic gait and develop a mask-like appearance of the face as well as emotional instability (Fairhall and Neal 1943, in Natl. Inst. Health Bull. No. 182; Flinn, Neal, Reinhart et al. 1940, in U.S. Pub. Hlth. Serv. Bull. No. 247). The onset of manganism occurs between 6 months and 24 years of first exposure to manganese (Clayton and Clayton 1981, p. 1756). Manganese psychosis, characterized by uncontrollable laughter, impulsive behavior, euphoria, and insomnia, also occurs in many cases (Clayton and Clayton 1981, p. 1762). Although disabling, manganism is rarely fatal; in the early stages, the condition is reversible (Rom 1983, p. 494). The exposure concentrations associated with the development of manganism ranged from 6.8 to 42.2 mg/m³ in a group of manganese miners; other evidence suggests that exposures to concentrations of 5 mg/m³ and above are associated with definite signs and symptoms (Emara et al. 1971, in Br. J. Ind. Med. 28:78–82; Tanaka and Lieben 1969/Ex. 1–388).

Acute exposure to manganese dust and fumes causes metal fume fever, with chills, fever, nausea, sweating, and cough (Piscator 1976, in Environ. Res. 11:268–270). Symptoms onset 4 to 24 hours after the acute overexposure and disappear if exposure is not resumed for several days (Piscator 1976). Exposure to manganese dust also causes mild sensory irritation of the eyes, mucous membranes, and skin (Rom 1983, p. 493).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to manganese and its compounds is associated with a significant risk of acute and chronic disease in the form of pneumonitis and manganism, respectively. The Agency considers these adverse health effects material impairments of health and believes that the proposed PEL of 5 mg/m³, as a ceiling, is necessary to reduce a significant risk among workers in the agriculture industry. In addition, promulgation of this limit will make the PEL for manganese and its compounds consistent across all OSHA-regulated sectors.

MANGANESE FUME
CAS: 7439–96–5; Chemical Formula: MnO
H.S. No. 1236a

In construction, marine terminals, shipyards, and longshoring operations, OSHA currently has a ceiling limit of 5 mg/m³ for manganese fume (measured as manganese). There is no limit in agriculture. Because of this substance's potential to cause damage to the lungs and central nervous system, the ACGIH recommends an 8-hour TLV®–TWA of 1 mg/m³ and a 3-mg/m³ TLV®–STEL for manganese fume. NIOSH has no REL for this substance but concurs (Ex. 8–47, Table N1) that the limits OSHA is proposing are appropriate for this substance; these limits are 1 mg/m³ as

an 8-hour TWA and 3 mg/m³ as a 15-minute STEL. These are the limits recently established for this substance in general industry.

Manganese fume is the finely divided particulate generated when manganese metal is melted; the fume is gray or black in color. In experimental animals, daily 4-hour inhalation exposure to manganese dust (particle size not specified) caused fibrotic lung changes at concentrations of 10 to 20 mg/m³ for 3 to 6 months (ACGIH 1986, p. 354(86)). Russian studies have shown pulmonary effects in young rats administered intratracheal suspensions of mixed manganese oxides having particle sizes of less than 3 microns (Levina and Robachevsky 1955).

Symptoms of manganese poisoning in humans range from sleepiness and weakness in the legs (Fairhall 1957a) to difficulty in walking and uncontrolled laughter (Fairhall and Neal 1943). Health surveys of employees exposed to manganese fume have demonstrated a high incidence of pneumonia in these workers (Davies 1946). Tanaka and Lieben (1969/Ex. 1–388) found seven cases of pneumonia and 15 cases of borderline pneumonia among 144 workers exposed to manganese dust or fume concentrations greater than 5 mg/m³; three of these cases were associated with fume rather than dust exposure. Those workers exposed to fume levels below 5 mg/m³ exhibited no signs of pneumonia. In a separate study by Smyth, Ruhf, Whitman, and Dugan (1973/Ex. 1–990), three cases of manganese poisoning were detected among 71 employees exposed to 13.3 mg/m³ concentrations of manganese fume.

In the prior rulemaking, OSHA received one comment (Ex. 3–189) supporting the proposed limits for manganese fume and another (Ex. 3–829) objecting to the proposed change in PELs. In response to the latter commenter, OSHA emphasized that exposure to manganese fume has been demonstrated to cause toxic effects in both humans and animals. Workers exposed to manganese fumes have developed pneumonia (Tanaka and Lieben 1969/Ex. 1–388), and Stokinger (1981f, p. 1767) reports that the proposed 1-mg/m³ limit "is supported by the finding in animals that the higher oxides are more toxic, and the report of an occasional case of Mn poisoning in susceptible workers exposed to ferro Mn fumes around the 1-mg/m³ limit."

Based on this evidence, OSHA is proposing a 1-mg/m³ 8-hour TWA PEL and a 3-mg/m³ 15-minute STEL for manganese fume. The Agency preliminarily concludes that both a TWA limit and a STEL are required to protect employees in construction, maritime, and agriculture from the significant risks of manganism, lung damage, and pneumonitis, all of which constitute material health impairments that are associated with exposure to these fumes. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

MANGANESE CYCLOPENTADIENYL TRICARBONYL

CAS: 12079–65–1; Chemical Formula: C_5H_5—$Mn(CO)_3$

H.S. No. 1237

In construction, maritime, and agriculture, OSHA has no limit for manganese cyclopentadienyl tricarbonyl (MCT). The ACGIH has a TLV®–TWA of 0.1 mg/m³ (measured as manganese), with a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N1) that the limit OSHA is proposing is appropriate; this limit is an 8–hour TWA of 0.1 mg/m³. A skin notation is also proposed. Both the 0.1 mg/m³ 8–hour TWA PEL and the skin notation were recently established for MCT in general industry.

MCT is used as an antiknock agent in gasoline; no data on its physicochemical properties are available (ACGIH 1986, p. 356).

MCT is a convulsant and neuropathogen in animals. The oral LD_{50} in rats is 80 mg/kg, and the lowest lethal concentration by inhalation is 120 mg/m³ for 2 hours (RTECS 1990). Acutely poisoned animals exhibit the following symptoms regardless of the route of exposure: excitement, hyperactivity, tremors, toxic spasms, weakness, clonic convulsions, difficult breathing, and coma; at autopsy, liver and kidney damage is seen (Stara, Moore, Hysell, et al. 1974).

A Russian study reports that a single two-hour exposure to MCT at 120 mg/m³ was fatal to 80 percent of albino rats, although rabbits, guinea pigs, and rats survived a single 2–hour exposure to 20 to 40 mg/m³. Chronic exposure of rats for 11 months to concentrations averaging 1 mg/m³ for four hours daily caused delayed effects (seven months from onset of exposure) consisting of neuromuscular excitability, evidence of kidney damage, and decreased resistance to infection (Arkhipova, Tolgskaya, and Kochetkova 1965/Ex. 1–1046). The tails of 10 white mice were dipped in a gasoline mixture containing 1 gram MCT per 100 ml; a second group of mice had their tails immersed in gasoline without MCT. An equal number of fatalities were observed in the gasoline plus MCT and gasoline-only groups after four or five 2–hour applications, and all tails exhibited necrosis. The authors concluded that these effects were caused by the gasoline and not by the MCT (Arkhipova, Tolgskaya, and Kochetkova 1965/Ex. 1–1046). Further studies in rabbits showed that MCT applied dermally as an oil emulsion caused irritation of the skin. These authors also investigated the dermal toxicity of MCT solutions in tetrahydrofuran versus solutions of tetrahydrofuran in oil. All animals whose tails had been dipped in the hydrofuran solution of MCT died within an hour, while animals whose tails had been dipped in pure tetrahydrofuran did not (Arkhipova, Tolgskaya, and Kochetkova 1965/Ex. 1–1046). The same authors concluded that MCT is toxic at low concentrations, has cumulative properties, affects the nervous system, is irritating to the skin, and causes early histological changes in the respiratory tract.

More recent reports describe MCT-induced pulmonary edema and convulsions in the rat (Penney, Hogberg, Traiger, and Hanzlik 1985/Ex. 1–431). The ED_{50}s for convulsions were 32 mg/kg orally and 20 mg/kg intraperitoneally; LD_{50}s were 24 mg/kg orally and 14 mg/kg intraperitoneally. Necrosis of the bronchiolar tissue and pulmonary parenchymal damage were seen in mice and rats given intraperitoneal doses (Haschek, Hakkinen, Witschi et al. 1982/Ex. 1–1083).

Based on this evidence, OSHA has preliminarily concluded that occupational exposure to MCT poses a significant risk of neuropathic effects, kidney damage, skin irritation, and pulmonary edema, which together clearly constitute material impairments of health. The Agency is therefore proposing to establish an 8–hour TWA PEL of 0.1 mg/m³ for manganese cyclopentadienyl tricarbonyl, with a skin notation, to protect workers in construction, maritime, and agriculture against the significant risk of these effects. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

MANGANESE TETROXIDE

CAS: 1317–35–7; Chemical Formula: Mn_3O_4

H.S. No. 1238

OSHA has no exposure limit in construction, maritime, or agriculture for manganese tetroxide. The ACGIH recommends a TLV®–TWA of 1 mg/m³ (measured as manganese) for this brownish-black powder and its dust and fume. There is no NIOSH REL for this substance. OSHA is proposing a PEL of

1 mg/m³ TWA for manganese tetroxide (measured as Mn). This is the limit recently established for this substance in general industry.

Manganese tetroxide is a brownish black powder. It is generated during the pouring and casting of molten ferromanganese or the heating of manganese oxides (ACGIH 1986, p. 357).

Manganese tetroxide affects the nervous system and produces manganism in exposed individuals. This substance also damages the lungs. Findings from a Russian study indicate that intratracheal suspensions of manganese oxide, manganese dioxide, and manganese tetroxide particles (particle size less than 3 um) produced pneumonitis and other pulmonary effects in rats (Levina and Robachevskiau 1955/Ex. 1–1041). These investigators also determined that manganese tetroxide has greater toxicity than the lower oxides of manganese and that freshly prepared oxides are more potent than those stored for six months to one year. Two cases of manganese fume poisoning were reported in a plant where concentrations were between 2.7 and 4.7 mg/m³ (Whitlock, Amuso, and Bittenbender 1986/Ex. 1–455), but other investigators have questioned these air sampling results and believe that exposures to manganese tetroxide concentrations of 5 mg/m³ or less cause no harmful effects (Whitman and Brandt 1986/Ex. 1–1103).

In a 7-year study, Smyth and co-workers (1973/Ex. 1–990) investigated chronic manganese poisoning in workers exposed to both ferromanganese fumes and dust. Five of 71 employees suffered from chronic manganism; of these five cases, three resulted from fume exposure and two from dust exposure. Two of the three fume-exposed victims had been exposed over a five-year period to an estimated average ferromanganese concentration of 13.3 mg/m³; however, the third victim worked in an operation where ferromanganese concentrations of manganese were less than 1 mg/m³, which suggests that certain individuals may be hypersusceptible to manganese poisoning. The dust-exposed victims worked in areas where air concentrations were in the range of 30 to 50 mg/m³ throughout the study period (Smyth, Ruhf, Whitman, and Dugan 1973/Ex. 1–990).

Martonik (1976, as cited in ACGIH 1986, p. 357) reported that the fume of manganese has greater toxicity than the dust. During a two-year period, at least one case of acute manganese poisoning was documented at a fume concentration level of 7.5 mg/m³, and another case at the same welding operation may also have involved manganism.

NIOSH (Ex. 8–47; Tr. p. 3–86) commented in the earlier rulemaking that, based on the results of the Smyth and co-workers study (1973/Ex. 1–990), the 1–mg/m³ PEL being proposed by OSHA "may not be protective, especially to the potentially sensitive individual." OSHA is seeking comments on the appropriateness of the proposed limit for workplaces in construction, maritime, and agriculture.

Another commenter in the prior rulemaking (Ex. 3–189) asked OSHA to promulgate separate limits for the dust and fume of manganese tetroxide based on the relative toxicities of these two particulate forms. OSHA recognizes that some information in the literature (including some discussed above) points to the greater toxicity of the fume and that fumes are generally the more toxic form of particulate. However, the Agency notes that intratracheal suspensions of manganese tetroxide dust caused pneumonitis and other pulmonary effects in Russian workers (Levina and Robachevskiau 1955/Ex. 1–1041) and that several cases of manganism have been caused by manganese dust exposure (Smyth, Ruhf, Whitman, and Anger 1973/Ex. 1–990). The Agency thus believes that it would not be prudent at this time to distinguish between the dust and the fume but to propose an 8-hour TWA PEL that will protect against the effects of exposure to both forms of particulate.

In construction, maritime, and agriculture OSHA is proposing to establish a 1-mg/m³ 8-hour TWA PEL for manganese tetroxide. The Agency preliminarily concludes that this limit will provide protection to workers in these sectors from the significant risks of material health impairment in the form of chronic manganese poisoning, pneumonitis, and other respiratory and neurotoxic effects that are associated with exposure to manganese tetroxide at concentrations above 1 mg/m³. In addition, promulgation of these limits will make OSHA's PEL for this substance consistent across all regulated sectors.

MERCURY (ARYL AND INORGANIC COMPOUNDS)

CAS: 7439–97–6; Chemical Formula: Hg

H.S. No. 1240

For the construction and maritime industries, OSHA's current limit for all inorganic forms of mercury (Hg) (which includes aryl Hg) is 0.1 mg/m³ as an 8-hour TWA, with a skin notation; this limit was adopted from the 1970 ACGIH TLV® for inorganic mercury. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 0.1 mg/m³ with a skin notation or these substances. NIOSH (1973b) has recommended a 0.05-mg/m³ limit as an 8-hour TWA for mercury. OSHA is proposing to revise its exposure limit for aryl and inorganic mercury (measured as mercury) to 0.1-mg/m³ as a ceiling limit, together with a skin notation, and to apply this limit to workplaces in construction, maritime, and agriculture. NIOSH (Ex. 8–47, Table N1) concurs with this limit, which is the same PEL that was recently promulgated for these forms of mercury in general industry.

Mercury compounds are used in fungicides and preservatives; in dry cells; as catalysts and pigments; and in medicine. Mercuric nitrate was formerly used to manufacture fur felt hats (ACGIH 1986/Ex. 1–3, p. 359); the expression "mad as a hatter" derives from this use. When used in pesticidal applications and in accordance with the label, these substances are regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The soluble mercuric salts are highly poisonous when ingested; oral LD_{50}s for various animal species range from 1.0 to 18 mg/kg. Acute poisoning by mercury salts is rare in occupational settings; more important in this context are low-level chronic exposures, which are associated with the development of mercurialism. This disorder involves the central nervous system and results in tremors and neuropsychiatric disturbances (NIOSH 1973/Ex. 3–828, p. 18). The neurological effects of mercury poisoning are well known and have been extensively studied in humans for many years (see the discussion of mercury vapor, below). In addition, chronic low-level exposure to mercuric salts has been associated with glomerular disease (NIOSH 1973b/Ex. 3–828, p. 18).

In 1971, shortly after OSHA adopted the 0.1-mg/m³ TWA limit for inorganic mercury in construction and maritime, the ACGIH reduced the TLV®–TWA for all forms of mercury, including the inorganic compounds, to 0.05 mg/m³. ANSI also reduced its standard to 0.05 mg/m³ in 1972, and NIOSH recommended this same limit in 1973. The 0.05-mg/m³ limit was based largely on the study of Smith, Vorwald, Patil, and Mooney (1970/Ex. 1–373) of workers exposed to mercury levels ranging from less than 0.1 to 0.27 mg/m³ in chlor-alkali manufacturing plants. The authors reported a significant dose-related increase in the incidence of weight loss, tremors, abnormal reflexes, nervousness, and insomnia among workers exposed to concentrations of

0.1 mg/m³ or more. There were slight increases in incidences of insomnia and loss of appetite among workers exposed to 0.1 mg/m³ or less. Smith, Vorwald, Patil, and Mooney (1970/Ex. 1–373) concluded that a limit of 0.1 mg/m³ contained little or no margin of safety. Other studies (Bidstrup, Bonnell, Harvey, and Locket 1951/Ex. 1–1014; Turrian, Grandjean, and Turrian 1956) have also reported symptoms of mercury poisoning among workers exposed below 0.1 mg/m³. The 0.05-mg/m³ limit established by the ACGIH, ANSI, and NIOSH also followed the 1968 recommendation of an international committee (Permanent Commission and International Association on Occupational Health 1968).

In 1980, the ACGIH raised its recommended TLV® for aryl and inorganic mercury compounds from 0.05 mg/m³ to 0.1 mg/m³. In revising this limit, the ACGIH cited discrepancies in the literature regarding the ratio of blood and urinary mercury levels to airborne concentrations of mercury (Bell, Lovejoy, and Vizena 1973/Ex. 1–1078; Stopford et al. 1978/Ex. 1–1100). These studies reported that the mercury body burden to airborne concentration ratio was lower when personal sampling, rather than area sampling, was used. According to Bell, Lovejoy, and Vizena (1973/Ex. 1–1078), the lower ratio occurs when personal samples are being collected because contaminated clothing increases the amount of mercury inhaled, thus decreasing the ratio. On this basis, the ACGIH argued that the 0.05-mg/m³ limit may be too stringent when personal monitoring is performed. The ACGIH also believes that, in contrast to the effects of elemental or alkyl mercury, little mercury is deposited in the brain following exposure to aryl or inorganic mercury compounds. This reasoning led the ACGIH to adopt the higher 0.1-mg/m³ TLV®–TWA for aryl and inorganic compounds of mercury. However, the ACGIH (1986/Ex. 1–3, p. 358) also noted that, although central nervous system effects are less likely to occur from exposure to mercury salts than from other forms of mercury, the risk of renal and other effects would "presumably be just as great," and therefore that the higher limit for mercury salts "may be subject to debate" (ACGIH 1986/Ex. 1–3, p. 358).

In the prior rulemaking, Robert G. Smerko, President of the Chlorine Institute (Ex. 3–823; Tr. pp. 10–171 to 10–177), reviewed the pharmacologic evidence on the various forms of mercury and concluded that there is little difference in brain deposition between elemental mercury and mercury compounds:

> The ACGIH differentiated between aryl mercury and inorganic salts of mercury in comparison with elemental mercury vapor * * * While this is true for large doses of mercury, it overlooks the fact that absorbed elemental mercury is rapidly oxidized in the blood as reported by Clarkson et al. (1987). * * *
>
> Only when the rate of absorption exceeds the rate at which the body can oxidize mercury between the point of absorption and the brain does elemental mercury behave differently than aryl mercury and inorganic salts of mercury at the blood-brain barrier (Ex. 3–828, p. 7).

In light of this information, which counters the basis for the 0.1-mg/m³ ACGIH TWA®–TLV, and given the caution expressed by the ACGIH (1986/Ex. 1–3, p. 358) that the 0.1-mg/m³ TWA limit "may be subject to debate," OSHA preliminarily concludes that the PEL for aryl and inorganic mercury in construction, maritime, and agriculture should be 0.1 mg/m³ as a ceiling limit. The health studies cited above indicate that reducing the limit for these forms of mercury will ensure that employees are not at significant risk of adverse neuropathic effects from exposure to these forms of mercury and their compounds. Accordingly, OSHA is proposing to establish a 0.1-mg/m³ ceiling limit with a skin notation for aryl and inorganic mercury and its compounds (measured as mercury) in agriculture, maritime, and construction. OSHA is also proposing to add a skin notation to this limit to alert employers to the fact that mercury readily penetrates the skin to cause systemic poisoning; several cases of poisoning via this route have been reported (NIOSH 1973b; Ex. 3–828). OSHA is proposing to establish a PEL of 0.1 mg/m³ as a ceiling with a skin notation for aryl mercury and the inorganic compounds. OSHA believes that this limit is necessary to protect exposed workers in these sectors from the significant risks of neuropathy and systemic toxicity (both of which constitute material impairments of health) that are associated with exposure to these substances at higher levels. In addition, promulgation of these limits will make OSHA's PEL for these substances consistent across all regulated sectors.

MERCURY (VAPOR)
CAS: 7439–97–6; Chemical Formula: Hg
H.S. No. 1241

OSHA's current limit for mercury (including vapor) in construction and maritime is 0.1 mg/m³ as an 8-hour TWA, with a skin notation. There is no limit in agriculture. The ACGIH recommends a TLV®–TWA of 0.05 mg/m³ for mercury vapor, measured as mercury, and a skin notation. NIOSH has a REL of 0.05 mg/m³ for this substance as an 8-hour TWA. The Agency is proposing to reduce its limit for mercury vapor in construction and maritime to a PEL of 0.05 mg/m³ TWA, with a skin notation, and to extend this limit to agriculture. NIOSH (Ex. 8–47, Table N1) concurred that this limit is appropriate when it was established recently in general industry.

Elemental mercury is a silvery, odorless, heavy liquid. Elemental mercury is the only heavy metal that exists in a liquid state at ambient temperature. Because of this unique property, elemental mercury is used in many devices such as thermometers, barometers, manometers, electrical instruments, mercury vapor and fluorescent lamps, dental amalgams, and batteries. It is also used as a lubricant and in the production of caustic soda and chlorine (ACGIH 1986/Ex. 1–3, p. 359; HSDB 1986).

Exposure to mercury vapor presents both an acute and chronic health hazard. Inhalation of high concentrations of mercury vapor for relatively brief periods can cause pneumonitis, bronchitis, chest pain, dyspnea, coughing, stomatitis, gingivitis, salivation, and diarrhea (NIOSH 1973b/Ex. 3–828; Ashe, Largent, Dutra et al. 1953/Ex. 1–502). Chronic mercurialism is manifested by central nervous system effects, including tremor, a variety of neuropsychiatric disturbances, and loss of appetite (Kazantzis 1968; Smith, Vorwald, Patil, and Mooney 1970/Ex. 1–373).

Severe organ damage occurred in rabbits exposed for 4 hours to an average mercury vapor concentration of 28.8 mg/m³. Damage was observed in the kidneys, liver, brain, heart, lungs, and colon (Ashe, Largent, Dutra et al. 1953/Ex. 1–502). A study by Smith, Vorwald, Patil, and Mooney (1970/Ex. 1–373) of 567 male workers exposed to a mean exposure level of 0.065 mg/m³ (S.D. ± 0.085) showed a significant dose-related increase in the incidence of weight loss, tremors, abnormal reflexes, nervousness, and insomnia among workers exposed to concentrations of 0.1 mg/m³ or higher. There were slight increases in the incidence of insomnia and loss of appetite among workers exposed to 0.1 mg/m³ or less. Smith, Vorwald, Patil, and Mooney (1970/Ex. 1–373) concluded that a limit of 0.1 mg/m³ contained little or no margin of safety. Six of 75 workers regularly exposed to 0.05 to 0.1 mg/m³ of mercury vapor in a glassware manufacturing plant reported insomnia, and one was

found to have tremors (Danziger and Possick 1973/Ex. 1–504). One of 11 workers employed in a mercury mine or refining plant and exposed at vapor concentrations below 0.1 mg/m³ had sore gums, loose teeth, or excess salivation (Rentos and Seligman 1968/Ex. 1–523).

Two studies report no evidence of mercury vapor poisoning in industrial settings where characteristic exposures ranged between 0.05 and 0.1 mg/m³ (Danziger and Possick 1973/Ex. 1–504; McGill, Ladd, Jacobs, and Goldwater 1964/Ex. 1–520). In workers exposed at levels above 0.1 mg/m³, however, toxic symptoms were seen (Rentos and Seligman 1968/Ex. 1–523). Turrian, Grandjean, and Turrian (1956) found that 33 percent of workers exposed to the vapor at levels above 0.05 mg/m³ exhibited hyperexcitability, while only 8 percent of those exposed below that level manifested this symptom. About 20 percent of workers in both groups exhibited tremor. The ACGIH notes that, after exposure to the vapor, "a relatively high percentage of the absorbed mercury remains in the brain," compared with the amount deposited in the brain after exposure to the aryl and inorganic compounds (ACGIH 1986/Ex. 1–3, p. 359). The ACGIH accordingly recommends a higher TLV®–TWA for aryl and inorganic mercury than for mercury vapor (see, however, the discussion of aryl and inorganic mercury in this preamble).

Despite some of the uncertainties in the studies described above regarding the dose-response relationship between airborne exposure levels and health effects, OSHA believes that the data suggest that the current TWA PEL of 0.1 mg/m³ is not sufficiently protective. Given the severity of the neuropathic effects caused by mercury poisoning, OSHA believes that a reduction in the airborne limit is necessary to ensure that workers in construction, maritime, and agriculture are not at significant risk of mercury-related neuropathic effects. Therefore, OSHA is proposing to revise its PEL for elemental mercury vapor to 0.05 mg/m³ as an 8-hour TWA in agriculture, construction, and maritime. In addition, because skin absorption is a significant route of exposure and leads to systemic poisoning, OSHA is proposing a skin notation. The Agency preliminarily concludes that the 0.05 mg/m³ TWA limit and skin notation will substantially reduce the significant risks of acute and chronic mercury poisoning (which constitute material health impairments) that have been demonstrated to occur at exposure levels above 0.05 mg/m³. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

MERCURY, (ORGANO) ALKYL COMPOUNDS
CAS: 7439–97–6
H.S. No. 1242

OSHA's current limit for organo-mercury compounds in the construction and maritime industries is 0.01 mg/m³ as an 8-hour TWA, with a skin notation. The ACGIH has a TLV®–TWA of 0.01 mg/m³ and a TLV®–STEL of 0.03 mg/m³, with a skin notation, for these compounds, measured as mercury. The Agency is proposing to retain its permissible exposure limits of 0.01 mg/m³ as an 8-hour TWA, with a skin notation, in construction and maritime, to add a 0.03 mg/m³ STEL and to extend these limits to agriculture. In the prior rulemaking for general industry, NIOSH concurred (Ex. 8–47, Table N1) with these limits, which are now established in that sector.

Alkyl mercury compounds include volatile liquids, such as dimethyl and diethyl mercury, as well as many complex salts, which are usually solids. These compounds are used as fungicides in seed dressings, folial sprays, and preservatives for wood, paper pulp, textiles, and leather. When used in pesticidal applications and in accordance with the label, these substances are regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Alkyl mercury compounds pose greater health hazards than do the inorganic compounds of mercury because they can penetrate the blood-brain barrier and the placenta very quickly. The primary toxic effects associated with exposure to the organic compounds of mercury are injuries to the central and peripheral nervous systems and to the kidneys (Casarett and Doull 1975/Ex. 1–1144). In addition, data concerning mouse and rat exposures to alkyl mercury compounds have demonstrated toxicity to the gastrointestinal system, pancreas, liver, gonads, and cardiovascular system. Suppression of the immune system and impairment of the endocrine system have also been observed (Shakbazyan, Shevchenko, Borisenko et al. 1977/Ex. 1–933). Fatalities in mice have been reported following exposures of 10 to 30 mg/m³ for 3 to 5 hours (Trakhtenberg 1950/Ex. 1–447).

Methyl mercury is among the most damaging of the alkyl compounds to humans because it accumulates in the body and causes developmental effects (Wilson 1977/Ex. 1–457). A three-month exposure to approximately 1 mg/m³ diethyl mercury caused death in two individuals (Hill 1943/Ex. 1–786). Another fatal case of alkyl mercury poisoning has also been described (Hook, Lundgren, and Swensson 1954/Ex. 1–333). On the basis of his work with laboratory animals, Trakhtenberg (1950/Ex. 1–447) stated that even a concentration as low as 0.00001 mg/m³ could not be tolerated by humans on a continuing basis. However, a later study reported no consistent, acute effects of mercury poisoning at air concentrations between 0.01 and 0.1 mg/m³, despite the fact that brief excursions considerably above this range occurred (Dinman, Evans, and Linch 1958/Ex. 1–311). Organic mercury compounds can be absorbed through the skin (Sax and Lewis 1989).

There is no sharp distinction between acute and chronic exposures to alkyl mercury compounds. The latency for effects associated with acute exposures may be as long as several weeks, while the latency for chronic effects may be several years. Symptoms associated with exposure to alkyl mercury include numbness and tingling of the lips and extremities, ataxia, hearing impairment, and emotional disturbances (Permanent Commission and International Association on Occupational Health 1969, pp. 897–898). In addition, severe ocular effects, including gross constriction of the visual field and blindness, have been reported among exposed humans (Hunter, Bomford, and Russell 1940). Severe intoxication can result in clonic seizures, incontinence, and periods of spasticity; less severe poisoning is associated with dizziness, excess salivation, lacrimation, and gastrointestinal disturbances. In addition, acute exposure to alkyl mercury causes eye, skin, and mucous membrane irritation, skin burns, and, occasionally, skin sensitization (Dales 1972; Proctor, Hughes, and Fischman 1988, pp. 310–311).

OSHA is retaining its 8-hour TWA PEL of 0.01 mg/m³ and its skin notation in construction and maritime. The Agency is proposing to add a 15-minute STEL of 0.03 mg/m³ for the alkyl compounds of mercury (measured as Hg) in the construction and maritime industries and to extend both of these limits to agriculture. The Agency preliminarily concludes that exposure to the alkyl mercury compounds poses significant risks of severe neuropathic and other systemic injuries, irritation and skin sensitization, all of which constitute material health impairments. OSHA believes that both the short-term and 8-hour limits are necessary to substantially reduce these risks. In

addition, promulgation of these limits will make OSHA's PELs for these substances consistent across all regulated sectors.

METHYL ACETYLENE

CAS: 74–99–7; Chemical Formula: CH_3CCH

H.S. No. 2104

The current OSHA PEL for methyl acetylene in general industry, construction, and maritime is 1000 ppm as an 8-hour TWA. The Agency has no PEL for methyl acetylene in agriculture. The ACGIH has a TLV®–TWA of 1000 ppm and a TLV®–STEL of 1250 ppm for this substance. NIOSH has no REL for methyl acetylene but concurs with the PEL being proposed (Ex. 8–47, Table N3A). OSHA is proposing a PEL of 1000 ppm as an 8-hour TWA for methyl acetylene in agriculture. Promulgation of this limit will make the PEL for methyl acetylene consistent across all OSHA-regulated sectors.

Methyl acetylene is a colorless, flammable gas with a sweet odor. It is used in organic synthesis, as a rocket propellant, and as a fuel for welding torches (Braker and Mossman 1980, p. 450; ACGIH 1986, p. 368). This gas is shipped in liquefied form and thus presents a cryogenic hazard to those handling it.

In animals, methyl acetylene causes central nervous system effects and anesthesia at high concentrations. Rats exposed to a 42,000-ppm concentration initially became hyperactive and then exhibited lethargy and ataxia. The rats became completely anesthetized after 95 minutes of exposure to this concentration, but all rats survived such exposure for 5 hours and then recovered completely within 40 minutes of cessation of exposure. Examination of the lungs of rats at autopsy showed lung edema, alveolar hemorrhage, bronchiolitis, and pneumonitis (Horn, Weir, and Reese 1957). Dogs and rats exposed to a 28,700-ppm concentration of methyl acetylene became ataxic within minutes; dogs also exhibited staggering, excess salivation, and muscular fasciculations. Exposure to this concentration for 5 hours daily, 5 days per week for 6 months killed 40 percent of the rats (Horn, Weir, and Reese 1957).

There are no reports of adverse acute or chronic effects in humans exposed to methyl acetylene. Based on the effects noted in animals, however, methyl acetylene can be expected to depress the central nervous system and cause narcotic signs and symptoms in humans (Proctor, Hughes, and Fischman 1988, p. 317). Contact of the skin with liquefied methyl acetylene can cause frostbite (Braker and Mossman 1980, p. 451).

Based on this evidence, OSHA is proposing a 1000-ppm 8-hour TWA limit to protect workers in agriculture from the significant risk of central nervous system effects caused by exposure to higher concentrations of methyl acetylene. The Agency preliminarily concludes that this limit is necessary to substantially reduce a significant risk of material health impairment in exposed workers. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

METHYLACRYLONITRILE

CAS: 126–98–7; Chemical Formula: $CH_2=C(CH_3)C\equiv N$

H.S. No. 1251

In construction, maritime, and agriculture, OSHA has no PEL for methylacrylonitrile. The ACGIH recommends a 1-ppm TLV®–TWA and a skin notation to protect workers who are occupationally exposed to this substance. OSHA is proposing an 8-hour TWA permissible exposure limit of 1 ppm, with a skin notation, for methylacrylonitrile. NIOSH (Ex. 8–47, Table N1) has no REL but concurs that the proposed limit is appropriate. This limit and skin notation were recently established for methacrylonitrile in general industry.

Methylacrylonitrile (which is also called isopropene cyanide) is a colorless liquid that is used in the manufacture of plastic elastomers and coatings. This substance is also a chemical intermediate used to produce acids, amines, esters, nitriles, and amides.

Methylacrylonitrile is a chemical asphyxiant that is extremely toxic in animals, both by inhalation and dermal absorption. The LC_{50} in rats is 328 ppm for 4 hours, and the oral LD_{50} in the same species is 120 mg/kg (RTECS 1990). The dermal LD_{50} in rabbits is 320 mg/kg (RTECS 1990). Acutely poisoned animals convulsed before death (RTECS 1990). Death is believed to be caused by the metabolism of the acrylonitrile to cyanide, which, in turn, reacts with cytochrome oxidase in mitochondria and inhibits cellular respiration (Peter and Bolt 1985). Beagles exposed for 90 days to 13.5 ppm convulsed and lost motor control in their hind limbs; at autopsy, microscopic brain lesions were detected in one of the dogs (ACGIH 1986/Ex. 1–3, p. 370).

Based on this evidence of methylacrylonitrile's acute toxicity, OSHA is proposing a 1-ppm 8-hour TWA PEL and a skin notation for methylacrylonitrile in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit and notation will substantially reduce the significant risk of material health impairment in the form of nervous system damage that is associated with this substance. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

METHYL BROMIDE

CAS: 74–83–9; Chemical Formula: CH_3Br

H.S. No. 1253

OSHA's current PEL for methyl bromide in construction, shipyards, marine terminals, and longshoring operations is a 20-ppm ceiling with a skin notation; the ACGIH TLV®–TWA is 5 ppm, with a skin notation. NIOSH recommends that, based on its carcinogenicity, the REL for this substance be set at the lowest feasible level (Ex. 8–47, Table N6B). OSHA is proposing a permissible exposure limit of 5 ppm (8-hour TWA), with a skin notation, for methyl bromide; this is the limit recently established for this substance in general industry.

Methyl bromide is a colorless, nonflammable gas that has no taste and no odor at room temperature. At concentrations above 5 ppm, it has a sweetish odor. Methyl bromide was formerly used as a refrigerant and fire extinguishant, but these uses are now banned. Methyl bromide finds current use in industry in insect and rodent control and in soil fumigation (Farm Chemicals Handbook 1990, p. C192). Methyl bromide also is used as a methylating agent, to extract oils from nuts, seeds, and flowers, and to degrease wool (HSDB 1984). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Methyl bromide has acute neurotoxic effects in both animals and humans and, at high concentrations, this substance causes pulmonary effects. The oral LD_{50} in rats is 214 mg/kg, and the LC_{50} in the same species is 302 ppm for 8 hours (RTECS 1990). Rabbits exposed to a 70-ppm concentration of methyl bromide for 7 hours/day for 15 days showed signs of severe neurotoxicity, and many died (Skiov et al. 1981; Irish et al. 1940). Monkeys repeatedly exposed to a 65-ppm concentration of methyl bromide convulsed, lost their equilibrium, and became paralyzed (Irish et al. 1940). In a 90-day bioassay, methyl bromide was administered to rats by gavage; it caused a statistically significant increase in the incidence of squamous-cell carcinoma of the forestomach in

animals of both sexes (Danse et al. 1984).

Methyl bromide is a gas and thus primarily presents an inhalation hazard in industrial use, although there are suggestions that it can also be absorbed through the skin (Schifferli 1942). It is hypothesized that methyl bromide has a greater potential for toxicity than do other organic bromides because its greater lipophilicity provides increased access to the brain.

Various studies demonstrate methyl bromide's toxicity in humans. Ingram (1951/Ex. 1–175) reported ill effects (symptoms not specified) after exposure to methyl bromide at concentrations of 100 ppm. Similar exposure concentrations were also reported by Hine (1969/Ex. 1–70) in a case study of two date packers in California. Hine (1969/Ex. 1–70) also noted that methyl bromide has been responsible for more deaths among occupationally exposed pesticide workers in California than have the organophosphates. Johnson, Setzer, Lewis, and Anger (1977/Ex. 1–87) indicated that 34 packers became sick when exposed to an average methyl bromide concentration of 50 ppm; however, concentrations in the packing room may have been as high as 100 to 150 ppm during the purging of the fumigation chamber.

Watrous (1942/Ex. 1–275) described nausea, vomiting, and headache in 90 workers who were exposed for two weeks to concentrations "generally below" 35 ppm. Human fatalities caused by overexposure to methyl bromide have been estimated to occur at concentrations of 8000 ppm for a few hours and at concentrations as high as 60,000 ppm for 2 hours (Proctor, Hughes, and Fischman 1988, p. 322; RTECS 1990). Other authorities (Astley Clarke et al. 1945) believe that exposure to a 10,000 ppm concentration for more than a few minutes could be lethal.

In the prior rulemaking, NIOSH noted that methyl bromide is carcinogenic in animals and should be a candidate for a full Section 6(b) rulemaking (Ex. 8–47, Table N6B; Tr. pp. 3–97, 3–98). However, OSHA believes that the data to regulate methyl bromide as a carcinogen are incomplete at this time. Another commenter in the prior rulemaking (Ex. 116) urged OSHA to adopt a ceiling rather than 8-hour TWA limit for methyl bromide. OSHA believes that the proposed 5-ppm TWA will provide a substantial margin of safety against the methyl bromide exposures that have been shown to produce sickness in humans (generally in the 50- to 150-ppm range).

The presence of neurologic symptoms (nausea, headache, and vomiting) on exposure to concentrations of methyl bromide below 35 ppm indicates that the current ceiling limit of 20 ppm is not adequate to protect workers from the effects of methyl bromide poisoning. Accordingly, OSHA is proposing a PEL of 5 ppm as an 8-hour TWA, with a skin notation, to protect construction, maritime, and agricultural workers more adequately against these incapacitating symptoms. The Agency preliminarily concludes that this limit will reduce this significant risk substantially. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PENTABORANE

CAS: 19624–22–7; Chemical Formula: B_5H_9

H.S. No. 1304

In construction, shipyards, marine terminals, and longshoring, OSHA's current limit for pentaborane is 0.005 ppm as an 8-hour TWA. There is no PEL in agriculture. The ACGIH has the same 8-hour TLV®–TWA but also has a 15-minute TLV®–STEL of 0.015 ppm. OSHA is retaining its 8-hour TWA permissible exposure limit of 0.005 ppm in construction and maritime, is proposing to add a 0.015-ppm 15-minute STEL in these sectors, and is also proposing to extend both limits to agriculture. NIOSH has no REL for this substance but concurs (Ex. 8–47, Table N1) that these limits are appropriate. These are the limits recently established for pentaborane in general industry.

Pentaborane is a colorless liquid with a strong and penetrating odor. Pentaborane is used as a rocket propellant (ACGIH 1986, p. 459).

In both humans and animals, inhalation of pentaborane vapor causes central nervous system effects (Svirbely 1954a/Ex. 1–385; Rozendaal 1951/Ex. 1–525; Lowe and Freeman 1957/Ex. 1–518; Cordasco, Cooper, Murphy, and Anderson 1962/Ex. 1–545). The 5-minute LC_{50}s for rats and mice are 67 and 40 ppm, respectively; for 60-minute exposures, these values are 10 and 8 ppm, respectively (Weir, Seabaugh, Mershon, Burke, and Weeks 1964). Rats exposed repeatedly to a 3-ppm concentration of pentaborane exhibited tremors, hyperexcitability, belligerence, and weight loss (Svirbely 1954a/Ex. 1–385). Rats, rabbits, monkeys, and dogs exposed repeatedly to pentaborane vapor at concentrations of 1 ppm for four weeks or 0.2 ppm for six months lost weight (Levinskas, Paslian, and Bleckman 1958/Ex. 1–517). In the same experiments, rats and rabbits exposed at 1 ppm showed reduced activity and impaired locomotor ability, respectively, and monkeys and dogs exhibited apathy, loss of appetite, insensitivity to pain, loss of mobility, tremor, and impaired coordination. The ACGIH (1986/Ex. 1–3, p. 459) notes that the 0.2-ppm concentration reported in the Levinskas, Paslian, and Bleckman (1958/Ex. 1–517) study was a calculated rather than measured value and that the actual exposure level in this study was probably closer to 0.01 ppm. Pentaborane is also believed to be rapidly absorbed through the skin in toxic amounts (Parmeggiani 1983, p. 318).

Humans accidentally overexposed to pentaborane have experienced tremors, convulsions, behavioral changes, loss of memory, impaired judgment, and other symptoms of central nervous system intoxication (Svirbely 1954a/Ex. 1–385; Rozendaal 1951/Ex. 1–525; Lowe and Freeman 1957/Ex. 1–518; Cordasco, Cooper, Murphy, and Anderson 1962/Ex. 1–545). The onset of symptoms may be delayed in humans for as long as 24 hours, and severe overexposure may cause death. A fatality occurred when a worker was severely overexposed both by inhalation and percutaneous absorption; the victim in this case convulsed and had severe opisthotonic spasms. Autopsy revealed necrotizing pneumonia in both lungs, fatty changes and centrilobular degeneration in the liver, and widespread degeneration of the brain (Yarbrough, Garrettson, Zolet et al. 1986). A worker in a nearby building at the time of the accident survived but experienced such severe nervous system injury that he had to be institutionalized (Yarbrough et al. 1986). Based on findings in animals, it is estimated that exposure to an 8-ppm concentration of pentaborane for 1 hour would cause slight toxicity in humans, while exposure to 30 ppm for the same interval would cause death (Proctor, Hughes, and Fischman 1988, p. 396).

Based on this evidence of pentaborane's extreme toxicity, OSHA is proposing to retain its 8-hour TWA PEL of 0.005 ppm in construction and maritime, to add a 15-minute STEL of 0.015 ppm for pentaborane in construction and maritime, and to extend both limits to agriculture. The Agency preliminarily concludes that these limits will protect workers in these sectors against the significant risk of central nervous system effects, such as tremors and convulsions, behavioral changes, and loss of judgment, potentially associated with exposure to pentaborane. OSHA preliminarily finds that these neuropathic effects constitute material health impairments within the meaning of the Act. In addition, promulgation of these limits will make

OSHA's PELs for this substance consistent across all regulated sectors.

PHENYL MERCAPTAN

CAS: 108–98–5; Chemical Formula: C_6H_5SH

H.S. No. 1316

In construction, maritime, and agriculture, OSHA has no exposure limit for phenyl mercaptan. The ACGIH has a TLV®-TWA of 0.5 ppm, and NIOSH recommends a 15-minute ceiling limit of 0.1 ppm for phenyl mercaptan. OSHA is proposing a permissible exposure limit of 0.5 ppm as an 8-hour TWA for phenyl mercaptan in construction, maritime and agriculture. This is the limit recently established for this substance in general industry.

Phenyl mercaptan, also called benzenethiol, is a colorless liquid with an offensive, garlic-like odor. It is used as a chemical intermediate in the manufacture of pharmaceuticals, insecticides, fungicides, and other chemicals (HSDB 1985). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The primary acute hazards of exposure to phenyl mercaptan are central nervous system stimulation followed by post-convulsive CNS depression, severe eye and skin irritation, systemic toxicity to spleen, kidney, lung, and liver tissues, and narcotic effects (Fairchild and Stokinger 1958). This substance is highly toxic by inhalation, ingestion, and percutaneous absorption (Clayton and Clayton 1981, p. 2080).

Phenyl mercaptan has 4-hour inhalation LC_{50} values of 33 and 28 ppm in rats and mice, respectively (RTECS 1990), and the oral LD_{50} in rats is 46 mg/kg (RTECS 1990; McCord and Witheridge 1949/Ex. 1–882). In rabbits and rats, dermal LD_{50} values are 134 mg/kg and 300 mg/kg, respectively (RTECS 1990). The responses of acutely poisoned animals to phenyl mercaptan exposure were uniform regardless of species, and progressed from CNS stimulation to incoordination, skeletal and muscular paralysis, and respiratory depression, followed at high concentrations by coma and death. High doses (not further specified) administered via inhalation produced lung, liver, and kidney changes in mice (Doull and Plzak 1962; Fairchild and Stokinger 1958/Ex. 1–415); Schafer 1972/Ex. 1–362). In rabbits, phenyl mercaptan is a severe eye and skin irritant (McCord and Witheridge 1949/Ex. 1–882; Robles 1975; Schafer 1972/Ex. 1–362).

In humans, phenyl mercaptan is a moderately severe skin irritant and neurotoxin; it causes headaches and dizziness at unspecified levels (Fairchild and Stokinger 1958/Ex. 1–415; McCord and Witheridge 1949/Ex. 1–882). In the prior rulemaking, NIOSH (Ex. 8–47, Table N7; Tr. p. 3–99) stated that, in its opinion, the limit for phenyl mercaptan should be a ceiling rather than a time-weighted average. In response to NIOSH, OSHA stated that a TWA limit set at 0.5 ppm will ensure that workplace concentrations are consistently maintained at levels low enough to protect against phenyl mercaptan's acute toxic effects.

Based on this evidence of phenyl mercaptan's toxicity, OSHA is proposing an 8-hour TWA limit of 0.5 ppm. The Agency preliminarily concludes that this limit will protect construction, maritime, and agriculture workers from the significant risks of CNS effects, skin irritation, and systemic injury, all material impairments of health that are potentially associated with exposure to phenyl mercaptan at the uncontrolled levels permitted by the absence of an OSHA limit for this substance. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PROPYLENE GLYCOL DINITRATE

CAS: 6423–43–4; Chemical Formula: $C_3H_6N_2O_6$

H.S. No. 1342

In construction, maritime, and agriculture, OSHA has no exposure limit for propylene glycol dinitrate (PGDN). The ACGIH recommends a TLV®-TWA of 0.05 ppm, with a skin notation. NIOSH has no REL for PGDN but concurs (Ex. 8–47, Table N1) with the limit OSHA is proposing, which is an 8-hour TWA PEL of 0.05 ppm. This is the limit recently established for this substance in general industry.

When freshly prepared, propylene glycol dinitrate is a colorless liquid with a disagreeable odor. It is used as a torpedo propellant (ACGIH 1986, p. 502).

Exposure to this substance affects blood pressure, causes methemoglobinemia, methemoglobinuria, and respiratory toxicity, injures liver and kidney tissues, and distorts vision in humans and animals. Propylene glycol dinitrate can also cause headache and incoordination, both indicators of nervous system effects.

The oral LD_{50} value in rats is 250 mg/kg (RTECS 1990). In all species studied, death occurs by anoxia, which is caused by the almost complete conversion of hemoglobin to methemoglobin (Clark and Litchfield 1969/Ex. 1–543). Ocular instillation of the liquid caused transient conjunctival redness (Jones, Strickland, and Siegel 1972/Ex. 1–742). Twenty-day skin exposures in rabbits at a 2-g/kg dose caused weakness and cyanosis; one of five rabbits died, and this animal's hemoglobin and hematocrit values had decreased. When the dose was increased to 4 g/kg, the rabbits' methemoglobin values rose to 34.5 percent by the time of death (Jones, Strickland, and Siegel 1972/Ex. 1–742). Continuous 90-day inhalation exposures to a 10-ppm concentration caused kidney and liver changes in dogs; exposures to 35 ppm caused heavy iron deposits in the liver, spleen, and kidneys. Female (but not male) rats showed a drop in blood pressure within 30 minutes after injection of PGDN doses above 5 mg/kg. Rhesus monkeys displayed mydriasis in 90-day exposures at 35 ppm but no change in avoidance behavior during a visual discrimination and acuity threshold test (Jones, Strickland, and Siegel 1972/Ex. 1–742).

In humans, eight-hour exposures to 0.2 ppm or higher concentrations of propylene glycol dinitrate resulted in visual distortion and headache (Stewart, Peterson, Newton et al. 1974). Although subjects developed a tolerance for the headache response, the visual effects of exposure were cumulative. Impaired balance occurred after 6.5 hours of exposure to a 0.5-ppm concentration, and a 40-minute exposure to 1.5 ppm caused eye irritation. Subjects exposed at 0.5 ppm for 8 hours experienced a consistent elevation in diastolic pressure but no pulmonary irritation. At concentrations of 0.03 to 1.5 ppm, no hematologic effects were observed (Stewart, Peterson, Newton et al. 1974). Studies of human exposures to levels below 0.1 ppm do not report chronic neurotoxicity (Horvath, Ilka, Boyd, and Markhan 1981/Ex. 1–557).

Based on this evidence in humans and animals, OSHA is proposing an 8-hour TWA limit of 0.05 ppm for propylene glycol dinitrate. The Agency preliminarily concludes that this limit will protect construction, maritime, and agricultural workers against the significant risks of hepatotoxic, hematologic, and central nervous system effects (all of which constitute material health impairments) that exist from workplace exposure at the levels currently permitted by the absence of an OSHA PEL. In addition, promulgation of these limits will make OSHA's PEL for this substance consistent across all regulated sectors.

Preliminary Conclusions. OSHA preliminarily concludes that significant

risks are associated with occupational exposure to the group of neuropathic toxicants shown in Table C1–1. The effects caused by such exposures include brain lesions, nausea, vomiting, general depression of the central nervous system, interference with sensory and motor functions, and alterations in the ability of the brain to process information. Affected workers may experience drowsiness, dizziness, loss of ability to concentrate, mood changes, reduced awareness, learning difficulties, unsteadiness, and auditory and visual disturbances. In addition, employees experiencing these effects are imperiled and are likely to hurt themselves or others in accidents caused by their reduced functional capacities. OSHA believes that promulgation of the proposed exposure limits for these neurotoxins will substantially reduce such risks and afford protection to construction, maritime, and agricultural workers against these material health impairments. In addition, promulgation of these limits will make OSHA's PELs for these substances consistent across all regulated sectors.

2. Substances for Which Proposed Limits are Based on Avoidance of Narcotic Effects

Introduction. OSHA is proposing new or revised limits for 24 substances based primarily on evidence showing that occupational exposure to these substances causes narcosis. The narcotic effects of exposure to such substances as the alcohols, aliphatic hydrocarbons, and chlorinated hydrocarbons have been recognized as serious for many years. Table C2–1 lists these chemicals, along with their CAS numbers, HS numbers, 1987–1988 ACGIH TLV®s, and NIOSH RELs. In addition, Table C2–1 shows OSHA's current permissible exposure limits (PELs) for these substances in construction, shipyards, marine terminals, and longshoring. OSHA has no PELs that apply to workers in agricultural workplaces. The right-hand column in Table C2–1 shows OSHA's current PELs for these substances in general industry; these are the limits being proposed today for construction, maritime, and agricultural workplaces.

TABLE C2–1. SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON AVOIDANCE OF NARCOSIS

H.S. No./chemical name	CAS No.	Current OSHA PEL in construction, shipyards, marine terminals, and longshoring*	1987–1988 ACGIH TLV®**	NIOSH REL ***	Proposed OSHA PEL in construction, maritime, and agriculture
1044 Butane	106–97–8		800 ppm TWA		800 ppm TWA.
1049 sec-Butyl alcohol	78–92–2	150 ppm TWA	100 ppm TWA, 150 ppm STEL		100 ppm TWA.
1050 tert-Butyl alcohol	75–65–0	100 ppm TWA	100 ppm TWA, 150 ppm STEL		100 ppm TWA, 150 ppm STEL.
2034 Chlorobromomethane	74–97–5	200 ppm TWA	200 ppm TWA, 250 ppm STEL		200 ppm TWA.
2044 Cumene	[illegible]	50 ppm TWA, Skin.	50 ppm TWA, Skin		50 ppm TWA, Skin.
1111 Cyclopentane	287–92–3		600 ppm TWA		600 ppm TWA.
2076 Ethyl alcohol	64–17–5	1000 ppm TWA	1000 ppm TWA		1000 ppm TWA.
1163 Ethyl bromide	74–96–4	200 ppm TWA	200 ppm TWA, 250 ppm STEL		200 ppm TWA, 250 ppm STEL.
2078 Ethyl chloride	75–00–3	1000 ppm TWA	1000 ppm TWA	Handle with caution in the workplace.	1000 ppm TWA.
1185 Gasoline	8006–61–9		300 ppm TWA, 500 ppm STEL		300 ppm TWA, 500 ppm STEL.
1194 Heptane	142–82–5	500 ppm TWA	400 ppm TWA, 500 ppm STEL	85 ppm TWA, 440 ppm Ceiling, (15-min).	400 ppm TWA, 500 ppm STEL.
1201 Hexane isomers	None		500 ppm TWA, 1000 ppm STEL	100 ppm TWA, 510 ppm Ceiling, (15-min).	500 ppm TWA, 1000 ppm STEL.
1218 Isoamyl alcohol (primary and secondary).	123–51–3	100 ppm TWA	100 ppm TWA, 125 ppm STEL		100 ppm TWA, 125 ppm STEL.
1221 Isophorone	78–59–1	25 ppm TWA	5 ppm Ceiling	4 ppm TWA	4 ppm TWA.
1254 Methyl chloride	74–87–3	100 ppm Ceiling	50 ppm TWA, 100 ppm STEL	(*)	50 ppm TWA, 100 ppm STEL.
1255 Methyl chloroform (1,1,1-Trichloroethane).	71–55–6	350 ppm TWA	350 ppm TWA, 450 ppm STEL	350 ppm Ceiling (15-min).	350 ppm TWA, 450 ppm STEL.
2113 Naphtha (coal tar)	8030–30–6	100 ppm TWA			100 ppm TWA.
1296 Octane	111–65–9	400 ppm TWA	300 ppm TWA, 375 ppm STEL	75 ppm TWA, 385 ppm Ceiling (15-min).	300 ppm TWA, 375 ppm STEL.
1306 Pentane	109–66–0	500 ppm TWA	600 ppm TWA, 750 ppm STEL	120 ppm TWA, 610 ppm Ceiling (15-min).	600 ppm TWA, 750 ppm STEL.
1307 2-Pentanone (Methyl propyl ketone).	107–87–9	200 ppm TWA	200 ppm TWA, 250 ppm STEL	150 ppm TWA	200 ppm TWA, 250 ppm STEL.
1371 Stoddard solvent	8052–41–3	200 ppm TWA	100 ppm TWA	350 mg/m³ TWA, 1800 mg/m³ Ceiling (15-min).	100 ppm TWA.
1372 Styrene	100–42–5	100 ppm Ceiling	50 ppm TWA, 100 ppm STEL	50 ppm TWA, 100 ppm Ceiling (15-min).	50 ppm TWA, 100 ppm STEL.
1397 Toluene	108–88–3	200 ppm TWA	100 ppm TWA, 150 ppm STEL	100 ppm 8-hour TWA, 200 ppm Ceiling (10-min).	100 ppm TWA, 150 ppm STEL.
1406 Trichloroethylene	79–01–6	100 ppm TWA	50 ppm TWA, 200 ppm STEL	25 ppm TWA*	50 ppm TWA, 200 ppm STEL.

* OSHA's PELs do not currently apply in Agriculture; OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TLV®-TWA is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times in any working day, with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10 hour/day, 40 hour/week exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

* NIOSH considers this substance a potential occupational carcinogen and recommends that exposures be reduced to the lowest feasible concentration

Description of the Health Effects. Narcosis is caused by a general depression of central nervous system (CNS) function. When the CNS becomes sufficiently depressed, the awareness or consciousness of affected persons is diminished. Initial symptoms of narcosis include drowsiness, difficulty in concentration, and mood changes; these effects may progress to slurred speech, dizziness, loss of coordination, and, in more severe cases, loss of consciousness, coma, and death. Except in more serious cases, CNS depression is reversible if exposure ceases. However, because narcosis adversely affects the concentration and coordination of affected workers, these workers and their co-workers are at increased risk of injuries and accidents caused by slowed reaction times, incoordination, and mistakes and errors in judgment.

The mechanism by which exposure to substances induces narcosis is poorly understood. It is believed that central nervous system depressants may have the same mechanism of action as general anesthetics, which appear to produce a reversible effect on electrically excitable neuronal membranes.

Dose-Response Relationship and Narcotic Effects

The induction of narcosis following exposure to narcotic agents appears to follow the classical S-shaped (sigmoidal) dose-response relationship. As exposure level increases, both the percent of exposed persons affected and the severity of the effect increase. Although it is not known whether a true threshold exists for the occurrence of the molecular events leading to narcosis (i.e., disruption of electrical impulses in neurons), there is usually a level at which most exposed individuals will manifest the onset of symptoms associated with narcosis. The no-effect level for a particular substance is determined largely by individual susceptibility, the extent to which the material is absorbed, and the rate at which it is metabolized and eliminated.

The following discussions describe OSHA's preliminary findings for the substances in this group and illustrate the material health impairments potentially associated with workplace exposure to these central nervous system depressants.

BUTANE

CAS: 106–97–8; Chemical Formula: C_4H_{10}
H.S. No. 1044

In construction, shipyards, marine terminals, longshoring operations, and agriculture, OSHA has no limit for butane. The 1987–1988 ACGIH TLV®–TWA is 800 ppm; there is no NIOSH REL. OSHA is proposing an 8-hour TWA PEL of 800 ppm for butane in construction, maritime, and agriculture, and NIOSH (Ex. 8–47, Table N1) concurs that this limit is appropriate. This is the limit recently established for this substance in general industry.

Butane is used to calibrate pressure and temperature gauges and to fill the thermobulbs on these instruments. It also finds wide use as an intermediate in organic synthesis and in the manufacture of aviation, heating, and motor fuel (Braker and Mossman 1980, p. 89). The odor of butane is not detectable at airborne concentrations below 50,000 ppm (Braker and Mossman 1980, p. 89). Because butane is shipped as a liquefied gas under pressure, contact with the liquid form may cause frostbite.

The primary risk of exposure to butane is narcosis. In rats, the 4-hour LC_{50} is 658 g/m³, or about 280,000 ppm; in mice, this value is 680 g/m³ for 2 hours (NIOSH 1977i/Ex. 1–1182). n-Butane causes anesthesia in mice at airborne concentrations of 130,000 ppm after exposure for 25 minutes. This substance also is a relatively weak cardiac sensitizer in dogs (Reinhardt, Azar, Maxfield et al. 1971; Stoughton and Lamson 1936).

Humans exposed to a 1000-ppm concentration of butane for a single 8-hour day or to a 500-ppm concentration for 2-week periods of 8-hour workdays showed no harmful subjective or abnormal physiological responses but did show a reduced visual evoked response (VER) wave amplitude during the second week (Stewart, Herrman, Baretta et al. 1977/Ex. 1–575). In contact with the eyes or skin, liquefied butane causes burns or frostbite (Clayton and Clayton 1981, p. 3182).

Based on this evidence, OSHA is proposing an 8-hour TWA permissible exposure limit (PEL) of 800 ppm for butane; this limit would apply to workplaces in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risks of drowsiness, narcosis, and other central nervous system effects associated with exposures at the uncontrolled levels permitted by the absence of an OSHA limit. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

sec-BUTYL ALCOHOL

CAS: 78–92–2; Chemical Formula: $CH_3CH_2CHOHCH_3$
H.S. No. 1049

OSHA's current limit for sec-butyl alcohol in construction, shipyards, marine terminals, and longshoring operations is 150 ppm as an 8-hour TWA. There is no PEL in agriculture. NIOSH has no REL for sec-butyl alcohol. The 1987–1988 ACGIH TLV®s for this substance are 100 ppm as an 8-hour TWA and 150 ppm as a 15-minute STEL. However, the ACGIH deleted the STEL for this substance in 1990 because there was no evidence that a STEL was needed; in OSHA's prior air contaminants rulemaking, the American Industrial Hygiene Association commented that no STEL was necessary to protect against sec-butyl alcohol's toxic effects (Ex. 8–16). After reviewing the available toxicologic evidence, OSHA agreed with this position (54 FR 2423), and the final rule for general industry (29 CFR 1910.1000, Table Z–1–A) thus contains only an 8-hour TWA PEL for this substance. Accordingly, OSHA is proposing an 8-hour TWA PEL of 100 ppm for sec-butyl alcohol in construction, maritime, and agriculture, and NIOSH concurs (Ex. 8–47, Table N1) that the proposed limit is appropriate. This is the limit recently established for this substance in general industry.

sec-Butyl alcohol is a colorless liquid with a strong, wine-like odor. It is used as a solvent for natural resins and linseed and cottonseed oils, and as a chemical intermediate. sec-Butyl alcohol is also an ingredient in a wide variety of commercial products, including polishes, flotation agents, flavors, perfumes, dyestuffs, wetting agents, paint removers, brake fluids, and industrial cleaners (HSDB 1985).

sec-Butyl alcohol is a narcotic and irritant both in humans and animals. The oral LD_{50} in rats is 6480 mg/kg, and the lowest lethal concentration in the same species is 16,000 ppm for 4 hours (RTECS 1990). Mice exposed to sec-butyl alcohol concentrations of 3300 to 19,800 ppm showed loss of coordination, prostration, and narcosis (Rowe and McCollister, in Clayton nd Clayton

1982, pp. 4582–4585). Liquid sec-butyl alcohol instilled into the eyes of rabbits caused severe corneal injury (Smyth, Carpenter, Weil, and Pozzani 1954).

Excessive exposure to sec-butyl alcohol is reported to have caused eye, nose, and throat irritation; headaches; nausea; fatigue; and dizziness in exposed humans (Rowe and McCollister, in Clayton and Clayton 1982, p. 4585). Workers exposed repeatedly to sec-butyl alcohol concentrations of 100 ppm, however, reportedly experienced no symptoms at this concentration (ACGIH 1986, p. 77/Ex. 1–3).

Based on this evidence in humans and animals, OSHA is proposing a PEL for sec-butyl alcohol in construction, maritime, and agriculture of 100 ppm as an 8-hour TWA. The Agency believes that this limit will afford workers in these sectors protection against the significant risks of narcosis and irritation that are associated with exposures to sec-butyl alcohol at concentrations above the proposed PEL. The Agency preliminarily concludes that the proposed limit will substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

tert-BUTYL ALCOHOL
CAS: 75–65–0; Chemical Formula:
$(CH_3)_3COH$
H.S. No. 1050

In construction, marine terminals, shipyards, and long-shoring operations, OSHA currently has a limit of 100 ppm as an 8-hour TWA for tert-butyl alcohol. There is no PEL in agriculture. The 1987–1988 ACGIH TLV®–TWA for this substance is 100 ppm as an 8-hour TWA, with a TLV®–STEL of 150 ppm. NIOSH has no REL for tert-butyl alcohol but concurs (Ex. 8–47, Table N1) with the limits OSHA is proposing. OSHA is retaining the 100 ppm 8-hour TWA PEL in construction and maritime, is proposing 150 ppm as a 15-minute STEL in these two sectors, and is proposing both the PEL and the STEL in agriculture. These are the limits recently established for this substance in general industry.

At room temperature, tert-butyl alcohol is a colorless, crystalline solid with a camphor-like odor (ACGIH 1986/Ex. 1–3; HSDB 1985). tert-Butyl alcohol is an ingredient of perfumes, flavorings, flotation agents, cleaning compounds, paint removers, fruit essences, plastics, and lacquers. This substance is also used as an octane booster in gasoline, to extract water from substances, and in pharmaceutical production. In addition, tert-butyl alcohol is an intermediate in the production of resins, artificial musk, and other substances (HSDB 1985). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Although similar to the other butyl alcohols in many respects, tert-butyl alcohol is more volatile and has a greater potential for narcotic effects than other butyl alcohols (Weese 1928/Ex. 1–1073). The oral LD_{50} in rats is 3.5 g/kg (RTECS 1990). Acutely poisoned animals showed loss of coordination and other signs of narcosis (Schaffarzick and Brown 1952/Ex. 1–868). Repeated daily doses of tert-butyl alcohol that produced signs of narcosis were not fatal in experimental animals (Schaffarzick and Brown 1952/Ex. 1–868).

In humans, tert-butyl alcohol is known to cause slight redness and irritation when the liquid is applied to the skin (Oettel 1936). Exposure to concentrations described only as "excessive" is reported to have caused eye, nose, and throat irritation and narcotic symptoms, including headache, nausea, dizziness, and fatigue, in exposed workers (Schwartz and Tulipan, in Clayton and Clayton 1982, p. 4587).

Based on this evidence, OSHA is retaining the 8-hour TWA PEL of 100 ppm in construction and maritime, proposing to add a 15-minute STEL of 150 ppm in construction, maritime, and proposing to extend both PELs to agriculture. The Agency preliminarily concludes that this combination of limits will protect workers in these sectors against the significant risk of narcosis that is potentially associated with occupational exposures to this substance. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

CHLOROBROMOMETHANE
CAS: 74–97–5; Chemical Formula:
CH_2BrC1
H.S. No. 2034

OSHA's limit for chlorobromomethane in general industry, construction, and maritime is 200 ppm as an 8-hour time-weighted average (TWA). There is no limit in agriculture. The 1987–1988 ACGIH TLV®–TWA for chlorobromomethane is 200 ppm as an 8-hour TWA, and the TLV®–STEL is 250 ppm. There is no NIOSH REL for this substance, but NIOSH concurs (Ex. 8–47, Table N3A) with the limit being proposed. The Agency is retaining the current limit in construction and maritime and is proposing an 8-hour TWA PEL of 200 ppm for chlorobromomethane in agriculture. This is the limit recently established for this substance in general industry.

Chlorobromomethane (also called methylene chlorobromide) is a colorless to pale yellow liquid that has a sweet, chloroform-like odor (ACGIH 1986, p. 125; Sittig 1985, p. 228). It is used as a fire extinguishing agent and as a chemical intermediate in organic synthesis (HSDB 1986; Clayton and Clayton 1982, p. 3455).

In both humans and animals, acute exposure to chlorobromo-methane causes irritation of the eyes, nose, and mucous mem-branes, and central nervous system depression (narcosis); in animals, chronic exposure causes lung, liver, and kidney damage. The oral LD_{50} in rats is 5000 mg/kg (RTECS 1989). The lowest lethal concentration in rats is 28,800 ppm for 15 minutes (RTECS 1989). Acutely poisoned animals showed incoordination and narcosis before death; pulmonary edema and interstitial pneumonitis were also seen in these animals (Comstock, Fogelman, and Oberst 1953). Inhalation studies have shown that rats exposed to 3000 ppm of chlorobromomethane experienced light narcosis (Comstock, Fogelman, and Oberst 1953). In mice, fatty degeneration of the liver and kidneys occurred after a single dose of 3000 mg/kg was administered by stomach tube; however, a dose of 500 mg/kg caused no organ damage (Highman, Suirbely, von Oettingen et al. 1948). Transient corneal epithelial injury and conjunctival edema occurred in rabbit eyes after the eyes had been sprayed with a liquid mixture of chlorobromomethane (75%) and dichlorodifluoromethane (25%) from a fire extinguisher (Grant 1986, p. 210). Dermal testing in rabbits caused moderate irritation, hyperemia, and exfoliation of the skin (Rutstein 1963). Rats, rabbits, and dogs exposed 7 hours/day, 5 days/week for 14 weeks to a 1000-ppm concentration of chlorobromomethane showed no signs of toxicity (Suirbely, Highman, Alford et al. 1947). Female rats and dogs exposed to a 500-ppm concentration of chlorobromomethane for 7 hours/day, 5 days/week for a period of 6 months showed some liver pathology at autopsy. Elevated blood bromide was the only observed effect in male rats, guinea pigs, and rabbits exposed to the same dose. Histopathological changes in the livers and testes as well as elevated blood bromide were observed in guinea pigs, rats, and rabbits when the concentration of chlorobromo-methane was increased

to 1000 ppm (Suirbely, Highman, Alford et al. 1947).

Three firefighters exposed to very high but unknown vapor concentrations of chlorobromomethane experienced irritation of the eyes and throat, disorientation, headache, and nausea. Two of the victims later became comatose, one suffered convulsive seizures, and another required resuscitation following respiratory arrest; all recovered fully following supportive treatment (Rutstein 1963). An individual who was sprayed in the face by a fire extinguisher containing chlorobromomethane suffered from an immediate and severe burning sensation of the eyes. Medical examination of this individual revealed a partial loss of corneal epithelium and hyperemic and edematous conjunctivae and eyelids, but no permanent eye damage resulted from this exposure (Grant 1986, p. 210).

Based on the evidence presented above, OSHA preliminarily concludes that agricultural workers exposed to chlorobromomethane at the levels permitted by the absence of a limit are at significant risk of experiencing eye irritation and narcosis. The Agency believes that establishing a PEL of 200 ppm as an 8-hour TWA will protect workers in agriculture from these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CUMENE

CAS: 98–82–8; Chemical Formula: $C_6H_5C_3H_7$

H.S. No. 2044

OSHA's current permissible exposure limit for cumene in general industry, construction, and maritime is 50 ppm as an 8-hour TWA; this limit also has a skin notation. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 50 ppm, with a skin notation, for cumene. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing to establish an 8-hour TWA PEL of 50 ppm, with a skin notation, for cumene in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Cumene is a colorless liquid with a sharp, aromatic odor. This substance is used as a solvent and in the production of phenol, acetone, and alpha-methylstyrene (ACGIH 1986, p. 151; Genium MSDS 1987, No. 395).

Cumene causes eye, nose, skin, and upper respiratory tract irritation in humans and animals; at high concentrations, it causes central nervous system depression in animals. The oral LD_{50} in rats is 1400 mg/kg, and the LC_{50} in mice is 24,700 mg/m³ for 2 hours (RTECS 1987). The dermal LD_{50} in rabbits is 12,300 mg/kg (ACGIH 1986, p. 151; RTECS 1987). Rabbits showed mild eye irritation when 86 mg of cumene was instilled into their eyes, and a dermal dose of 10 mg caused mild skin irritation in rabbits (RTECS 1987). Fifty percent of mice exposed to 2040 ppm for 7 hours died; the signs and symptoms of acutely poisoned mice included dilation of cutaneous blood vessels, central nervous system depression, and respiratory depression (Werner, Dunn, and von Oettingen 1944). Repeated inhalation exposures to concentrations of 2000 ppm caused ataxia and lethargy in rabbits (Proctor, Hughes, and Fischman 1988, p. 166). At autopsy, rats exposed to 500 ppm cumene daily for 5 months had hyperemia and congestion of the lungs, liver, and kidneys (Fabre et al. 1955, in ACGIH 1986, p. 151).

In humans, exposure to a concentration of 200 ppm cumene caused nasal irritation and central nervous system effects (RTECS 1987). Inhalation of high (not further specified) vapor concentrations may cause dizziness, incoordination, and unconsciousness (Clayton and Clayton 1981, pp. 3309–3310). Liquid cumene can cause erythema and irritation when in contact with the skin, and repeated contact can cause dermatitis (Gerarde 1959; Genium MSDS 1987, No. 395). Of 102 workers exposed to cumene vapors, 48 percent showed increased bilirubin concentrations, alteration of enzymatic activity, changed lipid metabolism, altered liver and hepatobiliary function, and dyskinesia (Putalova 1979).

Based on this evidence in humans and animals, OSHA preliminarily concludes that cumene causes primary irritation and central nervous system depression in exposed workers. The Agency believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects, which constitute material impairments of health. OSHA therefore believes that establishing a 50-ppm 8-hour TWA limit, with a skin notation, is necessary to significantly reduce these risks among agricultural workers. Promulgation of this limit will also make OSHA's PEL for cumene consistent across all OSHA-regulated sectors.

CYCLOPENTANE

CAS: 287–92–3; Chemical Formula: $CH_2CH_2CH_2CH_2CH_2$

H.S. No. 1111

OSHA has no limit for cyclopentane in construction, shipyards, marine terminals, longshoring operations, or agriculture. For workplaces in all of these sectors, OSHA is proposing a PEL of 600 ppm as an 8-hour TWA, which is consistent with the 1987–1988 ACGIH TLV® for this substance. NIOSH has no REL for cyclopentane but concurs with the proposed limit (Ex. 8–47, Table N1). This is the limit recently established for this substance in general industry.

Cyclopentane is a mobile, colorless, and flammable liquid. This substance occurs as an impurity in technical grade hexane, and it finds industrial use in the cracking of aromatics (Clayton and Clayton 1981, p. 3226). Cyclopentane also is an ingredient in a wide variety of commercial products, including analgesics, insecticides, antitumor agents, sedatives, and other pharmaceuticals (Clayton and Clayton 1981, p. 3226). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The existing acute toxicity data in animals indicate that cyclopentane is a potent narcotic agent. Mice exposed to a 38-ppm concentration of cyclopentane exhibited loss of reflexes, and some exposed animals died (von Oettingen 1940). In experimental animals, there is no margin of safety between the minimal narcotic concentration and the lethal dose (von Oettingen 1940). Applied to the skin of guinea pigs, liquid cyclopentane causes slight redness and dryness (Brown and Bax 1971). Almost no data are available concerning the chronic effects of cyclopentane exposure.

Abbritti, Siracusa, Cianchetti, and co-workers (1976/Ex. 1–406) reported that petroleum solvents used in the Italian shoe industry contain up to 18 percent cyclopentane, and that workers exposed to these solvents have developed polyneuropathy. Oettel (1936/Ex. 1–921) reported that skin exposure to such solvents caused burning and skin blistering after 15 minutes of occluded contact. It has not been determined, however, whether the irritation was caused by cyclopentane or by cyclopentane and other substances, such as n-hexane, in the solvent.

Based on this evidence in humans and animals, OSHA is proposing a PEL of 600 ppm as an 8-hour TWA for cyclopentane in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that occupational exposure to cyclopentane in these industries poses a significant risk of irritation and narcosis to exposed workers and that the proposed limit is necessary to substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this

substance consistent across all regulated sectors.

ETHYL ALCOHOL

CAS: 64–17–5; Chemical Formula: CH_3CH_2OH

H.S. No. 2076

OSHA's permissible exposure limit for ethyl alcohol in general industry, construction, and maritime is 1000 ppm as an 8-hour TWA; there is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 1000 ppm for ethyl alcohol. NIOSH has no REL for this substance but concurs with the PEL being proposed (Ex. 8–47, Table N3A). OSHA is proposing a PEL of 1000 ppm as an 8-hour TWA for ethyl alcohol in agriculture. Promulgation of this limit will make the PEL for ethyl alcohol consistent across all OSHA-regulated sectors.

Ethyl alcohol is a clear, colorless, flammable liquid. It is a volatile liquid with a wine-like odor that is recognizable at a concentration of approximately 100 ppm (ACGIH 1986, p. 242.2; AIHA 1987, Table 5.1). Ethyl alcohol is used in alcoholic beverages, pharmaceuticals, perfumes, gasolines, antiseptics, synthetic rubber, paints, lacquers, explosives, antifreeze, cosmetics, and food flavorings. It is also used as a solvent, in organic synthesis, and in the manufacture of denatured alcohols. When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). Industrial ethyl alcohol contains denaturant ingredients to render it unfit or undesirable for human ingestion (HSDB 1989; Gosselin, Smith, and Hodge 1984, p. II–174; ACGIH 1986, p. 242.2).

Ethyl alcohol is an irritant of the eyes and mucous membranes in both animals and humans; exposure to high concentrations causes central nervous system depression. The oral LD_{50} in rats is 7060 mg/kg, the LC_{50} in the same species is 20,000 ppm for 10 hours, and the lowest lethal dose in rabbits by dermal application is 20 g/kg (RTECS 1989). Rats exposed to a 22,800-ppm concentration of ethyl alcohol for 8 hours became deeply anesthetized, and exposure to a 16,000-ppm concentration for 8 hours caused some deaths in these animals. Incoordination was observed in rats exposed to 10,750 ppm for 2 hours; however, exposure to the same level for only 30 minutes caused no observable effects (Smyth 1956; Loewy and von der Heide 1918, in Clayton and Clayton 1982, p. 4545). In studies conducted on rabbits, ethyl alcohol produced mild to moderate irritation when in contact with the eyes or skin of these animals (RTECS 1989). Administered to rats and mice by various routes (oral, inhalation, intraperitoneal, intravenous, intracerebral, intratesticular, intrauterine), ethyl alcohol causes fetotoxic, embryotoxic, teratogenic, and developmental effects in the offspring and reproductive effects in the parents (RTECS 1989).

Humans exposed to a 1000-ppm concentration of ethyl alcohol for an unspecified time exhibited mild signs of poisoning, while exposure to a 5000-ppm concentration for an unspecified time caused stupor and sleepiness (Browning 1956, in ACGIH 1986, p. 242.2). Transient irritation of the eyes, nose, and respiratory tract occurred in individuals exposed for brief periods to 5000 to 10,000 ppm concentrations of ethyl alcohol. Continuous coughing and lacrimation occur when humans are exposed to a 15,000-ppm concentration of ethyl alcohol; exposure to concentrations above 20,000 ppm were described as intolerable (Lester and Greenberg 1951; Rowe and McCollister, in Clayton and Clayton 1982, p. 4551).

Based on this evidence, OSHA is proposing a 1000-ppm 8-hour TWA limit to protect workers in agriculture from the significant risks of irritation and central nervous system depression potentially associated with exposure to ethyl alcohol. OSHA preliminarily concludes that this limit is necessary to substantially reduce a significant risk of material health impairment in exposed workers in this sector. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ETHYL BROMIDE

CAS: 74–96–4; Chemical Formula: C_2H_5Br

H.S. No. 1163

In construction, shipyards, marine terminals, and long-shoring operations, OSHA currently has an 8-hour TWA limit of 200 ppm for ethyl bromide. There is no PEL in agriculture. The 1987–1988 ACGIH TLV®–TWA was 200 ppm, and the TLV®–STEL was 250 ppm. NIOSH has no REL for ethyl bromide. In construction and maritime, OSHA is retaining the 8-hour TWA PEL for ethyl bromide of 200 ppm and is proposing to add a 15-minute STEL of 250 ppm; the Agency is also proposing to extend both the 8-hour TWA and 15-minute STEL to agriculture. These are the limits recently established for these substances in general industry.

Ethyl bromide is a colorless, highly volatile, flammable liquid with an ether-like odor; it becomes yellow when exposed to light and air. Ethyl bromide was formerly used as an anesthetic agent and is currently used as a grain and fruit fumigant, a chemical intermediate, a refrigerant, and an ethylating agent, particularly for gasoline (HSDB 1986). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In addition to being a narcotic, ethyl bromide is an irritant of the respiratory system and a liver and kidney toxin. The oral LD_{50} in rats is 1350 mg/kg, and the lowest lethal concentration in rats is 26,980 ppm for 1 hour (RTECS 1990). When liquid ethyl bromide is applied to the skin of mice, it causes necrosis; instilled into rabbit eyes, it produces irritation (AIHA 1978). Skin absorption in toxic amounts is apparently possible, but no acute toxicity data exist for this route of exposure (Clayton and Clayton 1981, p. 3484). Prolonged exposure (6 months) to a 540-ppm concentration of ethyl bromide for 4 hours/day caused changes in liver function and liver damage that was visible at autopsy (Karimullina and Gizatullina, in Clayton and Clayton 1982, p. 3484). A National Toxicology Program carcinogenicity bioassay in rats and mice exposed by inhalation to 100 ppm (rats) or 200 ppm (mice) ethyl bromide for 6 hours/day for 2 years showed some evidence of carcinogenicity in rats (brain tumors) and clear evidence of carcinogenicity in female mice (uterine tumors) (NTP 1989).

Volunteers exposed to ethyl bromide concentrations of 6500 ppm for 5 minutes reported experiencing vertigo, headaches, and mild eye irritation (Sayers, Yant, Thomas, Berger 1929). Acute overexposure causes nervous system signs and symptoms that include redness of the face, dilation of the pupils, increased heart rate, cyanosis, and labored breathing. Several human deaths have been reported from the use of ethyl bromide as a general anesthetic (von Oettingen 1955/Ex. 1–876). These deaths were caused either by respiratory or cardiac arrest at the time the anesthetic was administered or by delayed effects on the heart, liver, or kidneys (von Oettingen 1955/Ex. 1–876).

In construction and maritime, OSHA is retaining its PEL of 200 ppm as an 8-hour TWA and is proposing to add a 15-minute STEL of 250 ppm for ethyl bromide; OSHA is also proposing to extend both limits to agriculture. The Agency preliminarily concludes that both limits are necessary to reduce the significant risks of narcosis, kidney and liver damage, and respiratory irritation that are potentially associated with occupational exposure to elevated levels

of ethyl bromide in these sectors. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

ETHYL CHLORIDE

CAS: 75–00–3; Chemical Formula: CH_3CH_2Cl

H.S. No. 2078

OSHA's PEL for ethyl chloride in general industry, construction, and maritime is 1000 ppm as an 8-hour TWA; there is no PEL in agriculture. The ACGIH has a TLV®–TWA for ethyl chloride of 1000 ppm; NIOSH considers this substance a carcinogen and recommends that it be handled with caution in the workplace. OSHA is proposing an 8-hour TWA PEL of 1000 ppm for ethyl chloride in agriculture. Promulgation of this limit will make the PEL for ethyl chloride consistent across all OSHA-regulated sectors.

Ethyl chloride is a colorless, flammable gas with an ethereal, slightly pungent odor. It is used as an anesthetic, a chemical intermediate, and sometimes as a refrigerant (Clayton and Clayton 1981, p. 3480; Braker and Mossman 1980, p. 306).

Ethyl chloride causes central nervous system depression at high concentrations in both animals and humans; it is also a mucous membrane irritant and a liver and kidney toxin in laboratory animals. Ethyl chloride also can be absorbed through the skin and mucous membranes (Parmeggiani 1983, p. 1078; AIHA 1978). The LC_{50} for rats is 160 g/m^3 for 2 hours (RTECS 1987). Exposure to a 23- or 24-percent concentration of ethyl chloride (230,000 or 240,000 ppm) caused guinea pigs to lose consciousness in 5 to 10 minutes; some of the animals died from this exposure. However, all guinea pigs survived a 30-minute exposure to a 9.1-percent (91,000 ppm) concentration of ethyl chloride, although histopathological changes were noted in the lungs, liver, and kidneys of these animals at autopsy (Sayers, Yant, and Thomas 1929). In guinea pigs exposed to a 40,000-ppm concentration of ethyl chloride, incoordination was seen after 3 minutes; after 40 minutes of exposure, these animals showed signs of eye irritation and were unable to stand. Although all animals survived 4.5 hours of exposure to this concentration, death occurred in some animals after 9 hours. At autopsy, pathological changes were seen in the liver, lungs, and kidneys (Sayers, Yant, and Thomas 1929). Animals exposed for prolonged periods to ethyl chloride concentrations that cause narcosis demonstrate a fall in blood pressure, irregularity in breathing, and damage to the liver, heart, and lungs (von Oettingen 1955, 1964). A recent study involving mice exposed continuously for 11 days to 250, 1250, or 5000 ppm ethyl chloride showed that the only treatment-related effects in these animals were an increase in relative liver weight and size and a slight increase in hepatocellular vacuolization (Landry, Johnson, Phillips, Weiss 1989).

In humans, exposure to a 13,600-ppm concentration of ethyl chloride caused signs of mild intoxication after 17 minutes; at a 25,000-ppm concentration, incoordination developed. At a concentration of 33,600 ppm, exposed individuals became intoxicated within 30 seconds of exposure and lost coordination after 5 minutes (Sayers, Yant, Thomas, and Berger 1929). Liquefied ethyl chloride spilled on the skin or splashed into the eyes may cause frostbite (AIHA 1978). Two persons are reported to have developed acute allergic eczematous dermatitis after ethyl chloride was sprayed on their skin in an allergy test (van Ketel 1976).

Based on this evidence, OSHA preliminarily concludes that workers in agriculture who are potentially exposed to ethyl chloride at the levels permitted by the absence of a limit are at significant risk of experiencing narcosis and other exposure-related effects. The Agency believes that establishing an 8-hour TWA PEL of 1000 ppm will substantially reduce the risk of these material impairments of health. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

GASOLINE

CAS: 8006–61–9; Chemical Formula: None

H.S. No. 1185

In construction, maritime, and agriculture, OSHA has no PEL for gasoline. The 1987–1988 ACGIH TLV®s for this substance are 300 ppm as an 8-hour TWA and 500 ppm as a 15-minute STEL. NIOSH has no REL for gasoline. OSHA is proposing an 8-hour TWA PEL of 300 ppm and a 15-minute STEL of 500 ppm for this substance in these sectors. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

Gasoline is a clear, flammable, volatile liquid with a characteristic odor; it is used as an engine fuel. Gasoline is a complex mixture of at least 250 hydrocarbons (ACGIH 1986, p. 283; Page and Mehlman 1989, p. 869).

In addition to narcosis, exposure to gasoline has been associated with irritation of the eyes and upper respiratory tract, liver and kidney damage, and, in animals, cancer of the liver and kidney (Page and Mehlman 1989). The LC_{50} in rats is 300 g/m^3 for 5 minutes (RTECS 1990). Splashed into the eyes of rabbits, gasoline causes transient corneal injury and blepharospasm (Grant 1986, p. 714). A two-year carcinogenicity bioassay (inhalation) in mice and rats revealed that exposure of these animals to concentrations of gasoline vapors caused increased incidences of renal tumors in male rats and of liver tumors in female mice (Page and Mehlman 1989; EPA 1987).

Volunteers exposed to vapor concentrations of 500 to 900 ppm for 1 hour became dizzy and, at 900 ppm, five of six volunteers reported experiencing eye irritation (Drinker, Yaglou, and Warren 1943). At the 160 to 270 ppm level, these volunteers experienced eye and upper respiratory tract irritation after 8 hours of exposure (Drinker, Yaglou, and Warren 1943). A later study by Davis, Schafer, and Bell confirmed the eye irritation effects of exposure to gasoline vapor from three different unleaded gasoline samples (Davis, Schafer, and Bell 1960). Results of epidemiologic studies in humans exposed to gasoline vapors at service stations, refineries, and distribution terminals have generally been inconclusive; most studies have failed to demonstrate an increase in cancer risk in these populations (EPA 1987; Page and Mehlman 1989).

OSHA is proposing an 8-hour TWA PEL of 300 ppm and a 15-minute STEL of 500 ppm for this substance in these sectors. These are the final limits recently established for gasoline in general industry.

However, as OSHA indicated in its brief submitted to the U.S. Court of Appeals for the Eleventh Circuit in the pending case *AFL* v. *OSHA*, Nos. 89–7185, et al., OSHA also notes ACGIH's finding that eye and throat irritation may occur in workers exposed at concentrations of 160 to 270 ppm (54 FR 2424/2).

After the close of the record of the general industry rulemaking, the American Petroleum Institute (API) submitted to OSHA a journal article published by McDermott and Killiany (1978) stating that the vapor that produced eye and throat irritation in the range of 160 to 270 ppm was not of the same hydrocarbon content as the vapor typically encountered in occupational exposure scenarios. However, this same paper recommends a TLV® of 300 ppm based on the use of the mixtures equation to calculate the TLV®. However, if benzene's contribution to the toxicity of gasoline is considered,

McDermott and Killiany (1978), the resulting TLV® would be approximately 100 ppm (using a 1 ppm benzene TLV®).

In its brief, OSHA stated "that, because eye and throat irritation may well constitute a material impairment of health, the evidence of gasoline's irritation effects at levels below 300 ppm warrants further study in the next PEL update." OSHA will consider whether the evidence does or does not support a lower PEL in the next PEL update and that decision will cover all segments of industry. OSHA does not intend to resolve that issue in this rulemaking because the Agency's priority in this rulemaking is to extend the updated general industry PELs to all other industry segments. OSHA believes that attempting to résolve the irritation issue and complex issues in this rulemaking would make this rulemaking difficult to complete in a timely fashion.

HEPTANE

CAS: 142–82–5; Chemical Formula: $CH_3(CH_2)_5CH_3$

H.S. No. 1194

The current OSHA limit for heptane in construction, shipyards, marine terminals, and longshoring operations is 500 ppm as an 8-hour TWA. There is no PEL in agriculture. The 1987–1988 ACGIH TLV®s for heptane are 400 ppm as an 8-hour TWA and 500 ppm as a 15-minute STEL. The NIOSH REL for heptane is 85 ppm as a 10-hour TWA or 440 ppm as a 15-minute limit. OSHA is proposing an 8-hour TWA PEL of 400 ppm and a 15-minute STEL of 500 ppm. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

Heptane is a clear, flammable liquid that is highly volatile. This substance is used as a solvent, a chemical intermediate, a standard for octane rating determinations, and a research chemical (HSDB 1986).

Heptane is a central nervous system depressant in both humans and animals. The intravenous LD_{50} in mice is 222 mg/kg (RTECS 1990). Three of four mice died after a 3-to-4 minute exposure to a 48,000-ppm concentration of n-heptane (Proctor, Hughes, and Fischman 1988, p. 269). Rats were given 1 ml/kg heptane daily for 2 days or daily for 7 days or twice weekly for 45 days, after which their livers were homogenized and assayed. The function of specific liver enzyme systems in the livers of rats exposed for 7 or 45 days was significantly impaired (Goel, Rao, and Pandya 1982).

The American Industrial Hygiene Association (1959) reports that exposure to a 1000-ppm concentration of heptane for 6 minutes caused slight dizziness in human volunteers; exposure to higher concentrations (5000 ppm) caused vertigo, in coordination, inappropriate behavior, loss of appetite, and nausea. When heptane is applied to the skin, it causes immediate irritation; blisters occurred in the affected area 5 hours after contact (NIOSH Criteria Document 1977).

As discussed below in connection with pentane and the hexane isomers, the NIOSH REL is the same for all of the C_5–C_8 alkanes (i.e., 350 mg/m³ (85 ppm) as a 10-hour TWA and 1800 mg/m³ (440 ppm) as a 15-minute ceiling). These RELs are based on NIOSH's belief that all C_5–C_8 alkanes possess a potential neurotoxic capability similar to that of n-hexane. OSHA disagrees with NIOSH on this point, believing instead that the neurotoxicity caused by exposure to n-hexane results from the action of a unique metabolite, 2,5-hexanedione. In the prior rulemaking, the majority of record commenters agreed with OSHA that n-hexane is uniquely neurotoxic (Exs. 3–593, 3–896, and 3–1246). A recent study (Perebelli, Brugnone, Coches, De Rosa, and Bartolucci (1986) confirms that, both in rats and occupationally exposed workers, the principal metabolites are 2-heptanol, 3-heptanol, 2-heptanone, 4-heptanone, and 2,5-heptanedione.

The Agency is aware that heptane is more acutely toxic than hexane and concludes preliminarily that it is appropriate to reduce the limit for heptane to a level below the level being proposed for the hexane isomers (i.e., 500 ppm as an 8-hour TWA and 1000 ppm as a 15-minute STEL). Therefore, in construction, maritime, and agriculture, OSHA is proposing for heptane a 400-ppm PEL as an 8-hour TWA and 500-ppm as a 15-minute STEL. The Agency believes that the proposed TWA and STEL will substantially reduce the significant occupational risks of heptane-related narcosis in construction, maritime, and agricultural workplaces. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

HEXANE ISOMERS (OTHER THAN N-HEXANE)

CAS: None; Chemical Formula: $(CH_3)_3C_3H_5$; $n(CH_3)_4C_2H_2$

H.S. No. 1201

OSHA has no limit in construction, maritime, or agriculture for the hexane isomers. The 1987–1988 ACGIH TLVs® for the hexane isomers are 500 ppm as an 8-hour TWA and 1000 ppm as a 15-minute STEL. NIOSH has a recommended TWA limit for these isomers of 100 ppm, supplemented by a 510-ppm 15-minute ceiling. OSHA is proposing an 8-hour TWA PEL of 500 ppm and a 15-minute STEL of 1000 ppm for the hexane isomers (other than n-hexane) in construction, maritime, and agriculture. These are the limits recently established for these substances in general industry.

The hexanes are clear, highly volatile liquids that have a mild gasoline-like odor. Their principal uses are in solvents for oils, glues, paints, and coatings and as constituents of rubber solvents and petroleum ether. The hexanes also find use as chemical intermediates and as gasoline additives (ACGIH 1986, p. 107).

Mice exposed to a 30,000-ppm concentration of hexane for 30 to 60 minutes became narcotized, and death occurred when the concentration was increased to 35,000 or 40,000 ppm (Flury and Zernik 1931; Swann et al. 1974).

These isomers are known to be irritants of the eyes, skin, and respiratory tract as well as central nervous system depressants. Prolonged or repeated contact of the skin with liquids containing hexane produces redness, swelling, and dermatitis (Genium MSDS 1989). A study by Drinker, Yaglou, and Warren (1943/Ex. 1–730) shows that humans exposed to 1400 to 1500 ppm of hexane experienced nausea and headache. Patty and Yant (1929) found that a 10-minute exposure to a 5000-ppm concentration caused giddiness and dizziness in exposed subjects. A study by Nelson, Enge, Ross et al. (1943/Ex. 1–66) showed no effects in unacclimated subjects exposed to hexane isomers in concentrations of 500 ppm, but narcotic effects have often been seen in subjects exposed at levels above 1000 ppm (Elkins 1959d, as cited in ACGIH 1986, Ex. 1–3, p. 307). The ACGIH based its limit primarily on the Nelson, Enge, Ross et al. (1943/Ex. 1–66) study.

NIOSH recommends limits for the hexane isomers of 100 ppm as a 10-hour TWA and 510 ppm as a 15-minute short-term limit. These recommendations are based on human and animal evidence showing that exposure to n-hexane, one of the hexane isomers, at concentrations below 500 ppm is associated with the development of polyneuropathy (Inoue, Takeuchi, Takeuchi et al. 1970/Ex. 1–75; Miyagaki 1967/Ex. 1–198). Thus, the NIOSH REL does not distinguish between n-hexane and the other hexane isomers; instead, NIOSH (Ex. 1–233) concluded that all of the C_5–C_8 alkanes are potential neuropathic agents and should have the same exposure limit.

In the prior rulemaking, OSHA disagreed with NIOSH's view; OSHA does not believe that all C_6–C_8 alkanes are potential neuropathic agents. Instead, OSHA is persuaded that 2,5-hexanedione, a metabolite of n-hexane (but not of the other hexane isomers) is responsible for the unique neurotoxic properties of n-hexane. OSHA's view thus agrees with that of the ACGIH, which states that "it seems unlikely that all the hexanes would follow the same metabolic route in the body [as n-hexane], in view of the marked variations in structure of the molecule" (ACGIH 1986/Ex. 1–3, p. 307). The majority of commenters in OSHA's prior air contaminants rulemaking supported OSHA's conclusion in that rulemaking that n-hexane is uniquely toxic because of the presence of 2,5-hexanedione and that the other alkanes are not toxic in this way (Exs. 3–593, 3–896, and 3–1246).

Based on the evidence discussed above, OSHA is proposing PELs of 500 ppm (8-hour TWA) and 1000 ppm (15-minute STEL) for the hexane isomers in construction, maritime, and agriculture. OSHA preliminarily finds that workers exposed to the hexane isomers in these sectors are at significant risk of experiencing narcosis and of developing neuropathy at exposure levels above those proposed. The Agency believes that establishing the proposed limits will substantially reduce these occupational risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

ISOAMYL ALCOHOL (PRIMARY AND SECONDARY)

CAS: 123–51–3; Chemical Formula: $(CH_3)_2CHCH_2CH_2OH$—Primary; $(C_2H_5)_2CHOH$—Secondary

H.S. No. 1218

In construction, shipyards, marine terminals, and long-shoring operations, OSHA's limit for the isoamyl alcohols is 100 ppm as an 8-hour TWA. There is no PEL in agriculture. The 1987–1988 ACGIH limits are an 8-hour TLV®–TWA of 100 ppm and a 15-minute TLV®–STEL of 125 ppm for the primary and secondary isoamyl alcohols. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the proposed limits. In construction, maritime, and agriculture, OSHA is retaining the Agency's 8-hour TWA limit of 100 ppm, proposing to add a 125-ppm 15-minute STEL, and proposing to extend both limits to agriculture.

Isoamyl alcohol is a flavoring agent, a lubricating and hydraulic oil additive, and a chemical intermediate. This substance also finds wide use as a solvent for fats, oils, and alkaloids (HSDB 1986). Isoamyl alcohols are colorless liquids with alcohol-like odors.

In rats, the oral LD_{50} for the primary isoamyl alcohol is 1300 mg/kg; the dermal LD_{50} in rabbits is 3212 mg/kg (RTECS 1990). Applied to the skin or eyes of rabbits, isoamyl alcohol causes minimal to severe irritation (Rowe and McCollister, in Clayton and Clayton 1982, pp. 4594–4599). Rats exposed to a 2000-ppm concentration for 8 hours survived, and rabbits administered 0.7 g/kg isoamyl alcohol orally developed stupor and lost control of voluntary movement (Munch 1972). By oral and subcutaneous administration, isoamyl alcohol caused malignant tumors of the liver and blood in rats (RTECS 1990; Gibel, Lohs, and Wildner 1975).

Smyth (1956/Ex. 1–759) reported that the principal effect of inhalation exposure to this substance in humans is narcosis. Nelson, Enge, Ross, and co-workers (1943/Ex. 1–66) reported that unacclimatized human volunteers reported upper respiratory tract irritation after brief (3- to 5-minute) exposures to an isoamyl alcohol concentration of 100 ppm and objectionable eye and mucous membrane irritation after short-term exposures to 150 ppm.

Based on this evidence, OSHA is retaining the 8-hour TWA PEL of 100 ppm and proposing to add a 15-minute STEL of 125 ppm for the isoamyl alcohols (primary and secondary) in construction and maritime; the Agency is also proposing to extend both limits to agriculture. OSHA preliminarily concludes that these limits are necessary to reduce the significant risk of eye irritation and narcotic effects associated with exposure to these substances. In addition, promulgation of these limits will make OSHA's PELs for these substances consistent across all regulated sectors.

ISOPHORONE

CAS: 78–59–1; Chemical Formula: $C_9H_{14}O$

H.S. No. 1221

In construction, shipyards, marine terminals, and long-shoring, the Agency's current limit for isophorone is 25 ppm as an 8-hour TWA. There is no PEL in agriculture. The ACGIH has established a 5-ppm TLV® as a ceiling limit, and NIOSH recommends a workplace standard of 4 ppm as a 10-hour TWA and concurs (Ex. 8–47, Table N1) with OSHA's proposed limit which is 4 ppm as an 8-hour TWA. This is the limit recently established for this substance in general industry.

Isophorone is a colorless liquid at room temperature, and it has a camphor-like odor. This substance is used as a solvent for lacquers, plastics, oils, fats, gums, resin systems, nitrocellulose, and vinyl-resin copolymers and as a chemical intermediate. It also finds use in the manufacture of pesticides (HSDB 1986).

Studies in animals and with human volunteers indicate that exposures to high concentrations of isophorone cause narcotic, nephrotoxic, and irritant effects. The oral LD_{50} in rats is 2330 mg/kg, and the LC_{50} in the same species is 1840 ppm for 4 hours (RTECS 1990). The dermal LD_{50} in rabbits is 1500 mg/kg (RTECS 1990). Applied to the skin of rabbits, isophorone causes mild irritation; in the eyes, however, this substance causes severe irritation (RTECS 1990). A paper by Smyth, Seaton, and Fischer (1942/Ex. 1–378) reported that guinea pigs and rats exposed to 550 ppm isophorone for 6 weeks exhibited degenerative changes in the kidneys and liver at autopsy. At an exposure level of 25 ppm, no adverse effects were noted, but at 50 ppm, the liver of one animal and the kidneys of four others were damaged. The entire group of 20 animals exposed to a 50-ppm concentration survived, but two of 16 animals died after this concentration was raised to 100 ppm (Smyth, Seaton, and Fischer 1942/Ex. 1–378). A 2-year bioassay for carcinogenicity involving the administration by gavage of 250 or 500 mg/kg isophorone showed a dose-related and statistically significant increase in kidney and preputial gland tumors in male rats and an increase in the incidence of liver tumors in male mice in the high-dose group (Bucher, Huff, and Kluwe 1986).

Human volunteers exposed for a few minutes to isophorone vapor at concentrations between 40 and 400 ppm experienced eye, nose, and throat irritation; several subjects exposed at the 200-ppm level reported headache, nausea, faintness, dizziness, and a feeling of suffocation (Smyth and Seaton 1940a/Ex. 1–377). Silverman, Schulte, and First (1946/Ex. 1–142) reported that volunteers exposed to a 25-ppm concentration of isophorone, the current OSHA PEL in construction and maritime, complained of irritation of the eyes, nose, and throat. Workers exposed for a 1-month period to levels of 5 to 8 ppm isophorone demonstrated fatigue and malaise; however, when the workplace level was reduced to between 1 and 4 ppm, there were no complaints of adverse effects (NIOSH Criteria Document 1978).

Based on this evidence in humans and animals, OSHA is proposing in construction and maritime to reduce its 8-hour TWA PEL of 25 ppm to an 8-hour

TWA PEL of 4 ppm, and to extend this limit to agriculture, to protect workers in these sectors against the significant risk of fatigue, nausea, and headaches that have been demonstrated to occur at isophorone levels of between 5 and 8 ppm. The Agency preliminarily concludes that the proposed limit will substantially reduce these occupational risks for workers in these sectors. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

METHYL CHLORIDE

CAS: 74–87–3; CHEMICAL FORMULA: CH_3Cl

H.S. No. 1254

In construction, marine terminals, shipyards, and long-shoring, OSHA's current limit for methyl chloride is 100 ppm as a ceiling. There is no PEL in agriculture. The ACGIH has a 50-ppm 8-hour TLV®–TWA limit and a 100-ppm TLV®–STEL for this substance, and NIOSH recommends the lowest feasible limit because it considers methyl chloride a potential occupational carcinogen. The PELs being proposed by OSHA for construction, maritime, and agricultural workplaces are 50 ppm as an 8-hour TWA and 100 ppm as a 15-minute STEL. These are the limits recently established for these substances in general industry.

Methyl chloride (also called chloromethane) is a colorless, sweet-smelling gas. This substance is used as a methylating agent in the manufacture of a wide variety of substances and as a blowing agent in polystyrene foam production (ACGIH 1986, p. 380). Formerly, methyl chloride found wide use as a refrigerant (Grant 1986, p. 612). Methyl chloride also is used as an anti-knock fuel additive and an ingredient of fungicides and pesticides (Proctor, Hughes, and Fischman 1988, p. 325). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

There is considerable evidence in animals demonstrating that exposure to methyl chloride produces narcosis and other central nervous system effects and causes kidney and liver damage, and that this substance is a teratogen, reproductive toxin, and carcinogen. The LC_{50} in rats is 5300 mg/m^3 for 4 hours (RTECS 1990). Acutely poisoned animals showed the following signs of narcosis: apathy, staggering gait, drowsiness, hind-limb paralysis, spasticity, and convulsions (IARC 1986, Vol. 41, p. 170). At autopsy, kidney damage and hemorrhage of the lung were seen (IARC 1986, Vol. 41, p. 170). Rats exposed to a 3500-ppm concentration for 6 hours/day for as many as 12 days developed diarrhea, forelimb incoordination, hind-limb paralysis, and convulsions (Morgan et al. 1982). Mice exposed continuously or intermittently over the same period to 400 ppm showed brain lesions at autopsy (Landry, Quast, Gushow, and Mattsson 1985). Adverse kidney and blood effects were seen in mice exposed for up to 9 days to a 2400-ppm concentration of methyl chloride (Landry et al. 1985). By inhalation, methyl chloride has caused fetal growth retardation and impaired male reproductive capacity in rats and heart defects in the offspring of exposed mice (IARC 1986, p. 176; RTECS 1990). Male mice repeatedly exposed to a 1000-ppm concentration of methyl chloride over a 2-year period had a statistically significant increase in the incidence of tumors of the kidney (NIOSH CIB 1984).

Methyl chloride has caused death in several cases of human poisoning; these fatalities have occurred both as a result of high acute exposures and of long-term exposures to lower concentrations (Scharnweber, Spears, and Cowles 1974/Ex. 1–664). Victims of methyl chloride poisoning display the following signs of narcosis before death: staggering gait, slurred speech, drowsiness, weakness, and paralysis; convulsions, cyanosis, and coma also occur (Scharnweber, Spears, and Cowles 1974/Ex. 1–664; Spevak, Nadj, and Felle 1976; Hansen, Weaver, and Venable 1953). Survivors of near-lethal exposures may evidence mild neurologic or psychiatric effects for as long as 13 years after the incident (Gudmundsson 1977).

The Dow Chemical Company (as cited in ACGIH 1986/Ex. 1–3, p. 380) studied the methyl chloride exposures of employees in 54 job classifications over a 4-month period. Exposures ranged from 5 to 78 ppm methyl chloride (8-hour TWAs), averaged 30 ppm over the work shift, and occasionally included peaks as high as 440 ppm. Medical examination of these workers revealed no detectable effects of methyl chloride exposure. However, average 8-hour exposures in the range of 195 to 475 ppm caused symptoms of weakness, drowsiness, staggering gait, thickness of the tongue, and memory lapses in some of the exposed employees (Dow Chemical Company, as cited in ACGIH 1986/Ex. 1–3, p. 380). Repko and co-workers (1976/Ex. 1–1165) found that workers exposed to concentrations of methyl chloride ranging from 7.4 to 70 ppm but averaging 33.6 ppm displayed a significant performance decrement, and that exposures below 100 ppm produced significant but transitory changes in functional capacity.

In the prior air contaminants rulemaking, NIOSH stated that it believes that methyl chloride is an appropriate substance for a Section 6(b) rulemaking because, according to NIOSH criteria, methyl chloride is a potential occupational carcinogen (Ex. 8–47; Tr. 3, pp. 97–98). However, OSHA notes the inconclusive nature of the evidence of methyl chloride's carcinogenicity and the fact that the International Agency for Research on Cancer (IARC) has concluded that this evidence is inadequate in both humans and animals (IARC 1986, p. 177). The only study in humans, an assessment of cancer risk in a small group of butyl rubber workers, was, in IARC's words, "uninformative with regard to the carcinogenic hazard of this chemical" (IARC 1986, p. 176).

Results of the two studies evaluating neurological effects are less than definitive as to LOEL for these effects. In the Dow study, medical examination of workers exposed to 5 to 78 ppm TWAs revealed no effects, although some workers exposed to 195 to 475 ppm (8-hour TWAs) complained of symptoms such as weakness, drowsiness, and memory loss. Repko reported significant performance decrement in workers exposed to concentrations between 7 and 70 ppm.

OSHA will continue to monitor the literature and determine whether to reevaluate the level for methyl chloride in the first PEL update. At that time, a level for all industry sectors will be considered at the same time. Currently, OSHA is proposing the same level in construction, maritime, and agriculture that was recently established for general industry.

Accordingly, OSHA is proposing to establish an 8-hour TWA PEL of 50 ppm and a 15-minute STEL of 100 ppm for methyl chloride in construction, maritime, and agriculture. The Agency preliminarily concludes that these limits will substantially reduce the significant risk of narcotic effects, including functional impairment, performance decrements, headaches, dizziness, slurred speech, and staggering gait that have been associated with occupational exposure to this substance. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

METHYL CHLOROFORM (1,1,1-TRICHLOROETHANE)

CAS: 71–55–6; Chemical Formula: CH_3CCl_3

H.S. No. 1255

OSHA currently has an 8-hour TWA limit of 350 ppm for methyl chloroform in construction, shipyards, marine terminals, and longshoring operations. There is no PEL in agriculture. The 1987–1988 ACGIH TLV®–TWA was 350 ppm and the TLV®–STEL was 450 ppm for this substance; NIOSH recommends a 15-minute ceiling limit of 350 ppm. In construction and maritime, the Agency is retaining its 8-hour TWA limit and is proposing to add a STEL of 450 ppm; OSHA is also proposing to extend both limits to agriculture. NIOSH concurs that these limits are appropriate but would express them as ceilings rather than as TWAs (Ex. 8–47, Table N7). These are the limits recently established for these substances in general industry.

Methyl chloroform, which is also called trichloroethane, is a clear, nonflammable liquid with a sweetish, chloroform-like odor (Clayton and Clayton 1982, p. 3509; ACGIH 1986, p. 382). This substance is a widely used metal degreaser (HSDB 1987). It is also used as an insecticidal fumigant and as a solvent, dry cleaning agent, and chemical intermediate (Hayes 1982, p. 152; HSDB 1987). Methyl chloroform was formerly used in agriculture to degreen citrus fruits and to fumigate strawberries (HSDB 1987). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The primary health effects associated with exposure to methyl chloroform are narcosis, irritation, and liver damage. The oral LD_{50} values range from 5.6 to 11.2 g/kg for rats, mice, rabbits, and guinea pigs (RTECS 1990). The LC_{50} in rats is 18,000 ppm for 4 hours (RTECS 1990). Skin absorption is apparently not a significant route of exposure: The dermal LD_{50} in rabbits is greater than 16 g/kg (Torkelson, Oyen, McCollister, and Rowe 1958/Ex. 1–768). Applied to the skin or eyes, however, methyl chloroform causes mild-to-moderate irritation (RTECS 1990). Rats exposed for 1 hour to a methyl chloroform concentration of 10,000 ppm lost their coordination and lapsed into semiconsciousness (Hayes 1982, p. 152). Guinea pigs exposed to 1000 ppm for 3 hours/day, 5 days/week for 3 months showed, at autopsy, lung inflammation and fatty infiltration of the liver (Hayes 1982, p. 152). Repeated exposures to 500 ppm of methyl chloroform vapor for 7 hours/day, 5 days/week for 6 months caused no apparent adverse effects in guinea pigs, rabbits, or monkeys (Torkelson, Oyen, McCollister, and Rowe 1958/Ex. 1–768). Tests in rats and mice for teratogenicity and carcinogenicity have demonstrated negative results (Schwetz, Leong, and Gehring 1975/Ex. 1–757; NIOSH 1976m; Weisberger 1977/Ex. 1–694); IARC 1979, Vol. 20, p. 515–531).

In humans, narcotic effects begin to occur at methyl chloroform concentrations of approximately 350 ppm (Mackay et al. 1987). For example, reaction times, manual dexterity, and perceptual speed were adversely affected in volunteers exposed to 350 ppm (Gamberale and Hultengren 1973). Deaths from anesthesia and/or cardiac sensitization have been noted in employees working in confined areas (NIOSH 1976m).

Three recent studies (McLeod et al. 1987, Karlsson et al. 1987, and Mackay et al. 1987) suggest, respectively, that methyl chloroform causes chronic cardiac toxicity on long-term exposure, may have toxic effects on brain cells, and may cause behavioral changes after 3.5-hour exposures to concentrations of approximately 350 ppm. Kramer and co-workers (1978/Ex. 1–515) conducted an epidemiological study of men and women exposed for periods ranging from several months to 6 years to methyl chloroform at levels that occasionally exceeded 200 ppm; when compared with matched-pair controls, no adverse exposure-related effects were found in the exposed individuals (Kramer, Ott, Fulkerson et al. 1978/Ex. 1–515).

Based on this evidence in humans and animals, OSHA is proposing in construction and maritime to retain its PEL of 350 ppm as an 8-hour TWA and to add a STEL of 450 ppm for methyl chloroform; OSHA is also proposing to extend both limits to agriculture. The Agency preliminarily concludes that these limits will protect workers in these sectors against the significant risk of narcotic, irritant, and hepatotoxic effects, which together constitute material health impairments that are potentially associated with exposure to methyl chloride. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

NAPHTHA (Coal Tar)

CAS: 8030–30–6; Chemical Formula: H.S. No. 2113

In general industry, construction, and maritime, OSHA's PEL for coal tar naphtha is 100 ppm. There is no PEL in agriculture. Neither ACGIH nor NIOSH has a limit for this substance. OSHA is proposing an 8-hour TWA PEL of 100 ppm for coal tar naphtha in agriculture. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

Coal tar naphtha is a mixture of inconstant nature prepared from the distillation of coal; this substance contains xylenes, ethyl benzene, cumene, and occasionally, toluene. Coal tar naphtha, also called coal tar distillate, aromatic petroleum naphtha, coal tar light oil, and coal tar oil, is a light oil fraction distilling to 200 °C (Grayson 1985, p. 1154). Coal tar naphtha is used for waterproofing, in paints, pipe coatings, on roads, in sealants, in roofing operations, and in pesticides.

Coal tar naphtha is toxic by inhalation and skin absorption. Exposure causes moderate eye and upper respiratory tract irritation and narcosis (Cralley and Cralley 1985, p. 176). Exposure to 1500 ppm for 8 hours was lethal to rats (Carpenter, Geary, Meyers et al. 1977). If benzene is present in the naphtha mixture in substantial quantities, exposure may lead to bone marrow effects (Clayton and Clayton 1981, p. 3392). Rodents, canines, and primates exposed to high (not further specified) concentrations of coal tar naphtha on subchronic experimental regimens showed decreased weight gain, central nervous system depression, decreased white blood cell counts, and, in some animals, cataracts (Nau, Neal, and Thornton 1966, in Clayton and Clayton 1981, p. 3418; Carpenter, Geary, Meyers et al. 1977). Rats exposed to a 3200-ppm concentration for 2 months survived but showed damage to the liver and kidney at autopsy (NIOSH/OSHA 1981).

Humans can detect the odor of coal tar naphtha at an airborne concentration of 0.07 ppm, and some exposed individuals experience the onset of sensory irritation at 25 ppm (Carpenter, Geary, Meyers et al. 1977). Acute exposure to this substance (concentrations no specified) leads to eye, nose, and throat irritation, and to narcotic signs and symptoms, including vertigo, nausea, dyspnea, and, if benzene is present, neurotoxicity. Chronic exposure also causes central nervous system depression and, depending on the benzene content, hematopoietic system changes (Sundmeyer, in Clayton and Clayton 1981, p. 3392).

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 100 ppm for coal tar naphtha in agriculture. The Agency believes that this limit is necessary to reduce the significant risk of irritation, narcosis, and hematopoietic changes associated with exposure to coal tar naphtha. In addition, promulgation of this limit will make

OSHA's PEL for this substance consistent across all regulated sectors.

OCTANE

CAS: 111–65–9; Chemical Formula: $CH_3(CH_2)_6CH_3$

H.S. No. 1296

OSHA's limit for octane in construction, shipyards, marine terminals, and longshoring operations is 400 ppm as an 8-hour TWA. There is no PEL in agriculture. The 1987–1988 ACGIH TLV®s for this substance were 300-ppm as an 8-hour TWA and 375-ppm as a 15-minute STEL; NIOSH (1977a/Ex. 1–233) recommends a 75-ppm 10-hour TWA and a 385-ppm 15-minute ceiling limit. In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA PEL of 300 ppm and a 15-minute STEL of 375 ppm for octane. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

Octane is a colorless, flammable liquid that has 17 isomers and an odor like that of gasoline. Octanes occur naturally in natural gas and crude oil and are found industrially in some petroleum solvents and in gasoline. The n-octane isomer is used as a solvent and in organic synthesis, while the iso-octane isomer is a standard used to measure the anti-knock properties of gasolines (ACGIH 1986, p. 448; Proctor, Hughes, and Fischman 1988, p. 384).

Mice exposed to octane concentrations of 6600 to 13,700 ppm developed narcosis within 30 to 90 minutes (Fuhner 1921, as cited in ACGIH 1986/Ex. 1–3, p. 448). Mice exposed to a 16,000-ppm concentration of iso-octane suffered respiratory arrest, and exposure to approximately 7500 ppm of the n-isomer caused loss of the righting reflex in the same species (Swann, Kwon, Hogan, and Snellings 1974).

Flury and Zernik (1931h) believed the narcotic concentration of octane in humans to be 5000 ppm; Patty and Yant (1929) placed the narcotic concentration at 8000 ppm. The signs and symptoms of over-exposure to the vapors of octane include giddiness, vertigo, headache, and stupor; convulsions have also been reported (HSDB 1986). Repeated or prolonged skin contact causes dryness and cracking of the skin (New Jersey Fact Sheet 1987, p. 2).

As discussed above for the other C_6–C_8 alkanes, the limits recommended by NIOSH for octane (1977a/Ex. 1–233) are based on NIOSH's belief that all C_6–C_8 alkanes present a neurotoxic hazard similar to that of n-hexane. OSHA disagrees with this conclusion, finding instead (54 FR 2428) that the neurotoxic properties of n-hexane are unique among these substances and thus that a PEL lower than the one being proposed for octane is not warranted at this time.

Accordingly, OSHA is proposing limits for octane of 300 ppm as an 8-hour TWA and 375 ppm as a 15-minute STEL in the construction, maritime, and agriculture sectors. The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant risk of narcosis that is associated with octane exposures. OSHA believes that these limits will substantially reduce these significant risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

PENTANE

CAS: 109–66–0; Chemical Formula: C_5H_{12}

H.S. No. 1306

OSHA currently has an 8-hour TWA limit of 500 ppm for pentane in construction, shipyards, and marine terminals, and an 8-hour TWA of 1000 ppm in longshoring operations. There is no PEL in agriculture. The 1987–1988 ACGIH TLV®–TWA was 600 ppm and the TLV®–STEL was 750 ppm. NIOSH (1977a/Ex. 1–233; Ex. 8–47, Table N2) has recommended that workplace exposures to pentane not exceed 120 ppm as a 10-hour TWA and 610 ppm as a 15-minute ceiling limit. The Agency is proposing PELs of 600 ppm as an 8-hour TWA and 750 ppm as a 15-minute STEL in construction, maritime, and agriculture. This action will make OSHA's PELs for pentane consistent across all industry sectors and will provide protection against pentane's effects for workers in all OSHA-regulated sectors. The adoption of a 600 ppm PEL for all affected sectors is important (1) to maintain uniformity when health and feasibility data are equivocal, (2) to simplify enforcement, and (3) to reduce confusion about the limit among the public, employers, and employees.

Pentane, a colorless, flammable liquid with a gasoline-like odor, is usually encountered in volatile petroleum fractions, some of which are used as solvents. Pentane is used as a pesticide and is also an incidental additive in liquid grain fumigants. In addition, it finds use as a fuel, in dry cleaning, and in degreasing operations (HSDB 1986). Pure pentane is used as a blowing agent for plastics, in solvent extraction, and in ice manufacture. When used in accordance with the EPA label and in pesticidal applications, pentane is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act.

Exposure to pentane causes both narcosis and mucous membrane irritation in humans and animals. The lowest lethal concentration in mice is 325 g/m³ for 2 hours; at this concentration, behavioral signs and symptoms were apparent (RTECS 1990). The intravenous LD_{50} in mice is 446 mg/kg (RTECS 1990). Mice exposed to a 128,000 ppm concentration became deeply anesthetized, and one in four of these animals died (Swann, Kwon, Hogan et al. 1974). Respiratory irritation and mild narcosis occurred in animals of the same species at an exposure concentration of 32,000 to 64,000 ppm for 5 minutes (Swann, Kwon, Hogan et al. 1974). The reported lethal concentration in humans is 130,000 ppm (Flury and Zernik 1931j/Ex. 1–894; Swann, Kwon, and Hogan 1974/Ex. 1–124). According to Patty and Yant (1929), humans exposed for 10 minutes to 5000 ppm did not report experiencing any adverse symptoms.

A study by Gaultier, Rancurel, Piva, and Efthymioc (1973/Ex. 1–123) reports that five cases of polyneuropathy occurred among employees exposed to a solvent containing 80 percent pentane, 14 percent heptane, and 5 percent hexane. Based largely on these results, NIOSH (1977a/Ex. 1–233) recommended the same occupational limit for all C_5–C_8 alkanes as for the neuropathic agent n-hexane (350-mg/m³ TWA and 1800-mg/m³ 15-minute short-term limits; these limits approximately equal a 120-ppm 8-hour TWA and a 610-ppm 15-minute STEL for pentane). As discussed above in connection with the entries for heptane and octane, OSHA believes that n-hexane is uniquely neuropathic because it is metabolized to 2,5-hexanedione, and thus that all C_5–C_8 alkanes are not equally toxic. The Agency concluded in the prior rulemaking that a metabolite of n-hexane exhibits unique neurotoxic properties and thus that the Gaultier, Rancurel, Piva, and Efthymioc (1973/Ex. 1–123) study does not provide isomer-specific exposure data supporting limits at the levels of the NIOSH RELs.

Also in the prior rulemaking, one commenter (Ex. 3–898) objected to the proposed STEL for pentane on the grounds that the health evidence did not justify a STEL. OSHA responded to this commenter (54 FR 2428) by pointing out that a STEL is needed to protect workers from the significant neurotoxic effects potentially associated with the high short-term excursions possible in the absence of a STEL.

Based on this evidence and reasoning, the Agency is proposing to establish an 8-hour TWA of 600 ppm and a 15-minute

STEL of 750 ppm as the permissible exposure limits for pentane in construction, maritime, and agriculture. OSHA preliminarily concludes that these limits will protect exposed workers in these sectors from the narcosis associated with pentane exposure. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

2-PENTANONE (METHYL PROPYL KETONE)

CAS: 107-87-9; Chemical Formula: $CH_3COC_3H_7$

H.S. No. 1307

The current OSHA limit for 2-pentanone in construction, shipyards, marine terminals, and longshoring operations is 200 ppm as an 8-hour TWA. There is no PEL in agriculture. The 1987–1988 ACGIH TLV®–TWA was 200 ppm and the TLV®–STEL was 250 ppm; NIOSH (1978k) has recommended a 150-ppm limit as a 10-hour TWA. In construction and maritime, the Agency is proposing to retain the 200-ppm TWA PEL and to establish a 15-minute STEL of 250 ppm for 2-pentanone; in agriculture, OSHA is proposing both the 8-hour TWA and STEL.

2-Pentanone, also called methyl propyl ketone, is a clear, flammable liquid with a strong odor resembling that of acetone or ether. 2-Pentanone is used as a solvent and flavoring agent and in organic synthesis (ACGIH 1986, p. 408; Hawley's 1987, p. 780).

2-Pentanone has an oral LD_{50} in rats of 1600 mg/kg and an LC_{50} in the same species of 2000 ppm for 4 hours (RTECS 1990). The dermal LD_{50} for this substance in rabbits is 6500 mg/kg (RTECS 1990). Acutely poisoned animals display agitation, loss of balance, and staggering (Miller, Valaer, and Sayers 1940). Both the ACGIH- and NIOSH-recommended limits for 2-pentanone are based on the results of a study by Specht, Miller, Valaer, and Sayers (1940/Ex. 1–1179), which found that guinea pigs exhibited irritation and weakness on exposure to 2500 ppm and that exposure to 5000 ppm produced narcosis and coma. Based on this evidence, the authors concluded that 2-pentanone is considerably less toxic than methyl butyl ketone but is more toxic than methyl ethyl ketone, and, in addition, that 2-pentanone is likely to be more irritating than either methyl ethyl ketone or acetone. The ACGIH-recommended limits are thus based on a judgment that the 200-ppm TLV®–TWA and 250-ppm TLV®–STEL are low enough to prevent narcosis and irritation.

NIOSH (1978k) relied on the findings both of the Specht, Miller, Valaer, and Sayers (1940/Ex. 1–1179) study discussed above and of the Nelson, Enge, Rosa et al. study (1943/Ex. 1–66); these latter authors reported that volunteers complained of slight irritation on exposure to a 100-ppm concentration of methyl ethyl ketone. NIOSH reasoned that, because 2-pentanone had been found by Specht, Miller, Valaer, and Sayers (1940/Ex. 1–1179) to be at least as irritating as methyl ethyl ketone, a "slight reduction" in the limit was warranted for 2-pentanone (NIOSH 1978k). Therefore, NIOSH recommends a 150-ppm limit for 2-pentanone, and this recommendation was reiterated in the prior air contaminants rulemaking (Ex. 8–47, Table N2; Tr. 3–86).

However, OSHA believes that the combination of a 200-ppm TWA PEL and a 250-ppm STEL will ensure that concentrations of 2-pentanone in construction, maritime, and agricultural workplaces are maintained at levels that will prevent the occurrence of the adverse health effects associated with exposures to this chemical. Accordingly, the Agency is proposing to establish these limits to reduce the significant risk of narcosis that is associated with exposures to 2-pentanone at elevated short-term levels. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

STODDARD SOLVENT

CAS: 8052-41-3; Chemical Formula: C_9H_{20}

H.S. No. 1371

OSHA's current limit for Stoddard solvent in construction, shipyards, marine terminals, and longshoring operations is 200 ppm as an 8-hour TWA. There is no PEL in agriculture. The 1987–1988 ACGIH TLV®–TWA was 100 ppm, and NIOSH concurs that this limit is appropriate (Ex. 8–47, Table N1). OSHA is proposing to reduce the PEL for Stoddard solvent in construction and maritime to 100 ppm as an 8-hour TWA and to extend this limit to agriculture. This is the limit recently established for this substance in general industry.

Stoddard solvent is a refined petroleum solvent that is a mixture of straight- and branched-chain paraffins, naphthenes and aromatic hydrocarbons. This substance is widely used as a solvent and diluent in paints, coatings, and waxes and as a metal degreasing agent. It is also often found as an ingredient of herbicides (ACGIH, 1986, p. 537). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Stoddard solvent is an irritant of the eyes, skin, and mucous membranes and a narcotic in humans and animals. Inhalation of a 1400-ppm concentration of this substance for 8 hours was lethal to some rats and cats (Carpenter et al. 1975). Acutely poisoned animals showed signs of eye irritation, lost their coordination, and had a bloody discharge from the nose (Carpenter et al. 1975). Cats exposed to 1700 ppm for 2.5 to 7.5 hours convulsed before death (Carpenter et al. 1975). Rats exposed to a 330-ppm concentration of Stoddard solvent 6 hours/day for 65 days had tubular degeneration of the kidneys at autopsy (Carpenter et al. 1975).

Humans exposed to a 470-ppm concentration of Stoddard solvent for 15 minutes experienced eye irritation (RTECS 1990). In prolonged or repeated contact with the skin, this substance causes dermatitis (AIHA 1978). Inhalation of large (not further specified) concentrations causes nausea, vomiting, cough, and pulmonary irritation (Cohen and Maier 1974). Several studies suggest that chronic exposure to Stoddard solvent vapors for periods ranging from 3 months to 20 years may cause hepatic involvement, hypoplasia of the bone marrow, and aplastic anemia (Scott et al. 1959; Prager and Peters 1970).

The NIOSH RELs for Stoddard solvent are derived from NIOSH's recommended limits for all of the C_6–C_9 alkanes; NIOSH recommended the same limit for Stoddard solvent as for all C_6–C_9 alkanes both because of the lack of specific scientific data on Stoddard solvent's chronic effects and because of a report of polyneuropathy occurring among workers exposed to jet fuels containing mixtures of kerosene and gasoline. NIOSH reasoned that, although the C_9–C_{16} alkanes present in jet fuel may have been implicated, it was possible that the heavier hydrocarbon components may also have been responsible. Thus, the NIOSH RELs for Stoddard solvent reflect a concern that higher-molecular-weight hydrocarbons may be neuropathic. However, OSHA does not believe that there is evidence that the C_{10} and higher-molecular-weight hydrocarbons cause neuropathies, and NIOSH also now agrees, based on a reevaluation of the evidence, that a 100-ppm 8-hour TWA limit is appropriate for this substance (Ex. 8–47, Table N1).

OSHA is proposing to establish an 8-hour TWA PEL of 100 ppm in construction, maritime, and agriculture to reduce the significant risk of eye irritation, narcosis, polyneuropathy, and kidney damage that have been

demonstrated to occur in both humans and animals. The Agency believes that the proposed limit for Stoddard solvent is necessary to substantially reduce these risks to workers in these sectors. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

STYRENE

CAS: 100–42–5; Chemical Formula: $C_6H_5CH=CH_2$

H.S. No. 1372

OSHA's current permissible exposure limit for styrene in construction, shipyards, marine terminals, and longshoring operations is 100 ppm as a ceiling limit. There is no PEL in agriculture. In construction and maritime, OSHA is proposing to revise this limit to 50 ppm as an 8-hour TWA and to add a 15-minute STEL of 100 ppm, based on both the 1987–1988 ACGIH TLV®s and the NIOSH RELs, which are identical. OSHA is also proposing these limits in agriculture. NIOSH (Ex. 150, Table N1) concurs that the proposed limits are appropriate for styrene.

Styrene monomer is a colorless, oily liquid with an aromatic odor. It is used as a solvent for resins and synthetic rubber and as an intermediate in chemical synthesis. Styrene is also used as a flavoring agent and in the manufacture of polymerized synthetics (HSDB 1987; Proctor, Hughes, and Fischman 1988, p. 448).

Styrene is an irritant, a narcotic, and a neuropathic agent; some studies also show that animals exposed to styrene vapor develop tumors. The oral LD_{50} in rats is 5000 mg/kg, and the LC_{50} in the same species is 24 g/m³ for 4 hours (RTECS 1990). Rats and guinea pigs exposed to a 1300-ppm concentration of styrene for 8 hours/day, 5 days/week for 6 months experienced often-lethal pulmonary irritation (guinea pigs) and, at autopsy, showed increased kidney and liver weights (rats) (IARC 1979, Vol. 19, p. 239). Oral exposure to styrene has been shown to cause liver enzyme changes and other hepatic effects in rats and dogs (EPA 1987, pp. 6–7).

There is a considerable body of health-effects information in humans for styrene in the toxicological literature. Human volunteers exposed to an 800-ppm concentration of styrene for 4 hours experienced eye and throat irritation and also reported listlessness, drowsiness, and impaired balance (Carpenter et al. 1944). At a concentration of 376 ppm, five human volunteers experienced eye and respiratory tract irritation within 20 minutes and also demonstrated decrements in motor function (Stewart et al. 1968). Three subjects exposed to a 100-ppm concentration of styrene for 90 minutes had slower reaction times; on repeated exposure, sleepiness, fatigue, headache, difficulty in concentration, malaise, nasal irritation, and nausea occurred in another group of subjects (NIOSH 1983a, p. 150).

Effects attributable to central nervous system depression were seen in a 6-week study involving human subjects exposed to 20, 100, or 125 ppm styrene; the authors of the study reported visual-evoked-response and electroencephalogram changes in these subjects (NIOSH 1983a, p. 150). Other studies have reported irritation of the eyes and throat at concentrations ranging from 1 to 100 ppm (NIOSH 1983a, p. 151).

Workers in reinforced plastics (RP) facilities in many countries have also evidenced narcotic effects as a consequence of styrene exposure. Swedish, Dutch, and Czechoslovakian workers in RP plants complained of headache, fatigue, drowsiness, giddiness, and dizziness at exposure levels in the range of 4 to 195 ppm (NIOSH 1983a, p. 151). Respiratory effects have been observed in United States RP workers exposed to from 9 to 111 ppm styrene; symptoms included wheezing, shortness of breath, and chest tightness. Another study showed a significantly greater number of RP workers with abnormal pulmonary function when compared with workers from a nonstyrene facility (NIOSH 1983a, pp. 153–154).

Several animal and human studies have suggested that styrene may be a carcinogen. A nested case-control study conducted by McMichael, Spirta, Gamble, and Tousey (1976/Ex. 1–206) found significantly increased risks of lymphatic and hematopoietic cancer, lymphatic leukemia, and stomach cancer among workers exposed to both styrene and butadiene. A retrospective cohort mortality study by Meinhardt, Lemen, Crandall, and Young (1982/Ex. 1–199), also among workers exposed concurrently to styrene and butadiene, reported an excess risk of leukemia and aleukemia in these workers. In a study sponsored by the Chemical Manufacturers Association (Dow 1978, as cited in EPA 1987/Ex. 1–836), male and female Sprague-Dawley rats were exposed to styrene vapor at concentrations of 600 to 1200 ppm, 6 hours/day, 5 days/week, for 18 or 20 months. The higher exposure level was reduced to 1000 ppm after the first 2 months of exposure because of excessively reduced weight in the male rats. A statistically significant increased incidence of mammary tumors was reported in low-dose female rats (7 of 87) compared with controls (1 of 85); no increase in mammary tumors was reported among high-dose female rats. The authors questioned the significance of this response, since historical control animals from the same laboratory showed a higher background incidence of mammary tumors than did the controls used in this study.

In a 1979 NCI study (NCI 1979b/Ex. 1–948), male and female B6C3F1 mice and Fischer 344 rats were treated by gavage 5 days/week for 78 weeks (low-dose rat groups were treated for 103 weeks). The study was terminated at 91 weeks for mice and at 104 to 105 weeks for rats. Dose-related increases in alveolar/bronchiolar adenomas and carcinomas were observed only in the low-dose (150 mg/kg) and high-dose (300 mg/kg) male mice; the incidence of tumors for vehicle controls, low-dose, and high-dose male mice was 0/20, 6/44, and 9/43, respectively. Although the historical incidence of tumors among untreated controls was 12 percent (32/271), the historical incidence of vehicle controls was 0/40.

Based on this evidence, OSHA preliminarily finds that workplace exposures to styrene are associated with health effects ranging from narcosis to neuropathies and irritation; in addition, although the evidence is not conclusive, styrene exposure may cause cancer. The Agency believes that an 8-hour TWA PEL of 50 ppm and a STEL of 100 ppm are necessary to protect workers in construction, maritime, and agriculture against these significant occupational risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

TOLUENE

CAS: 108–88–3; Chemical Formula: $C_6H_5CH_3$

H.S. No. 1397

The current OSHA standard for toluene in construction, shipyards, marine terminals, and longshoring operations is 200 ppm as an 8-hour TWA limit. There is no PEL in agriculture. The 1987–1988 ACGIH TLV®–TWA for toluene was 100 ppm and the TLV®–STEL was 150 ppm; NIOSH recommends a 100-ppm 8-hour TWA and a 10-minute ceiling of 200 ppm. The Agency is proposing an 8-hour TWA PEL of 100 ppm and a STEL of 150 ppm for toluene; NIOSH (Ex. 8–47, Table N1) concurs with these limits. These are the limits recently established for these substances in general industry.

Toluene is a flammable, colorless liquid with an aromatic, hydrocarbon

odor. It is used primarily in the manufacture of benzene and other chemicals. Toluene is also a constituent of solvents, cements, inks, spot removers, cosmetics, antifreeze, and gasoline (HSDB 1989; Proctor, Hughes, and Fischman 1988, p. 477).

Toluene is an irritant of the eyes and skin and a central nervous system depressant in humans and animals. The oral LD_{50} in rats is 5000 mg/kg, and the lowest lethal concentration in the same species is 4000 ppm for 4 hours (RTECS 1990). The dermal LD_{50} in rabbits is 12,124 mg/kg (RTECS 1990). Cats exposed to 7800 ppm showed tremors and prostration within 80 minutes and a mild degree of narcosis within 2 hours (Carpenter et al. 1976). Exposure of cats to near-lethal toluene concentrations for 6 hours resulted in the development of vacuoles in the corneal epithelium (Grant 1986, p. 927). In contact with the skin, toluene causes marked irritation (Wolf et al. 1956). Chronic exposure to concentrations ranging from 100 to 1000 ppm caused behavioral symptoms in rats (Ishikawa and Schmidt 1973).

Von Oettingen, Neal, Donahue et al. (1942/Ex. 1–875) exposed human volunteers to toluene levels ranging from 50 ppm to 800 ppm for 8 hours/day. These authors report that exposures to 50 ppm cause drowsiness and headaches and that exposures at 100 ppm result in sleepiness, moderate fatigue, and headaches. At 200 ppm, effects included impairment of coordination and reaction times. Later studies by Ogata, Tomokuni, and Takatsuka (1970/Ex. 1–352) showed an increase in reaction time, a decrease in pulse rate, and a decrease in systolic blood pressure in humans exposed to 200 ppm toluene for 7 hours.

A study by Greenberg, Mayers, Heinmann, and Moskowitz (1942/Ex. 1–325) reported that painters exposed to toluene levels of 100 to 1100 ppm exhibited enlarged livers, a moderate decrease in red blood cell counts, enlarged red blood cells, and absolute lymphocytosis, but no leukopenia. Wilson (1943/Ex. 1–403) observed 1,000 workers exposed to toluene at levels ranging from 50 ppm to 1500 ppm for periods of 1 to 3 weeks. One hundred of these workers developed symptoms severe enough to require hospitalization. At levels less than 200 ppm, 60 of these employees experienced headache, fatigue, and lack of appetite. Those workers exposed to 200 to 500 ppm toluene experienced headache, nausea, bad taste in the mouth, lassitude, temporary amnesia, impaired coordination, and anorexia. Levels of exposure from 500 to 1500 ppm resulted in nausea, headache, dizziness, anorexia, marked loss of coordination, diminished reaction time, pronounced weakness, and heart palpitations. Red cell counts were also decreased, and two cases of aplastic anemia required lengthy hospital treatment; however, the author noted that he could not rule out the possibility that benzene contamination of the toluene was the cause of these blood effects. Aplastic anemia (including one fatal case) has been noted in six glue sniffers; toluene was the base solvent in the glue (Powars 1965/Ex. 1–433). A man who had inhaled toluene regularly at unspecified levels for 14 years developed permanent encephalopathy (Knox and Nelson 1966/Ex. 1–421).

In the prior air contaminants rulemaking, one commenter (Ex. 3–896) argued that a short-term exposure limit for toluene was not justified. OSHA responded that, as in the cases of octane and pentane, a short-term exposure limit is necessary to ensure that workers are not exposed at the elevated levels possible with a TWA limit alone, particularly in light of the fact that concentrations only slightly above the 8-hour TWA may cause incoordination and amnesia. For example, workers could be exposed to toluene at levels as high as several hundred ppm if the Agency only had an 8-hour TWA PEL for this substance.

OSHA is proposing to establish an 8-hour TWA PEL of 100 ppm and a STEL of 150 ppm for toluene in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that studies clearly indicate that a significant risk of hepatotoxic, behavioral, and nervous system effects exists at toluene levels substantially at or only slightly above the Agency's current PEL. OSHA believes that the proposed limits are necessary to substantially reduce this risk.

TRICHLOROETHYLENE

CAS: 79–01–6; Chemical Formula: CCl_2-CHCl

H.S. NO. 1406

OSHA's current limit for trichloroethylene in construction, shipyards, marine terminals, and longshoring operations is 100 ppm as an 8-hour TWA. There is no PEL in agriculture. The Agency is proposing a TWA PEL of 50 ppm and a STEL of 200 ppm for this substance in construction, maritime, and agriculture. The 1987–1988 ACGIH TLV®–TWA for trichloroethylene was 50 ppm and the TLV®–STEL was 200 ppm. The NIOSH REL is 25 ppm as a 10-hour TWA; however, NIOSH concurs (Ex. 8–47, Table N8A) with the limits being proposed.

Trichloroethylene is a colorless, nonflammable, noncorrosive liquid with the sweet odor characteristic of some chlorinated hydrocarbons. It is used as a carrier solvent for fungicides and insecticides, as a degreasing solvent, as a chemical intermediate, and in dry cleaning and extraction operations. It is also occasionally used as an anesthetic and analgesic (HSDB 1990; Proctor, Hughes, and Fischman 1988, p. 486).

Trichloroethylene is a central nervous system depressant, an irritant of the eyes and skin, and, in experimental animals, a carcinogen. The intraperitoneal LD_{50} in rats is 1282 mg/kg, and the oral LD_{50} and LC_{50} in mice are 2402 mg/kg and 8450 ppm for 4 hours, respectively (RTECS 1990). Acutely poisoned animals die of respiratory failure or cardiac arrest (ACGIH 1986, p. 595). Prolonged or repeated contact with the skin of rabbits caused severe irritation; dropped into the eyes of rabbits, trichloroethylene produced moderate irritation (RTECS 1990). Rats inhaling a trichloroethylene concentration of 3000 ppm for 8 hours did not become anesthetized; however, increasing the concentration to 4800 ppm caused deep anesthesia in these animals (Adams et al. 951). At doses that cause deep anesthesia, trichloroethylene is a cardiac sensitizer in dogs (Reinhardt et al. 1973). Rats, rabbits, and guinea pigs exposed repeatedly (7 hours/day, 5 days/week for 6 months) to trichloroethylene concentrations ranging from 400 to 3000 ppm showed increases in liver and kidney weights at autopsy (Adams et al. 1951).

There is a considerable body of carcinogenicity data on trichloroethylene. In an NCI bioassay (1976b/Ex. 1–168), mice given trichloroethylene by gavage developed hepatocellular carcinomas, but rats did not. The species difference in response was attributed to a difference in the way trichloroethylene is metabolized between the mouse and rat (Stott, Quast, and Watanabe 1982/Ex. 1–833). An inhalation study in mice, rats, and Syrian hamsters (Henschler, Romen, Reichert et al. 1980/Ex. 1–330) found an increase only in the occurrence of malignant lymphomas in mice, which the authors attributed to the strain of mouse used (NMRI).

Several other bioassays on trichloroethylene have recently been published. Fukuda, Takemoto, and Tsuruta (1983/Ex. 1–1109) exposed female rats and mice to 50, 150, or 450 ppm trichloroethylene for 103 weeks and

reported an increased incidence of lung tumors among mice only. Maltoni, Lefemine, and Cotti (1986/Ex. 1–1160) exposed rats and mice to 100, 300, or 600 ppm trichloroethylene and reported a significant increase of renal adenocarcinomas and Leydig cell tumors in rats, as well as a significant increase in hepatomas and lung tumors in mice. In 1988, the NTP reported an increase in the incidence of kidney tumors in rats given trichloroethylene by gavage; however, the NTP considered the tumor response to be weak (3 of 49 animals) and reported that the results were only statistically significant after corrections for high mortality were made.

In humans, trichloroethylene primarily affects the central nervous system and liver; some studies have shown that chronic exposure to concentrations below 100 ppm is associated with a variety of nervous disturbances. Bardodej and Vyskocil (1956/Ex. 1–461) have reported that workers report signs and symptoms of trichloroethylene poisoning, including tremors, giddiness, anxiety, and alcohol intolerance, when they are exposed to concentrations of trichloroethylene above 40 ppm.

Haas (1960) and Grandjean, Muchinger, Turrian et al. (1955/Ex. 1–324) reported nervous symptoms among workers exposed for 5 years or more to trichloroethylene concentrations ranging from 1 to 335 ppm; the frequency of complaints increased when average exposures exceeded 40 ppm.

A number of epidemiologic investigations having cohorts as large as 7688 workers have found no correlation between cancer mortality and exposure to trichloroethylene (Novotna, David, and Malek 1971; Axelson, Andersson, Hogstedt et al. 1978/Ex. 1–713; Tola, Vilhunen, Jarvinen, and Korkala 1980/Ex. 1–391).

Based on this evidence, OSHA preliminarily concludes that the current 100-ppm TWA PEL for trichloroethylene is insufficiently protective against this substance's exposure-related central nervous system (CNS) effects and, further, that exposure to trichloroethylene may present a possible carcinogenic hazard. As stated above, OSHA is proposing an 8-hour 50 ppm TWA and 200 ppm STEL for this substance in these sectors. These are the final limits recently established for general industry. However, as OSHA indicated in the Agency's brief to the U.S. Court of Appeals for the Eleventh Circuit in the pending case *AFL* v. *OSHA*, Nos. 89–7185, et al. "* * * OSHA concludes that the evidence may warrant a lower limit if the next PEL updated establishes the risk of central nervous system effects and carcinogenicity at levels below 50 ppm."OSHA does not intend to resolve that issue in this rulemaking, because its priority in this rulemaking is to extend the updated general industry PELs to all other segments. OSHA believes that attempting to resolve the issues of central nervous system effects and carcinogenicity and similar complex issues in this rulemaking would make this rulemaking difficult to complete in a timely fashion. OSHA will consider for all industry sectors whether a lower PEL is needed in the first PEL update.

Preliminary Conclusions for This Group of Narcotic Agents

OSHA preliminarily concludes that workers exposed to these narcosis-causing substances in construction, maritime, and agriculture workplaces are at significant risk of experiencing a broad range of narcotic effects, including loss of consciousness, incoordination, inability to concentrate, drowsiness, irritability, poor judgment, and inappropriate behavior. These highly undesirable and potentially serious health effects additionally have the potential to cause serious workplace accidents and injuries because they interfere with reaction times, muscle coordination, and the ability to make good decisions and exercise good judgment. The new or revised exposure limits being proposed by OSHA will protect workers in construction, maritime, and agriculture from experiencing these significant risks in their places of work and will contribute to a substantial reduction in these risks. In addition, promulgation of these limits will make OSHA's PELs for these substances consistent across all regulated sectors.

3. Substances for Which Proposed Limits Are Based on Avoidance of Sensory Irritation

Introduction. Exposure to many chemical agents is associated with the development of sensory irritation, which is initiated when these substances come into contact with the eyes, the mucous membranes, or the skin. OSHA is proposing to establish limits in agriculture, maritime, and construction for a large group of sensory irritants; the limits being proposed would, if established, provide protection against sensory irritation to workers in all of the sectors covered by OSHA. These substances are shown in Table C3–1, along with their current OSHA limits in construction and maritime and the limits being proposed for workplaces in agriculture, construction, and maritime.

Table C3–1 also shows the HS and CAS numbers for these substances and the 1987–1988 ACGIH TLV®s– and NIOSH REL (if any) for each substance. The right-hand column of Table C3–1 shows the proposed limits, which are identical to those recently promulgated for these substances in general industry (see 54 *FR* 2434).

TABLE C3–1.—LIST OF SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON AVOIDANCE OF IRRITANT EFFECTS

H.S. No./Chemical name	CAS No.	Current OSHA PEL in construction and maritime	1987–1988 ACGIH TLV® •••	NIOSH REL •••	Proposed OSHA PEL in construction, maritime, and agriculture
1001 Acetaldehyde	75–07–0	200 ppm TWA	100 ppm TWA, 150 ppm STEL		100 ppm TWA, 150 ppm STEL.
1002 Acetic acid	64–19–7	10 ppm TWA	10 ppm TWA, 15 ppm STEL		10 ppm TWA.
1004 Acetone	67–64–1	1000 ppm TWA	750 ppm TWA, 1000 ppm STEL	250 ppm TWA	750 ppm TWA, 1000 ppm STEL.
1007 Acrolein	107–02–8	0.1 ppm TWA	0.1 ppm TWA, 0.3 ppm STEL		0.1 ppm TWA, 0.3 ppm STEL.
1010 Allyl alcohol	107–18–6	2 ppm TWA, Skin.	2 ppm TWA, 4 ppm STEL, Skin		2 ppm TWA, 4 ppm STEL, Skin.
1012 Allyl glycidyl ether (AGE)	106–92–3	10 ppm Ceiling	5 ppm TWA, 10 ppm STEL, Skin	9.6 ppm Ceiling (15-min).	5 ppm TWA, 10 ppm STEL.
1013 Allyl propyl disulfide	2179–59–1	2 ppm TWA	2 ppm TWA, 3 ppm STEL		2 ppm TWA, 3 ppm STEL.
1021 Ammonia	7664–41–7	50 ppm TWA	25 ppm TWA, 35 ppm STEL	50 ppm Ceiling (5-min).	35 ppm STEL.
1022 Ammonium chloride fume	12125–02–9		10 mg/m³ TWA, 20 mg/m³ STEL.		10 mg/m³ TWA, 20 mg/m³ STEL.
2005 n-Amyl acetate	628–63–7	100 ppm TWA	100 ppm TWA		100 ppm TWA.
2017 Benzoyl peroxide	94–36–0	5 mg/m³ TWA	5 mg/m³ TWA	5 mg/m³ TWA	5 mg/m³ TWA.

TABLE C3-1.—LIST OF SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON AVOIDANCE OF IRRITANT EFFECTS—Continued

H.S. No./Chemical name	CAS No.	Current OSHA PEL in construction and maritime	1987-1988 ACGIH TLV® ***	NIOSH REL ***	Proposed OSHA PEL in construction, maritime, and agriculture
2018 Benzyl chloride	100-44-7	1 ppm TWA	1 ppm TWA	1 ppm Ceiling (15-min).	1 ppm TWA.
1036 Borates, tetra, Na (anhydrous).	1330-43-4		1 mg/m³ TWA		10 mg/m³ TWA.
1037 Borates, tetra, Na (decahydrate).	1303-96-4		5 mg/m³ TWA		10 mg/m³ TWA.
1038 Borates, tetra, Na (pentahydrate).	12179-04-3		1 mg/m³ TWA		10 mg/m³ TWA.
2020 Boron trifluoride	7637-07-2	1 ppm Ceiling	1 ppm Ceiling	(++)	1 ppm Ceiling.
1042 Bromine	7726-95-6	0.1 ppm TWA	0.1 ppm TWA, 0.3 ppm STEL		0.1 ppm TWA, 0.3 ppm STEL.
2021 Bromoform	75-25-2	0.5 ppm TWA, Skin.	0.5 ppm TWA, Skin		0.5 ppm TWA, Skin
1045 2-Butanone (MEK)	78-93-3	200 ppm TWA	200 ppm TWA, 300 ppm STEL	200 ppm TWA	200 ppm TWA, 300 ppm STEL.
1047 n-Butyl acetate	123-86-4	150 ppm TWA	150 ppm TWA, 200 ppm STEL		150 ppm TWA, 200 ppm STEL.
1053 n-Butyl lactate	138-22-7		5 ppm TWA		5 ppm TWA.
1054 Butyl mercaptan	109-79-5	0.5 ppm TWA	0.5 ppm TWA	0.5 ppm Ceiling (15-min).	0.5 ppm TWA.
2025 Butylamine	109-73-9	5 ppm Ceiling, Skin.	5 ppm Ceiling, Skin		5 ppm Ceiling, Skin.
1063 Camphor (synthetic)	76-22-2	2 mg/m³ TWA	2 ppm TWA, 3 ppm STEL		2 mg/m³ TWA.
1064 Caprolactam (dust)	105-60-2		1 mg/m³ TWA, 3 mg/m³ STEL		1 mg/m³ TWA, 3 mg/m³ STEL.
1065 Caprolactam (vapor)	105-60-2		5 ppm TWA, 10 ppm STEL		5 ppm TWA, 10 ppm STEL.
1077 Cesium hydroxide	21351-79-1		2 mg/m³ TWA		2 mg/m³ TWA.
1079 Chlorine	7782-50-5	1 ppm TWA	1 ppm TWA, 3 ppm STEL	0.5 ppm Ceiling (15-min).	0.5 ppm TWA, 1 ppm STEL.
2030 Chlorine trifluoride	7790-91-2	0.1 ppm Ceiling	0.1 ppm Ceiling		0.1 ppm Ceiling.
2031 Chloroacetaldehyde	107-20-0	1 ppm Ceiling	1 ppm Ceiling		1 ppm Ceiling.
2032 alpha-Chloroacetophenone	523-27-4	0.05 ppm TWA	0.05 ppm TWA		0.05 ppm TWA.
1083 Chloroacetyl chloride	79-04-9		0.05 ppm TWA		0.05 ppm TWA.
1084 o-Chlorobenzylidene malononitrile.	2698-41-1	0.05 ppm TWA	0.05 ppm Ceiling, Skin		0.05 ppm Ceiling, Skin.
2037 Chloropicrin	76-06-2	0.1 ppm TWA	0.1 ppm TWA		0.1 ppm TWA.
2040 Copper dust and mist (as Cu).	7440-50-8	1 mg/m³ TWA	1 mg/m³ TWA		1 mg/m³ TWA.
1101 Copper fume (as Cu)	7440-50-8	0.1 mg/m³ TWA	0.2 mg/m³ TWA		0.1 mg/m³ TWA.
1105 Cyanogen	460-19-5	10 ppm TWA	10 ppm TWA		10 ppm TWA.
1106 Cyanogen chloride	506-77-4		0.3 ppm Ceiling		0.3 ppm Ceiling.
2046 Cyclohexane	110-82-7	300 ppm TWA	300 ppm TWA		300 ppm TWA.
2047 Cyclohexene	110-83-8	300 ppm TWA	300 ppm TWA		300 ppm TWA.
2048 Cyclopentadiene	542-92-7	75 ppm TWA	75 ppm TWA		75 ppm TWA.
2052 Diacetone alcohol	123-42-2	50 ppm TWA	50 ppm TWA	50 ppm TWA	50 ppm TWA.
2053 Diazomethane	334-88-3	0.2 ppm TWA	0.2 ppm TWA		0.2 ppm TWA.
1119 Dibutyl phosphate	107-66-4	1 ppm TWA	1 ppm TWA, 2 ppm STEL		1 ppm TWA, 2 ppm STEL.
2057 o-Dichlorobenzene	95-50-1	50 ppm Ceiling	50 ppm Ceiling		50 ppm Ceiling.
1122 1,3-Dichloro-5,5-di-methyl hydantoin.	118-52-5	0.2 mg/m³ TWA	0.2 mg/m³ TWA, 0.4 mg/m³ STEL.		0.2 mg/m³ TWA, 0.4 mg/m³ STEL.
1127 Dichloroethyl ether	111-44-4	15 ppm Ceiling, Skin.	5 ppm TWA, 10 ppm STEL, Skin		5 ppm TWA, 10 ppm STEL, Skin.
1130 2,2-Dichloropropionic acid	75-99-0		1 ppm TWA		1 ppm TWA.
1137 Diethylamine	109-89-7	25 ppm TWA	10 ppm TWA, 25 ppm STEL		10 ppm TWA, 25 ppm STEL.
2062 Diethylaminoethanol	100-37-8	10 ppm TWA, Skin.	10 ppm TWA, Skin		10 ppm TWA, Skin.
1140 Diisobutyl ketone	108-83-8	50 ppm TWA	25 ppm TWA	25 ppm TWA	25 ppm TWA.
2066 Dimethylamine	124-40-3	10 ppm TWA	10 ppm TWA		10 ppm TWA.
2069 Dimethylphthalate	131-11-3	5 mg/m³ TWA	5 mg/m³ TWA		5 mg/m³ TWA.
1158 Epichlorohydrin	106-89-8	5 ppm TWA, Skin.	2 ppm TWA, Skin	(+)	2 ppm TWA, Skin.
2075 Ethyl acetate	141-78-6	400 ppm TWA	400 ppm TWA		400 ppm TWA.
2080 Ethyl amyl ketone	541-85-5	25 ppm TWA	25 ppm TWA		25 ppm TWA.
1162 Ethyl benzene	100-41-4	100 ppm TWA	100 ppm TWA, 125 ppm STEL		100 ppm TWA, 125 ppm STEL.
2077 Ethyl butyl ketone	106-35-4	50 ppm TWA	50 ppm TWA		50 ppm TWA.
1164 Ethyl ether	60-29-7	400 ppm TWA	400 ppm TWA, 500 ppm STEL		400 ppm TWA, 500 ppm STEL.
2079 Ethyl formate	109-94-4	100 ppm TWA	100 ppm TWA		100 ppm TWA.
1165 Ethyl mercaptan	75-08-1	0.5 ppm TWA	0.5 ppm TWA	0.5 ppm Ceiling (15-min).	0.5 ppm TWA.
2081 Ethylamine	75-04-7	10 ppm TWA	10 ppm TWA		10 ppm TWA.
1169 Ethylene glycol	107-21-1		50 ppm Ceiling		50 ppm Ceiling.
1171 Ethylidene norbornene	16219-75-3		5 ppm Ceiling		5 ppm Ceiling.
1179 Fluorine	7782-41-4	0.1 ppm TWA	1 ppm TWA, 2 ppm STEL		0.1 ppm TWA.
2086 Formic acid	64-18-6	5 ppm TWA	5 ppm TWA		5 ppm TWA.
1183 Furfural	98-01-1	5 ppm TWA, Skin.	2 ppm TWA, Skin		2 ppm TWA, Skin.
1184 Furfuryl alcohol	98-00-0	50 ppm TWA	10 ppm TWA, 15 ppm STEL, Skin.	50 ppm TWA	10 ppm TWA, 15 ppm STEL, Skin.
1187 Glutaraldehyde	111-30-8		0.2 ppm Ceiling		0.2 ppm Ceiling.
1196 Hexachlorocyclopentadiene.	77-47-4		0.01 ppm TWA		0.01 ppm TWA.

TABLE C3-1.—LIST OF SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON AVOIDANCE OF IRRITANT EFFECTS- -Continued

H.S. No./Chemical name	CAS No.	Current OSHA PEL in construction and maritime	1987-1988 ACGIH TLV$_a$ ***	NIOSH REL ***	Proposed OSHA PEL in construction, maritime, and agriculture
1197 Hexachloroethane	67-72-1	1 ppm TWA, Skin.	10 ppm TWA	(+)	1 ppm TWA, Skin.
2090 sec-Hexyl acetate	108-84-9	50 ppm TWA	50 ppm TWA		50 ppm TWA.
1204 Hexylene glycol	107-41-5		25 ppm Ceiling		25 ppm Ceiling.
1206 Hydrogen bromide	10035-10-6	3 ppm TWA	3 ppm Ceiling		3 ppm Ceiling.
2091 Hydrogen chloride	7647-01-0	5 ppm Ceiling	5 ppm Ceiling		5 ppm Ceiling.
1208 Hydrogen fluoride (as F)	7664-39-3	3 ppm TWA	3 ppm Ceiling	3 ppm TWA, 6 ppm Ceiling (15-min).	3 ppm TWA, 6 ppm STEL.
2092 Hydrogen peroxide (90%)	7722-84-1	1 ppm TWA	1 ppm TWA		1 ppm TWA.
1211 2-Hydroxypropyl acrylate	999-61-1		0.5 ppm TWA, Skin		0.5 ppm TWA, Skin.
2095 Iodine	7553-56-2	0.1 ppm Ceiling	0.1 ppm Ceiling		0.1 ppm Ceiling.
1217 Iron salts (soluble) (as Fe)	Varies with compound.		1 mg/m^3 TWA		1 mg/m^3 TWA.
2096 Isoamyl acetate	123-92-2	100 ppm TWA	100 ppm TWA		100 ppm TWA.
1224 Isopropyl acetate	108-21-4	250 ppm TWA	250 ppm TWA, 310 ppm STEL		250 ppm TWA, 310 ppm STEL.
1225 Isopropyl alcohol	67-63-0	400 ppm TWA	400 ppm TWA, 500 ppm STEL	400 ppm TWA, 800 ppm Ceiling (15-min).	400 ppm TWA, 500 ppm STEL.
1228 n-Isopropylamine	75-31-0	5 ppm TWA	5 ppm TWA, 10 ppm STEL		5 ppm TWA, 10 ppm STEL.
1226 Isopropyl ether	108-20-3	500 ppm TWA	200 ppm TWA, 310 ppm STEL		500 ppm TWA.
2101 Lithium hydride	7580-67-8	0.025 mg/m^3 TWA.	0.025 mg/m^3 TWA		0.025 mg/m^3 TWA.
2102 Maleic anhydride	108-31-6	0.25 ppm TWA	0.25 ppm TWA		0.25 ppm TWA.
1243 Mesityl oxide	141-79-7	25 ppm TWA	15 ppm TWA, 25 ppm STEL	10 ppm TWA	15 ppm TWA, 25 ppm STEL.
2105 Methyl acrylate	96-33-3	10 ppm TWA, Skin.	10 ppm TWA, Skin		10 ppm TWA, Skin.
1248 Methyl 2-cyano-acrylate	137-05-3		2 ppm TWA, 4 ppm STEL		2 ppm TWA, 4 ppm STEL.
1261 Methyl isobutyl carbinol	108-11-2	25 ppm TWA, Skin.	25 ppm TWA, 40 ppm STEL, Skin.		25 ppm TWA, 40 ppm STEL, Skin.
2106 Methyl isocyanate	624-83-9	0.02 ppm TWA, Skin.	0.02 ppm TWA, Skin		0.02 ppm TWA, Skin.
1263 Methyl mercaptan	74-93-1	0.5 ppm Ceiling	0.5 ppm TWA	0.5 ppm Ceiling (15-min).	0.5 ppm TWA.
2107 Methyl methacrylate	80-62-6	100 ppm TWA	100 ppm TWA		100 ppm TWA.
1264 Methyl n-amyl ketone	110-43-0	100 ppm TWA	50 ppm TWA	100 ppm TWA	100 ppm TWA.
1267 alpha-Methyl styrene	98-83-9	100 ppm Ceiling	50 ppm TWA, 100 ppm STEL		50 ppm TWA, 100 ppm STEL.
1270 o-Methylcyclo-hexanone	583-60-8	100 ppm TWA, Skin.	50 ppm TWA, 75 ppm STEL, Skin.		50 ppm TWA, 75 ppm STEL, Skin.
1298 Osmium tetroxide (as Os)	20816-12-0	0.002 mg/m^3 TWA.	0.002 mg/m^3 TWA, 0.006 mg/m^3 STEL.		0.002 mg/m^3 TWA, 0.006 mg/m^3 STEL.
1302 Paraffin wax fume	8002-74-2		2 mg/m^3 TWA		2 mg/m^3 TWA.
1314 Phenyl ether (vapor)	101-84-8	1 ppm TWA	1 ppm TWA, 2 ppm STEL		1 ppm TWA.
2127 Phenyl ether-biphenyl mixture.		1 ppm TWA			1 ppm TWA.
2129 Phosgene	75-44-5	0.1 ppm TWA	0.1 ppm TWA	0.1 ppm TWA, 0.2 ppm Ceiling (15-min).	0.1 ppm TWA.
1322 Phosphoric acid	7664-38-2	1 mg/m^3 TWA	1 mg/m^3 TWA, 3 mg/m^3 STEL		1 mg/m^3 TWA, 3 mg/m^3 STEL.
2131 Phosphorus pentachloride	10026-13-8	1 mg/m^3 TWA	0.1 ppm (1 mg/m^3) TWA		1 mg/m^3 TWA.
1325 Phosphorus trichloride	7719-12-2	0.5 ppm TWA	0.2 ppm TWA, 0.5 ppm STEL		0.2 ppm TWA, 0.5 ppm STEL.
1334 Potassium hydroxide	1310-58-3		2 mg/m^3 Ceiling		2 mg/m^3 Ceiling.
1343 Propylene glycol monomethyl ether.	107-98-2		100 ppm TWA, 150 ppm STEL		100 ppm TWA, 150 ppm STEL.
1350 Rosin core solder pyrolysis products, (as formaldehyde).			0.1 mg/m^3 TWA		0.1 mg/m^3 TWA.
1365 Sodium bisulfite	7631-90-5		5 mg/m^3 TWA		5 mg/m^3 TWA.
1367 Sodium hydroxide	1310-73-2	2 mg/m^3 TWA	2 mg/m^3 Ceiling	2 mg/m^3 Ceiling (15-min).	2 mg/m^3 Ceiling.
1368 Sodium metabisulfite	7681-57-4		5 mg/m^3 TWA		5 mg/m^3 TWA.
1376 Sulfur monochloride	10025-67-9	1 ppm TWA	1 ppm Ceiling		1 ppm Ceiling.
1377 Sulfur pentafluoride	5714-22-7	0.025 ppm TWA	0.01 ppm Ceiling		0.01 ppm Ceiling.
2147 Sulfuric acid	7664-93-9	1 mg/m^3 TWA	1 mg/m^3 TWA, 3 mg/m^3 STEL	1 mg/m^3 TWA	1 mg/m^3 TWA.
1387 Tetrahydrofuran	109-99-9	200 ppm TWA	200 ppm TWA, 250 ppm STEL		200 ppm TWA, 250 ppm STEL.
2157 Tetranitromethane	509-14-8	1 ppm TWA	1 ppm TWA		1 ppm TWA.
1389 Tetrasodium pyrophosphate.	7722-88-5		5 mg/m^3 TWA		5 mg/m^3 TWA.
1392 Thioglycolic acid	68-11-1		1 ppm TWA, Skin		1 ppm TWA, Skin.
1405 1,2,4-Trichlorobenzene	120-82-1		5 ppm Ceiling		5 ppm Ceiling.
1408 Triethylamine	121-44-8	25 ppm TWA	10 ppm TWA, 15 ppm STEL		10 ppm TWA, 15 ppm STEL.
2166 Turpentine	8006-64-2	100 ppm TWA	100 ppm TWA		100 ppm TWA.
1421 Vanadium (V_2O_5, respirable dust).	1314-62-1	0.5 mg/m^3 Ceiling.	0.05 mg/m^3 TWA	0.05 mg/m^3 Ceiling (15-min).	0.05 mg/m^3 TWA.

TABLE C3-1.—LIST OF SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON AVOIDANCE OF IRRITANT EFFECTS—Continued

H.S. No./Chemical name	CAS No.	Current OSHA PEL in construction and maritime	1987-1988 ACGIH TLV® ***	NIOSH REL ***	Proposed OSHA PEL in construction, maritime, and agriculture
1422 Vanadium (V_2O_5, fume)	1314-62-1	0.1 mg/m³ Ceiling.	0.05 mg/m³ TWA	0.05 mg/m³ Ceiling (15-min).	0.05 mg/m³ TWA.
1424 Vinyl acetate	108-05-4		10 ppm TWA, 20 ppm STEL	4 ppm Ceiling (15-min).	10 ppm TWA, 20 ppm STEL.
1427 Vinyl toluene	25013-15-4	100 ppm TWA	50 ppm TWA, 100 ppm STEL		100 ppm TWA.
1429 VM & P Naphtha	8032-32-4		300 ppm TWA		300 ppm TWA, 400 ppm STEL.
1431 Xylenes (o-, m-, and p-isomers).	1330-20-7	100 ppm TWA	100 ppm TWA, 150 ppm STEL	100 ppm TWA, 200 ppm Ceiling (10-min).	100 ppm TWA, 150 ppm STEL.
1435 Zinc chloride fume	7646-85-7	1 mg/m³ TWA	1 mg/m³ TWA, 2 mg/m³ STEL		1 mg/m³ TWA, 2 mg/m³ STEL.

* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time; OSHA's PELs do not currently apply in Agriculture.

** The ACGIH TLV®-TWA is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10 hour/day, 40 hour/week exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

+ NIOSH considers this substance a potential occupational carcinogen and recommends that exposures be reduced to the lowest feasible concentration.

++ No exposure limit recommended because of the absence of a reliable monitoring method; use appropriate engineering and work practice controls to reduce exposure to lowest feasible concentration.

Description of the Health Effects

Irritant effects are readily perceived by affected individuals. The symptoms of sensory irritation include stinging, itching, and burning of the eyes, tearing (or lacrimation), a burning sensation in the throat or nasal passages, rhinitis (nasal inflammation), cough, sputum production, chest pain, wheezing, and dyspnea (breathing difficulty). In the majority of cases, the onset of symptoms occurs rapidly upon exposure to the irritant; it is therefore easy to associate the causative agent with the irritant effect.

These effects may cause severe discomfort and be seriously disabling, as is the case with dyspnea or wheezing. The tearing, pain, and irritation associated with exposure of the eye to sensory irritants can be severe; in some cases, it is disabling. In addition to these primary effects, workers distracted by irritant effects are more likely than nonexposed workers to have accidents and thus to endanger both themselves and others. (Irritant effects thus also have substantial productivity impacts.)

The eye irritation caused by exposure to irritants is believed to result from stimulation of the sensory nerve endings in the cornea. There is little information available on the relationship between the severity of the effect and the physical or chemical properties of the irritating substance. In addition, the mechanism of action underlying this irritant effect is not well understood. Mechanisms that have been suggested include physical action of the irritant on nerve endings, binding of the irritant to sulfhydryl groups of protein, inhibition of cellular respiration, and cholinesterase inhibition (Grant 1986/Ex. 1-975). The symptoms of eye irritation are usually transient and do not generally persist after cessation of exposure; however, exposure to concentrations of lacrimators that exceed the levels associated with transient eye irritation may produce corneal or conjunctival injury that requires medical treatment (Grant 1986/Ex. 1-975).

Sensory irritation of the respiratory system primarily affects the upper respiratory tract and causes an increase in sputum production; inflammation of the nasal passages, trachea, and upper bronchial tree; and decreased cilial clearance. These effects produce a burning sensation in the nasal passages and throat; coughing; sneezing; and acute bronchitis. The development of bronchitis indicates that the cilial clearance mechanism has been compromised, and the accumulation of mucus that results increases the risk of secondary bacterial infection. Wheezing may also be apparent, particularly if the affected individual has a history of hyperreactive airway disease. If exposure is sufficiently intense, the irritant may reach the lower portion of the bronchial tree, causing a chemical burn of the parenchyma and the sudden collection of fluid in interstitial spaces and alveoli (pulmonary edema). Irritation-induced edema may have a delayed onset (12 hours or more) and can cause hypoxia and difficulty in breathing.

For the great majority of substances in this group, OSHA's current limits in construction and maritime are based on evidence showing that humans exposed to the chemical at a particular airborne concentration will experience sensory irritation. For a few substances in this group, evidence from animal experiments provided the basis for limit setting. Several general types of evidence were used by OSHA in proposing to revise existing limits or to propose limits for substances previously unregulated in construction, maritime, or agriculture:

- Consideration of new human evidence;
- Reinterpretation of the human data that formed the basis for setting the 1970 TLV® in construction and maritime;
- Consideration of evidence from industrial experience showing that employees are not experiencing irritation; and
- Evaluation of new evidence in animals.

The studies that provide the basis for most of the sensory irritant levels being proposed by OSHA today are generally controlled-exposure experiments that involved human volunteers or reports of adverse effects in workers.

Dose-Response Relationships and Sensory Irritation

Sensory irritation is considered a "threshold" phenomenon; that is, for any sensory irritant, there is an exposure concentration below which very few, if any, individuals will experience irritation. As exposure increases above this level, a larger proportion of exposed individuals will notice the effect and the effect will become increasingly severe. At some concentration above this no-observed-effect level (NOEL), all exposed persons will experience

sensory irritation, although the intensity of the response may vary.

The risk of experiencing irritation that is associated with exposures below the NOEL will be minimal (except in the hypersensitive individual), while the risk of experiencing the irritant effect will increase as exposure increases. At some point above the NOE level (i.e., at some dose of the substance) the response will be 100 percent, and all exposed persons will experience irritation. According to general toxicologic principles (see Section IV.A of this preamble), the shape of the curve that describes responses above the NOEL is sigmoidal, and the steepness of the curve is a function of the variability in individual responses to the particular irritant. For example, if nearly all persons exposed to the substance will experience a response at approximately the same concentration (dose), the curve will be steep; if, on the other hand, the percentage of people responding increases only slowly as concentration rises, the curve will be considerably flatter.

In addition to the relationship between increasing dose and increasing proportion of exposed persons being affected, the intensity of the response also increases with increasing exposure level. Slightly above the NOE level, affected individuals will experience itching and burning of the eyes, nose, and throat; this is a transient effect that disappears when exposure ceases. For some substances, workers may become inured to the sensations and higher exposure levels are necessary to elicit a subjective response. As exposure levels increase, the irritant effects become more severe, until they reach a point where objective signs of mucous membrane irritation are apparent (i.e., redness of the eyes, rhinitis, coughing, and lacrimation).

OSHA preliminarily concludes that exposure limits are needed for those substances for which PELs are being proposed in this rulemaking to protect workers in construction, maritime, and agriculture against sensory irritant effects that result in objective signs of irritation, such as coughing, wheezing, conjunctivitis, and tearing. Such levels of mucous membrane irritation may require medical treatment, adversely affect the well-being of employees, and place the affected individual at risk from increased absorption of the substance and decreased resistance to infection. Exposing workers repeatedly to irritants at levels that cause subjective irritant effects may cause workers to become inured to the irritant warning properties of these substances and thus increase the risk of overexposure. In addition, the long-term effects of repeated low-level sensory irritation have not been well studied.

Analyses of human case reports and the toxicologic data for the substances in this group of chemicals are presented below. In addition, OSHA's preliminary findings in each case are discussed in detail.

ACETALDEHYDE

CAS: 75-07-0; Chemical Formula: CH_3CHO

H.S. No. 1001

The current OSHA PEL for acetaldehyde in the construction and maritime industries is 200 ppm as an 8-hour TWA; there is currently no PEL for acetaldehyde in agriculture. The ACGIH TLV®-TWA for acetaldehyde is 100 ppm (180 mg/m^3), with a STEL of 150 ppm (270 mg/m^3); there is no NIOSH REL for this substance. OSHA is proposing an 8-hour TWA PEL of 100 ppm and a STEL of 150 ppm for acetaldehyde in the agriculture, construction, and maritime industries. These are the limits recently established for acetaldehyde in general industry.

Acetaldehyde is a flammable, colorless, fuming liquid with a pungent, fruity odor (Sax and Lewis 1989, p. 6; New Jersey Fact Sheet 1986, p. 1). Acetaldehyde is used in the production of herbicides, fungicides, and pesticides; in the production of rubber, disinfectants, cosmetics and perfumes; in the silvering of mirrors; in photography; in the manufacture of gelatin, glue, and casein products; as an additive in milk products and candies; and as a preservative in food products and leather (Merck 1983, p. 6; IARC 1986, p. 103; Parmeggiani 1983, p. 36; Hawley's 1987, p. 5; HSDB 1987). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Acetaldehyde is an irritant of the eyes, mucous membranes, and upper respiratory tract, a central nervous system depressant at high concentrations, and, in experimental animals, a carcinogen. The oral LD_{50} in rats is 1930 mg/kg, and the LC_{50} in the same species is 37 g/m^3 (20,550 ppm) for 30 minutes (RTECS 1990). Animals acutely poisoned by inhalation die of narcosis or pulmonary edema (Fairhall 1949). Cats exposed at a level of 1520 ppm acetaldehyde for 7 hours showed reversible irritation of the air passages (Iwanoff 1911). Instilled into the eyes of rabbits, acetaldehyde produced severe irritation; in contact with the skin of rabbits, this substance caused mild irritation (RTECS 1990). Rats exposed to acetaldehyde concentrations of 4000 to 5000 ppm 6 hours/day, 5 days/week for 4 weeks showed varying degrees of degradation of the nasal epithelium (Appelman 1982). Hamsters exposed to a 4560-ppm concentration of acetaldehyde for 3 months showed signs of ocular and nasal irritation and had increased numbers of erythrocytes; at autopsy, increased kidney and heart weights and severe histopathological changes in the respiratory tract were seen in these animals (Kruysse 1975). In carcinogenicity bioassays, acetaldehyde was found to produce adenocarcinomas and squamous cell carcinomas of the nasal mucosa in rats and laryngeal carcinomas in hamsters by inhalation; in addition, acetaldehyde is both embryotoxic and teratogenic when administered to rats and mice during pregnancy (IARC 1987, Suppl. 7, p. 77; IARC 1985, Vol. 36, p. 114). The International Agency for Research on Cancer (IARC) has concluded that the evidence for acetaldehyde's carcinogenicity in animals is sufficient (IARC 1987, Suppl. 7, p. 77).

Humans exposed to concentrations of 25 ppm or 50 ppm acetaldehyde for 15 minutes reported experiencing eye irritation, while those exposed to a 200-ppm acetaldehyde concentration for 15 minutes reported transient conjunctivitis; exposure to a 134-ppm concentration of acetaldehyde for 30 minutes produced mild upper respiratory tract irritation in these volunteers (Silverman 1946; EPA 1983). Repeated or prolonged skin contact with acetaldehyde produces erythema, burns, and dermatitis as a result of primary irritation (Proctor, Hughes, and Fischman 1988, p. 47). Chronic exposure to higher concentrations (above 200 ppm) may injure the corneal epithelium and cause persistent lacrimation, photophobia, and the sensation of a foreign body in the eye (Halbertsma 1927; Silverman 1946). Chronic exposure to low levels of acetaldehyde is reported to have a narcotic effect on the central nervous system, producing bronchial secretions and symptoms similar to those of chronic alcoholism (Fairhall 1949). A study of workers at a German chemical plant who were primarily exposed to acetaldehyde but were also exposed to other chemicals reported an excess of bronchial tumors and tumors of the oral cavity in these workers. Because of mixed exposure, the small number of cases, and the poorly defined exposure population, the International Agency for Research on Cancer concluded that the evidence for the carcinogenicity of acetaldehyde in

humans is inadequate (IARC 1987, Suppl. 7, p. 77).

OSHA believes that employees in the construction and maritime industries are at significant risk of experiencing conjunctivitis and irritation at the concentration of acetaldehyde that is permitted by the current 8-hour TWA limit of 200 ppm in force in these sectors, and that workers in agriculture are at risk of these same effects because acetaldehyde concentrations in this sector may currently be uncontrolled. Therefore, OSHA is proposing that the PEL for acetaldehyde in the agriculture, construction, and maritime industries be set at 100 ppm as an 8-hour TWA and 150 ppm as a 15-minute STEL. Promulgation of these limits will also make OSHA's PELs for this substance consistent across all regulated sectors.

ACETIC ACID

CAS: 64–19–7; Chemical Formula: CH_3COOH

H.S. No. 1002

The current OSHA PEL for acetic acid in the construction and maritime industries is 10 ppm as an 8-hour TWA; there is no PEL for acetic acid in agriculture. The ACGIH TLV®–TWA for acetic acid is 10 ppm (25 mg/m^3) and the STEL is 15 ppm (37 mg/m^3); there is no NIOSH REL for this substance. OSHA is retaining its 8-hour TWA PEL of 10 ppm in the construction and maritime industries and is proposing to establish this PEL in agriculture. The proposed action would make the PEL for acetic acid consistent across all OSHA-regulated industry sectors.

Acetic acid is a clear, colorless, flammable liquid with a pungent odor (Sax and Lewis 1989, p. 16). This substance is used as a fungicide to preserve high-moisture grain and to combat *Aspergillus flavus*, the organism that produces aflatoxin (Grayson 1985, p. 9). Acetic acid is also used in the manufacture of acetic anhydride, cellulose acetate, vinyl acetate monomer, and other chemicals; in the production of insecticides; as a food additive; and for many other purposes (Hawley's 1987, p. 7). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Acetic acid is a strong irritant of the eyes, skin, and upper respiratory tract in both animals and humans. In addition, exposure to high concentrations of this substance causes narcosis in experimental animals. The oral LD_{50} in rats is 3530 mg/kg, and the LC_{50} in mice is 5620 ppm for a 1-hour exposure (RTECS 1990). The dermal LD_{50} in rabbits is 1060 mg/kg (RTECS 1990). Concentrated (glacial) acetic acid caused severe injury when applied to rabbit eyes (Smyth 1951), and contact of the skin with concentrated solutions (50 percent or more) caused moderate to severe burns in guinea pigs (Roudabush 1965). Mice exposed to acetic acid vapors at concentrations greater than 1000 ppm showed signs of irritation of the conjunctiva and upper respiratory tract; at autopsy, these animals exhibited hepatic swelling and visceral congestion (Ghiringhelli 1957). Guinea pigs exhibited minor changes in respiration after exposure to a 5-ppm concentration; exposure to 100 ppm produced a significant increase in pulmonary flow resistance and a decrease in breathing rate and minute volume, which suggests that bronchial constriction is the primary mode of action for acetic acid irritation (Amdur 1961/Ex. 1–601).

Unacclimatized humans have experienced extreme eye and nasal irritation at acetic acid concentrations above 25 ppm and conjunctivitis at concentrations below 10 ppm (Ghiringhelli 1957). A study of 5 workers exposed to acetic acid concentrations ranging from 80 ppm to peaks of 200 ppm for 7 to 12 years found chronic conjunctivitis, blackening and hyperkeratosis of the skin of the hands, chronic bronchitis and pharyngitis, and erosion of exposed teeth in these individuals (Ghiringhelli 1957; Parmeggiani and Sassi 1954).

Based on this evidence in humans and animals, OSHA preliminarily concludes that workers in agriculture who are exposed to acetic acid at the levels permitted by the absence of a limit are at significant risk of experiencing eye, mucous membrane, and skin irritation. OSHA is accordingly proposing an 8-hour TWA of 10 ppm for this substance in agriculture; the Agency believes that this limit is necessary to reduce a significant risk of material health impairment. In addition, promulgation of this limit will make the PEL for acetic acid consistent across all regulated sectors.

ACETONE

CAS:

67–64–1; Chemical Formula: CH_3COCH_3

H.S. No. 1004

OSHA's current PEL for acetone in the construction and maritime industries is 1000 ppm as an 8-hour TWA. There is no PEL for acetone in agriculture. The NIOSH recommended exposure limit (REL) for acetone is 250 ppm as a 10-hour TWA. The ACGIH TLV®–TWA for acetone is 750 ppm (1780 mg/m^3) and 1000 ppm (2380 mg/m^3) as a 15-minute STEL. OSHA is proposing an 8-hour TWA PEL of 750 ppm, and a 15-minute STEL of 1000 ppm for acetone in the agricultural, construction, and maritime industries; these are the PELs recently promulgated for acetone in general industry.

Acetone is a colorless, highly volatile, flammable liquid with an aromatic odor (ACGIH 1986, p. 6). This substance is widely used as a solvent in paints, varnishes, and lacquers and in the leather and rubber industries. It is also used to clean and dry precision parts (ACGIH 1986, p. 6).

Acetone is irritating to the eyes, skin, nose, and throat; at high concentrations, this substance is a central nervous system depressant in both animals and humans. The oral LD_{50} in rats is 5800 mg/kg, the lowest lethal concentration in rats is 1600 ppm for 4 hours, and the dermal LD_{50} in rabbits is 20 g/kg (RTECS 1990). Animals exposed to acetone concentrations above 1000 ppm develop corneal epithelial and conjunctival injury (Grant 1986, p. 41; Carpenter 1948). Exposure of rats to a concentration of 52,200 ppm for 1 hour caused narcosis and, at 126,600 ppm, death (Flury 1934). Rats exposed to acetone at concentrations between 3000 and 16,000 ppm for 4 hours/day for 10 days showed behavioral changes but developed some tolerance when repeatedly exposed (Golberg 1964). Rats exposed to 19,000 ppm acetate for 3 hours/day, 5 days/week for 8 weeks showed no toxic effects (Brucker 1978).

In humans, irritation of the eyes, nose, and throat has been reported at acetone concentrations of 500 ppm (Nelson 1943), and headaches and lightheadedness are experienced at concentrations above 1000 ppm (Raleigh 1972). At a 12,000-ppm concentration of acetone, central nervous system depression severe enough to cause loss of consciousness may occur (Ross 1973). Workers exposed to an acetone concentration of 1000 ppm for 3 hours/day over a 7- to 15-year period complained of respiratory tract irritation, dizziness, and weakness (Vigliani and Zurlo 1955).

In an early controlled-exposure experiment, Nelson, Enge, Ross, et al. (1943/Ex. 1–66) exposed an average of 10 human subjects (both male and female) to a variety of solvents, including acetone, for 3 to 5 minutes. Subjects were asked to judge the level of sensory irritation as absent, slightly irritating, or very irritating. Tests were conducted in a 1200-cubic-foot gas cabinet equipped with an anemostat to distribute the air uniformly. Acetone was reported to produce slight irritation

on exposure to 300 ppm, but exposure to a concentration of 500 ppm produced a degree of eye, nose, and throat irritation that was still described by a majority of the subjects as "tolerable."

Another controlled study on acetone's effects was conducted more recently by Matsushita, Yoshimune, Inoue et al. (1989/Ex. 1–191). In this study, the authors exposed 25 healthy male subjects to 0, 100, 250, 500, or 1000 ppm acetone. Subjects were exposed for 3 hours in the morning and 3 hours in the afternoon, with a 45-minute period between exposures. Irritant responses were scored on a scale from 0 to 12, with a score of 12 representing severe irritation.

Most of the subjects exposed to a 500- or 1000-ppm acetone concentration reported experiencing irritation (graded between 4 and 5 in severity on an ascending severity scale of 1 to 10) during the first 90 minutes of exposure in the morning and the first 60 minutes of exposure in the afternoon. Subjects ceased to report irritation at the 90-minute mark during the afternoon exposure. A lesser degree of irritation was reported to occur among subjects exposed to a 100- or 250-ppm concentration of acetone; however, this irritation subsided after the first 90 minutes of exposure in each of the two exposure periods. Subjects exposed to 250 ppm or a higher concentration reported feeling general weakness and a sense of tension even as long as 24 hours after exposure. Blood and urine samples taken during and after exposure showed increasing blood and urinary acetone levels among subjects exposed to 250 ppm or higher. Following the exposure period, these levels fell to normal values within about 25 to 35 hours after exposure was terminated. The authors also reported an increased leukocyte count in subjects exposed to 500 or 1000 ppm acetone; the increased white cell count persisted for about 24 hours after the cessation of exposure. The authors attributed this increased leukocyte count to acetone's irritant properties (Matsushita, Yoshimune, Inoue, et al. 1989/Ex. 1–191).

In addition to the two controlled-exposure studies discussed above, two industry studies demonstrate acetone's irritant effects. One report by Parmeggiani and Sassi (1954/Ex. 1–759) indicated that six employees exposed to a 307- to 918-ppm concentration of acetone in a rayon acetate plant experienced eye and throat irritation, dizziness, and inebriation. Five of the employees showed objective signs of pharyngeal irritation, four had lung irritation, and three had conjunctivitis. Although the authors attribute the observed CNS effects to excessive concomitant exposure to carbon disulfide, the irritant effects are more likely to have been the result of exposure to acetone, because carbon disulfide is not a primary irritant by vapor inhalation (Proctor, Hughes, and Fischman 1988). The other study, by Vigliani and Zurlo (1955/Ex. 1–164), found that acetone production workers exposed to a 700-ppm concentration of acetone for 3 hours daily for 7 to 15 years experienced inflammation of the respiratory tract, stomach, and duodenum; giddiness; and loss of strength.

To summarize, OSHA believes that the studies discussed above show that acetone is capable of producing sensory irritation at concentrations below 1000 ppm and that long-term exposure to acetone at levels below 1000 ppm can cause CNS disturbances. In addition, the ACGIH (1986/Ex. 1–3, p. 6) reports that chronic exposure to acetone causes respiratory irritation and headaches. Therefore, OSHA concluded in the general-industry air contaminants rulemaking that the findings of these four studies (Nelson, Enge, Ross, et al. 1943/Ex. 1–66; Matsushita, Yoshimune, Inoue, et al. 1989/Ex. 1–191; Parmeggiani and Sassi 1954/Ex. 1–759; Vigliani and Zurlo 1955/Ex. 1–164) were consistent in demonstrating that acute and long-term effects of acetone exposure occur at levels below 1000 ppm. OSHA therefore believes that it is necessary to reduce the limit for acetone to 750 ppm as an 8-hour TWA and 1000 ppm as a STEL to protect workers in construction, maritime, and agriculture from the acute and chronic effects of acetone exposure. These are the limits recently established for acetone in general industry. OSHA preliminarily finds that the chemically induced sensory irritation associated with acute exposures to acetone can occur at levels only slightly above the 750-ppm level being established as an 8-hour TWA. In the absence of a STEL, the 750-ppm limit would permit excursions to levels as high as 12,000 ppm for brief periods. Such levels "depress the central nervous system, causing dizziness, weakness, and loss of consciousness" (Proctor, Hughes, and Fischman 1988). An 8-hour TWA of 750 ppm is necessary to protect workers against the bioaccumulation of acetone, chronic irritation of the respiratory tract, and headaches associated with long-term acetone exposures. In addition, promulgation of these PELs will make OSHA's limits for acetone consistent across all regulated sectors.

ACROLEIN

CAS: 107–02–8; Chemical Formula: $CH_2=CHCHO$

H.S. No. 1007

The current OSHA 8-hour TWA PEL for acrolein in the construction and maritime industries is 0.1 ppm. There is no PEL for acrolein in agriculture. The ACGIH TLV®–TWA for acrolein is 0.1 ppm (0.23 mg/m^3) and the TLV®–STEL is 0.3 ppm (0.69 mg/m^3). There is no NIOSH REL for acrolein. OSHA is retaining its 0.1 ppm 8-hour TWA limit in construction and maritime, proposing to add a STEL of 0.3 ppm in these sectors, and additionally proposing to extend both limits to agriculture. Adoption of the proposed limits will achieve consistency in the exposure limits for acrolein across all OSHA-regulated industry sectors.

Acrolein is a colorless or yellowish flammable liquid with a disagreeable, choking odor (ACGIH 1986, p. 11). Acrolein is used in the production of poultry feed protein supplements (Sittig 1985, p. 28); to control the growth of algae and aquatic weeds (Grayson 1985, p. 22); and is released during welding, during heat cracking of animal and some vegetable fats, and by the operation of internal combustion engines in confined spaces (Parmeggiani 1983, p. 50).

Acrolein is acutely toxic to both animals and humans and is highly irritating to the eyes, skin, and respiratory tract. The LC_{50} in rats is 300 mg/m^3 for 30 minutes, and the dermal LD_{50} in rabbits is 562 mg/kg (RTECS 1990). In early inhalation studies in cats (Iwanoff 1911), exposure to 10 ppm acrolein for 3.5 hours was found to cause only transient effects, including salivation, lacrimation, respiratory irritation, and mild narcosis. However, later studies reported that an exposure to 1 ppm of acrolein produced marked nose and eye irritation in 5 minutes or less (Cook 1945/Ex. 1–726). Over longer periods, studies have demonstrated fatalities in one of six rats exposed for 4 hours to airborne concentrations of acrolein at 8 ppm; at 16 ppm, the mortality was 100 percent (Smyth 1956/Ex. 1–759). Irritation of the upper respiratory tract is the primary symptom of acrolein inhalation, but lung edema can occur after exposure to high concentrations (Henderson and Haggard 1943a/Ex. 1–881). In addition, skin contact with acrolein causes skin burns and severe injury to the cornea in experimental animals (Smyth 1956/Ex. 1–759). Thirty-two of 57 male rats died following exposure to a 4-ppm concentration of acrolein for 6 hours per day for up to 62 days; bronchiolar necrosis and focal pulmonary edema

were noted in these animals at autopsy (Kutzman 1985).

Exposure of humans to high concentrations of acrolein causes tracheobronchitis and pulmonary edema, and the irritation threshold in humans is 0.25 ppm for 5 minutes (Beauchamp 1985). Human fatalities have been reported at acrolein levels as low as 10 ppm, and exposure to a 150-ppm concentration was lethal after 10 minutes (Henderson 1943; Prentiss 1937). In humans, skin contact caused irritation, erythema, and edema; splashed into the eye, acrolein caused blepharoconjunctivitis, lid edema, discharge, and corneal injury (Champeix 1967).

Based on this evidence in humans and animals, OSHA is retaining its current 0.1 ppm TWA limit in construction and maritime, proposing to extend the same 8-hour TWA limit to agriculture, and proposing to add a STEL of 0.3 ppm for acrolein that applies to all OSHA-regulated sectors. OSHA believes that these limits are necessary to protect employees in the agricultural, construction, and maritime industries from the exposure-related effects associated with acrolein, which include eye, nose, and severe pulmonary irritation. OSHA considers these effects material health impairments within the mean of the Act. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

ALLYL ALCOHOL

CAS: 107–18–6; Chemical Formula: $CH_2=CHCH_2OH$

H.S. No. 1010

The current OSHA PEL for allyl alcohol in the construction and maritime industries is 2 ppm as an 8-hour TWA, with a skin notation. There is no PEL for allyl alcohol in agriculture, and there is no NIOSH REL for this substance. The ACGIH TLV®–TWA for allyl alcohol is 2 ppm (4.8 mg/m³), with a STEL of 4 ppm (9.5 mg/m³) and a skin notation. OSHA is proposing to retain the 2-ppm 8-hour TWA PEL for allyl alcohol in the construction and maritime industries, to extend this 8-hour TWA limit to agriculture, and to add a STEL of 4 ppm for all sectors. These are the limits recently established for allyl alcohol in general industry.

Allyl alcohol is a colorless liquid with a pungent, mustard-like odor (Merck 1983, p. 44). Allyl alcohol is used as a fungicide, herbicide, and nematocide (Proctor and Hughes 1978, p. 93), in the refining and dewaxing of mineral oil (NIOSH/OSHA Occupational Health Guideline 1981, p. 3), and in many chemical manufacturing processes (Parmeggiani 1983, p. 128). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Allyl alcohol is highly toxic to both animals and humans; it causes lacrimation and is severely irritating to the eyes, skin, and respiratory tract. Allyl alcohol is readily absorbed through the skin (the dermal LD_{50} in rabbits is 45 mg/kg). The LC_{50} in rats is 76 ppm for an 8-hour exposure (RTECS 1990). Animals exposed to a 500-ppm concentration of allyl alcohol for 1 hour survived, while those exposed to 1000 ppm for 1 hour died (Smyth 1948). Post-mortem examination of rabbits exposed to a 1000-ppm concentration of allyl alcohol for 3 to 4 hours showed hemorrhages of the eyes, histologic changes in retinal ganglion cells, and severe damage and hemorrhage in practically all body tissues (McCord 1932, as cited in ACGIH 1986).

Repeated exposure of 10 rats to a 60-ppm concentration for 7 hours/day caused gasping during the first few exposures, persistent eye irritation, and the death of one rat (Dunlap 1958, as cited in Proctor, Hughes, and Fischman 1988, p. 62). Exposure of dogs, rats, rabbits, and guinea pigs to 7 ppm for 7 hours/day for 6 months resulted in focal necrosis of the liver and necrosis of the convoluted tubules of the kidneys. In mice, administration of allyl alcohol also produced liver necrosis, as well as marked hemolysis and depletion of erythrocyte glutathione (Ferrali et al. 1989). Exposure of animals to airborne concentrations of allyl alcohol produced a sensory irritant effect, and a concentration of 3.9 ppm was sufficient to decrease the respiratory rate in mice by 50 percent (Neilsen et al. 1984).

In humans, the most important adverse effects of occupational exposures to allyl alcohol are upper respiratory tract irritation and burns of the eyes. In a controlled human sensory response study (Dunlap, Kodama, Wellington, et al. 1958/Ex. 1–630), a 5-minute exposure to 25 ppm resulted in severe eye irritation. Milder irritation has been reported to occur at 5 ppm (McCord 1932, as cited in ACGIH 1986/Ex. 1–3, p. 18). Necrosis of the cornea and temporary blindness occurred in one individual exposed to allyl alcohol at a level irritating to the eyes and nose (Smyth 1956/Ex. 1–759). Skin absorption may lead to serious systemic injury (visceral congestion, periportal congestion of the liver, hematuria, and nephritis); in addition, when evaporation from the skin is prevented or reduced, skin contact with allyl alcohol causes burns (ACGIH 1986/Ex. 1–3, p. 18).

In reviewing the evidence for allyl alcohol, OSHA notes that severe eye irritation has been reported to occur in human subjects exposed to 25 ppm for as short an interval as 5 minutes (Dunlap, Kodama, Wellington, et al. 1958/Ex. 1–630); such an exposure would be permitted under the current limit in construction and maritime of 2 ppm as an 8-hour TWA. OSHA also notes that short-term exposure to allyl alcohol produces characteristic effects more severe than those caused by other sensory irritants; these effects include photophobia and blurred vision. The Agency believes that a STEL is necessary to prevent these effects in exposed workers.

To protect workers in maritime, construction, and agriculture from the significant health risks associated with exposure to allyl alcohol, the Agency is retaining the 8-hour TWA PEL of 2 ppm in construction and maritime, proposing a 4-ppm 15-minute STEL in construction and maritime, and proposing both limits in agriculture along with a skin notation. Promulgation of these limits will make the PELs for allyl alcohol consistent across all OSHA-regulated sectors.

ALLYL GLYCIDYL ETHER

CAS: 106–92–3; Chemical Formula: $C_6H_{10}O_2$

H.S. No. 1012

OSHA's current PEL for allyl glycidyl ether (AGE) in the construction and maritime industries is 10 ppm (45 mg/m³) as a ceiling limit. There is no PEL for AGE in agriculture. The ACGIH TLV®–TWA for AGE is 5 ppm (23 mg/m³), with a STEL of 10 ppm (47 mg/m³) and a skin notation. The NIOSH REL for AGE is 9.6 ppm (45 mg/m³) as a 15-minute ceiling. OSHA is proposing to retain the 8-hour TWA PEL of 5 ppm in construction and maritime, to extend this limit to agriculture, and to add a STEL of 10 ppm for AGE in the agricultural, construction, and maritime industries. These are the limits recently established for this substance in general industry.

Allyl glycidyl ether is a colorless liquid of characteristic, but not unpleasant, odor (ACGIH 1986, p. 20). This substance is used as a resin intermediate and as a stabilizer for vinyl resins and rubber (ACGIH 1986, p. 20).

Allyl glycidyl ether is irritating to the eyes, skin, and respiratory tract; it is also a skin sensitizer and may produce systemic toxicity. The LC_{50} in rats is 860 ppm for a 4-hour exposure (RTECS 1990). Rats exposed to a 600-ppm concentration of AGE for 8 hours/day

exhibited irritation of the eyes and respiratory tract, as well as corneal opacities. At necropsy, examination revealed pulmonary inflammation, bronchiectasis, and bronchopneumonia. Rats exposed to 260 ppm showed slight irritation of the eyes and respiratory distress (Hine et al. 1956; Hine and Rowe 1962, as cited in ACGIH 1986). Mice exposed for 4 days to 7.1 ppm AGE developed epithelial necrosis of the nasal turbinates and complete erosion of the olfactory epithelium. Squamous metaplasia of the respiratory epithelium was also observed in mice exposed to 7.1 ppm AGE for 9 days (Gaqnaire et al. 1987, as cited in HSDB). In mice, a 5.7-ppm concentration of AGE was found to decrease the respiratory rate by 50 percent (Gaqnaire et al. 1987).

Exposure of animals to AGE has also been shown to produce a variety of adverse systemic effects. Exposure of rats to a 300 ppm concentration for 7 hours/day for 50 days produced testicular atrophy (Hine et al. 1956; Hine and Rowe 1962). Testicular degeneration, along with cytotoxic effects on bone marrow cells, has also been observed in rats administered 400 mg/kg/day AGE by intramuscular injection (NIOSH 1978). In a recently-completed NTP bioassay, AGE was found to cause olfactory tumors in rats and mice exposed to 10 ppm for 6 hours/day for 2 years (NTP 1990, as cited in RTECS 1990).

In limited human exposure studies, AGE has been demonstrated to cause dermatitis and eye irritation (Hine, Kodama, Wellington et al. 1956/Ex. 1–331). In humans, skin sensitization occurs readily (Hine and Rowe 1963a, as cited in ACGIH 1986/Ex. 1–3, p. 20). In addition to primary irritation and sensitization, the potential exists for cross-sensitization with other epoxy agents (ACGIH 1986/Ex. 1–3, p. 20).

Based on this evidence in humans and animals, OSHA is proposing to make the PELs for allyl glycidyl ether consistent across all industry sectors by extending an 8-hour TWA PEL of 5 ppm to the agriculture sector and by adding a 10-ppm STEL to the limits that apply in all sectors. OSHA preliminarily concludes that these combined limits will reduce the significant risks of sensitization and primary irritation to which workers in these sectors could otherwise be exposed. In addition, promulgation of these PELs will make OSHA's limits consistent across all regulated sectors.

ALLYL PROPYL DISULFIDE

CAS: 2179–59–1; Chemical Formula: $CH_3=CHCH_2S_2C_3H_7$

H.S. No. 1013

The current OSHA PEL for allyl propyl disulfide in the construction and maritime industries is 2 ppm (12 mg/m ^{3}) as an 8-hour TWA. There is no PEL for allyl propyl disulfide in agriculture. The ACGIH TLV®–TWA for this substance is 2 ppm (12 mg/m ^{3}), with a 3 ppm (18 mg/m ^{3}) STEL. There is no NIOSH REL for allyl propyl disulfide. OSHA is retaining its 2-ppm TWA limit for allyl propyl disulfide in construction and maritime, extending this limit to the agricultural sector, and adding a 3-ppm STEL for all sectors. These are the limits recently established for this substance in general industry.

Allyl propyl disulfide is a liquid with a pungent, irritating odor; it is the chief volatile constituent of onion oil (ACGIH 1986, p. 20). Nearly all occupational exposures to allyl propyl disulfide occur in the processing of onions and onion products and in the processing of foods using the substance as a flavoring.

There are no toxicity data in animals for this substance. Allyl propyl disulfide's irritative effects on the human eyes, nose, and upper respiratory tract are well recognized. The most severe irritation effects have occurred when workers were exposed to allyl propyl disulfide in the vicinity of onion slicing machines, where average concentrations of 3.4 ppm have been measured (Feiner, Burke, and Baliff 1946/Ex. 1–604). Administration of allyl propyl disulfide to six normal human volunteers after a 12-hour fast caused a significant fall in blood glucose levels and a significant rise in serum insulin levels during the subsequent 4 hours (Augusti 1975).

OSHA believes that a PEL of 2 ppm is necessary to protect workers in agriculture from this substance's irritant effects and that a STEL of 3 ppm is necessary to prevent employees in agriculture, maritime, and construction from being exposed to short-term concentrations of allyl propyl disulfide of sufficient magnitude to cause severe irritant effects. The Agency preliminarily concludes that the TWA and STEL limits together will protect workers in these sectors from experiencing these significant risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

AMMONIA

CAS: 7664–41–7; Chemical Formula: NH_3

H.S. No. 1021

OSHA's current exposure limit for ammonia in the construction and maritime industries is 50 ppm as an 8-hour TWA. There is no PEL for ammonia in agriculture. The ACGIH TLV®–TWA for ammonia is 25 ppm (17 mg/m^3) with a STEL of 35 ppm (24 mg/m^3). The NIOSH REL for ammonia is 50 ppm (34.8 mg/m^3) as a 5-minute ceiling. OSHA is proposing a limit of 35 ppm as a 15-minute STEL in the agricultural, construction, and maritime industries. This is the limit recently established for ammonia in general industry.

Ammonia is a colorless gas with a strongly penetrating pungent odor (Sittig 1985, p. 71). It may occur as a clear liquid or as a gas, and is often supplied in pressurized cylinders (Genium MSDS 1985, No. 1). Ammonia is used as a fertilizer for direct application to fields in the anhydrous compressed gas form, as a feedstock in the manufacture of fertilizers and other chemical substances, and as a refrigerant (Grayson 1985, p. 94, Parmeggiani 1983, p. 149).

Ammonia is a severe irritant of the eyes, respiratory tract, and skin. The LC_{50} in rats is reported to be 2000 ppm for a 4-hour exposure (RTECS 1990). The toxic effects of ammonia appear to be restricted to its irritant properties. Post-mortem examination of cats and rabbits exposed to 9700 ppm for 1 hour showed severe effects on the upper respiratory tract as well as alveolar congestion, hemorrhage, emphysema, and edema (Boyd et al. 1944, as cited in Clayton and Clayton 1982, p. 3048). Longer-term exposure to lower concentrations produces irritant effects in animals, but no other systemic effects. For example, exposure of several species of animals to 215 or 1100 ppm for 8 hours per day, 5 days per week for six weeks produced no gross or histopathologic changes (Coon 1970, as cited in Clayton and Clayton 1981, p. 3048).

In humans, accidental exposure to high concentrations of ammonia (in the range of 2500 to 6000 ppm) has caused several deaths, with the victims developing chemical pneumonitis; burns of the eyes, face, and mouth; and severe local edema (Proctor, Hughes, and Fischmann 1988, p. 71; NIOSH 1974). At lower concentrations, ammonia gas is severely irritating to the eyes, upper respiratory tract, and moist skin. Exposure to levels in the range of 30 to 50 ppm for 10 minutes causes mild to moderate eye and upper respiratory tract irritation (MacEwen 1970, as cited in Clayton and Clayton 1982, p. 3049).

An unpublished study conducted by the Detroit Department of Health and cited by the ACGIH (1986/Ex. 1–3, p. 27) reports that ammonia concentrations in the range of 20 to 25 ppm elicited complaints of discomfort from workers engaged in blueprinting and copying operations. In addition, a study of pigs

conducted by Stombaugh et al. (1969) appeared to demonstrate that exposure to ammonia also causes systemic effects. The ACGIH established both a full-shift TLV®–TWA of 25 ppm to protect against ammonia's chronic effects and a TLV®–STEL of 35 ppm to protect against ammonia's irritant effects.

OSHA also reviewed NIOSH's recommended 5-minute ceiling limit for ammonia of 50 ppm. When making this recommendation, NIOSH relied on several reports that ammonia concentrations as low as 50 ppm are moderately irritating (Vigliani and Zurlo 1955/Ex. 1–164; Mangold 1971; Industrial Bio-test Laboratories 1973, all as cited in NIOSH 1974a/Ex. 1–238; MacEwen, Theodore, and Vernot 1970/Ex. 1–827; Pagnotto 1973, as cited in ACGIH 1986/Ex. 1–3, p. 27). NIOSH concluded that the "irritating or annoying effects * * * [of exposure to ammonia are] more dependent upon concentration than length of exposure," and that "a standard expressed as a time-weighted average is inappropriate since it would permit fluctuations to concentrations considerably higher than 50 ppm" (NIOSH 1974a/Ex. 1–238, p. 69). In the rulemaking for general industry, OSHA concluded that NIOSH's recommended 50-ppm ceiling limit was above the effect level reported in the Detroit Department of Health studies (1965–1970, as cited in ACGIH 1986/Ex. 1–3, p. 27) for sensory irritation. This view is supported by Proctor, Hughes, and Fischman (1988, p. 71), who report that even 5-minute exposures to 32 ppm caused nasal dryness in 10 percent of exposed volunteers and that 5-minute exposures to 50 ppm ammonia caused nasal irritation and dryness in 20 percent of exposed volunteers.

In the rulemaking for general industry, some rulemaking participants expressed the opinion that OSHA's current 50 ppm TWA PEL was adequate because exposed workers become acclimated to the irritant effects of ammonia. OSHA is concerned with this view of acclimatization because the long-term consequences of a continual assault on the sensory nerves are not known. In addition, acclimatization lessens the ability of workers to discern airborne concentrations of other hazardous materials.

Based on this evidence, OSHA is proposing a 15-minute STEL of 35 ppm in the agricultural, construction, and maritime industries to protect workers against this substance's irritant effects, which have been demonstrated to occur at and below 50 ppm. OSHA preliminarily concludes that the eye and upper respiratory tract irritation associated with ammonia exposure constitute material impairments of health and pose a significant risk to exposed workers in construction, maritime, and agriculture. Promulgation of this limit will also make OSHA's PEL for ammonia consistent across all regulated sectors.

AMMONIUM CHLORIDE (FUME)
CAS: 12125–02–9 Chemical Formula: NH_4C_1
H.S. No. 1022

There is no OSHA PEL for ammonium chloride fume in the agricultural, construction, or maritime industries. The ACGIH TLV®–TWA for this substance is 10 mg/m³ and the TLV®–STEL is 20 mg/m³. There is no NIOSH REL for this substance. OSHA is proposing a PEL of 10 mg/m³ as an 8-hour TWA and a STEL of 20 mg/m³ for ammonium chloride fume in the agricultural, construction, and maritime industries. These are the limits recently established for this substance in general industry.

Ammonium chloride is a white crystalline solid; fumes of this substance are evolved during galvanizing applications (ACGIH 1986, p. 28). Ammonium chloride is used in the manufacture of other ammonia compounds, in fertilizers, as a pickling agent in galvanization, in soldering flux, in melt-retarding snow treatment, and in urea formaldehyde products, (Hawley's 1987, p. 66).

Ammonium chloride fume is an irritant of the eyes, skin, and respiratory tract. The oral LD_{50} in rats is 1650 mg/kg (RTECS 1990). Instilled into rabbit eyes, ammonium chloride causes varying degrees of irritation, ranging from mild to severe (RTECS 1990). In mice, intravenous administration of ammonium chloride caused hyperventilation and clonic movements, followed in some cases by convulsions or coma (Warren and Schenker, 1960; Gosselin, Smith, and Hodge 1984, pp. III–23, 26).

In humans, exposure to ammonium chloride causes irritation of the eyes, nose, and throat. Repeated contact of this substance with the skin may cause dermatitis (Genium MSDS 1991). Welders exposed to ammonium chloride in welding fumes may develop asthma (Genium MSDS 1991).

OSHA believes that, in the absence of any limit on airborne exposure, employees in the maritime, construction, and agriculture industries are at significant risk of experiencing respiratory irritation as a result of exposure to high concentrations of ammonium chloride fume. OSHA preliminarily concludes that the proposed 8-hour TWA limit of 10 mg/m³ and a 15-minute STEL of 20 mg/m³ for ammonium chloride fume in the agricultural, construction, and maritime industries will substantially reduce these risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

n-AMYL ACETATE
CAS: 628–63–7; Chemical Formula: $CH_3COOC_5H_{11}$
H.S. No. 2005

In general industry, construction, and maritime, OSHA's current permissible exposure limit for n-amyl acetate is 100 ppm as an 8-hour TWA. There is no current limit in agriculture. The ACGIH has a TLV®–TWA of 100 ppm (532 mg/m³) for this substance; NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL of 100 ppm for n-amyl acetate in agriculture. This is the limit recently established in general industry.

n-Amyl acetate is a colorless liquid with a pear- or banana-like odor. This substance is used as a solvent for lacquers, paints, artificial leather, cements, and photographic film, and in the manufacture of phosphors in fluorescent lamps. n-Amyl acetate also finds use in printing and finishing fabrics; as a weevil attractant; as a flavoring agent; as an anti-inflammatory agent; as a warning odorant; as a partition solvent in penicillin manufacture, and in research (ACGIH 1986, p. 29; Hawley's 1987, p. 74; AIHA 1978; Clayton and Clayton 1981, p. 2278).

n-Amyl acetate is an irritant of the eyes, skin, and respiratory tract and causes narcosis, anesthesia, and liver damage at high concentrations. The oral LD_{50} in rats is 6500 mg/kg, and the lowest lethal concentration in the same species is 5200 ppm (length of time not specified) (RTECS 1990). The dermal LD_{50} in rabbits is 20 ml/kg (Smyth 1962). Much of the toxicological literature does not distinguish between the isomers of amyl acetate; however, all of the industrially important pentyl acetate isomers (i.e., n-amyl, iso-amyl, and sec-amyl acetate) produce similar toxicological effects (AIHA 1978; Pagnotto 1964). A group of six rats exposed to a 5200-ppm concentration of technical amyl acetate (principally the n-amyl acetate isomer) died after 8 hours of exposure (Smyth 1962). At autopsy, guinea pigs exposed to amyl acetate concentrations of between 9200 and 18,000 ppm for 30 minutes had congestion of the brain, lungs, liver, and kidneys; exposure to 2000 ppm for 30 minutes caused no pathological effects (Clayton and Clayton 1981, p. 2278).

Rabbits exposed to high concentrations of amyl acetate showed signs of liver damage (elevated serum enzymes); at autopsy, histopathological changes were seen in the liver cells (Querci and Mascia 1970). In standardized testing on rabbit eyes, amyl acetate was graded 2 (slightly injurious) on a scale of 1 to 10 (Smyth 1962). Amyl acetate has a defatting action on the skin, and prolonged contact may cause irritation (Smyth 1962). A recent study (Ballantyne, Tyler, and Auletta 1986) suggests that amyl acetate is a skin sensitizer.

Humans exposed to a 1000-ppm concentration of amyl acetate for 30 minutes experienced irritation of the eyes, nose, and throat, as well as headache, fatigue, and excessive salivation (Sax 1975). Exposure to a 200-ppm concentration of isoamyl acetate for 30 minutes is irritating to the human eye (Sandmeyer and Kirwin 1981, p. 2774). Exposure to higher concentrations of amyl acetate caused a burning sensation and hyperemia of the conjunctiva but no corneal damage (Flury and Zernik 1931; Patty 1949). Case reports of workers exposed to amyl acetate indicate that, in addition to irritation, exposure to the solvent causes liver injury, gastrointestinal disturbances, and blood changes (Browning 1953; Lehman and Flury 1943). A plumber using methyl-cellulose paint (containing mixed isomers of amyl acetate) in a small, unventilated room for several hours developed headache, nausea, and vomiting within a few hours, followed a few days later by orthopnea and paroxysmal nocturnal dyspnea, edema, and enlargement of the heart. The patient recovered completely in 6 months (Weissberg and Green 1979). Workers exposed chronically to amyl acetate showed upper respiratory and neurological effects (Zaikov and Babev 1978).

Based on this evidence in humans and animals, OSHA is proposing an 8-hour TWA PEL of 100 ppm for n-amyl acetate in agriculture; adoption of the proposed limit would establish the same PEL for workplaces in all OSHA-regulated industry sectors. The Agency preliminarily concludes that occupational exposure to n-amyl acetate causes irritation of the eyes, skin, and respiratory tract, and, at high concentrations, narcosis and liver damage. Accordingly, OSHA believes that, in the absence of a permissible exposure limit, workers in agriculture are potentially at significant risk for these exposure-related effects and that the proposed PEL will substantially reduce these risks.

BENZOYL PEROXIDE

CAS: 94–36–0; Chemical Formula: $(C_6H_5CO)_2O_2$

H.S. No. 2017

In general industry, construction, and maritime, OSHA's current permissible exposure limit for benzoyl peroxide is 5 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 5 mg/m³ for this substance; the NIOSH REL for benzoyl peroxide is 5 mg/m³ as a 10-hour TWA. OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ for benzoyl peroxide in agriculture. This is the limit recently established for this substance in general industry.

Benzoyl peroxide is used as a bleaching agent for flour, fats, oils and waxes; as a drying agent for unsaturated oils; in the production of cheese; as a polymerization catalyst in the plastics industry; as a burnout agent for acetate yarns; for embossing vinyl flooring; and in the pharmaceutical and cosmetic industries (Hawley's 1987, p. 134; ACGIH 1986, p. 54). This substance is a flammable and explosive white, granular, crystalline solid. It is tasteless and has a slight almond-like odor resembling that of benzaldehyde (IARC 1985, p. 267; ACGIH 1986, p. 54).

Benzoyl peroxide is an irritant of the eyes, mucous membranes, and skin in both humans and animals. The oral LD_{50} in rats is 7710 mg/kg (RTECS 1990). Eye squint, difficulty in breathing, salivation, lacrimation, erythema, and an increase followed by a decrease in motor activity were exhibited by rats during a 4-hour exposure to 24.3 mg/L of 78 percent benzoyl peroxide. All rats observed at 24 and 48 hours post-exposure had returned to normal in appearance (Proctor, Hughes, and Fischman 1988, p. 95). Benzoyl peroxide also causes both primary irritation and sensitization dermatitis (Proctor, Hughes, and Fischman 1988, p. 94; Cralley and Cralley 1985, p. 159). Humans exposed to benzoyl peroxide dust at a concentration of 12.2 mg/m³ (for an unspecified time) experienced pronounced irritation of the nose and throat. Similar exposure to concentrations of benzoyl peroxide dust ranging from 1.34 to 5.25 mg/m³ caused no irritation (ACGIH 1986, p. 54). Benzoyl peroxide lotion applied to the face of two people for acne treatment caused both facial erythema and edema, and patch tests with benzoyl peroxide were positive in these individuals (Proctor, Hughes, and Fischman 1988, p. 94).

Based on this evidence, OSHA preliminarily concludes that agricultural workers exposed to benzoyl peroxide at levels permitted by the absence of a limit are at significant risk of experiencing irritation of the eyes, mucous membranes, and skin as well as sensitization dermatitis. The Agency believes that establishing a PEL of 5 mg/m³ as an 8-hour TWA will protect agricultural workers from these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

BENZYL CHLORIDE

CAS: 100–44–7

H.S. No. 2018; Chemical Formula: $C_6H_5CH_2Cl$

In general industry, construction, and maritime, OSHA currently has an 8-hour TWA limit of 1 ppm for benzyl chloride. The Agency has no PEL for this substance in agriculture. The ACGIH TLV®–TWA for benzyl chloride is 1 ppm as an 8-hour TWA; the NIOSH REL is a 15-minute ceiling of 5 mg/m³ (equivalent to a 15-minute STEL of 1 ppm). OSHA is proposing an 8-hour TWA PEL of 1 ppm for benzyl chloride in agriculture. This is the limit recently established for this substance in general industry.

Benzyl chloride is a clear, colorless liquid that has an unpleasant, pungent odor that is detectable at 0.047 ppm (Genium MSDS 1986, No. 583). Benzyl chloride is used in the manufacture of many different products: dyes, plasticizers, gasoline additives, tanning agents, cosmetics, resins, benzyl compounds, pharmaceuticals, and photographic developers (Hawley's 1987, p. 135; ACGIH 1986, p. 55; Parmeggiani 1983, p. 262).

Benzyl chloride is a powerful lacrimator and an irritant of the eyes, mucous membranes, and upper respiratory tract. This substance is also a carcinogen and reproductive toxin in animals (IARC 1987, p. 148; RTECS 1990). The oral LD_{50} in rats is 1231 mg/kg, and the LC_{50} in the same species is 150 ppm for 2 hours (RTECS 1990). Rabbits and cats exposed to a 95-ppm concentration of benzyl chloride for 8 hours/day for 6 days showed signs of eye and respiratory tract irritation (ACGIH 1986, p. 55). This substance is a strong skin sensitizer in guinea pigs (HSDB 1985). Benzyl chloride has been tested for carcinogenicity in mice by skin application and in rats by subcutaneous injection. Injection-site sarcomas were observed in rats, and some mice developed skin carcinomas (IARC 1987, p. 148). Skin painting tests in female mice produced squamous-cell carcinomas of the skin (IARC 1987, p. 148). When this substance was administered to mice and rats by

gavage, mice of both sexes developed statistically significant increases in the incidence of papillomas and carcinomas of the forestomach; in female rats only, the incidence of thyroid C-cell tumors was also increased (IARC 1987, p. 148). Based on this evidence, the International Agency for Research on Cancer concluded that the evidence for the carcinogenicity of benzyl chloride in animals is sufficient (IARC 1987, p. 148). When administered to mice intraperitoneally or subcutaneously, benzyl chloride caused paternal reproductive effects (RTECS 1990). Benzyl chloride is mutagenic, with and without metabolic activation, in bacterial and mammalian test systems (RTECS 1990).

In humans, exposure to benzyl chloride causes immediate lacrimation (Proctor, Hughes, and Fischman 1988, p. 95). Splashed into the eye, the liquid causes corneal damage; in contact with the skin, it produces dermatitis (Proctor, Hughes, and Fischman 1988, pp. 95–96). Workers exposed to a 2-ppm concentration reported feeling irritable and weak and had headaches; a 1-minute exposure to a 16-ppm concentration was described as intolerable (Proctor, Hughes, and Fischman 1988, p. 95). There is an unconfirmed report that some benzyl chloride-exposed workers have experienced disturbances in liver function and mild leukopenia (Proctor, Hughes, and Fischman 1988, p. 95). There are two studies of workers exposed to benzyl chloride and other chlorinated toluenes (in Japan and the United Kingdom) that show excesses in the number of cases of respiratory tract and digestive system cancers in these workers. However, the International Agency for Research on Cancer concluded that these data are too limited to permit any differential risk estimation to be made for benzyl chloride (IARC 1987, p. 148).

The evidence described above shows that benzyl chloride exposure causes immediate and severe irritation of the eyes, mucous membranes, upper respiratory tract, and skin, as well as cancer and reproductive effects in animals. Accordingly, OSHA believes that, in the absence of a limit, workers in agriculture are potentially at significant risk of experiencing these exposure-related effects. The Agency believes that the proposed limit of 1 ppm for benzyl chloride is necessary to substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

BORATES, TETRA, SODIUM SALTS (ANHYDROUS, PENTAHYDRATE, AND DECAHYDRATE)
CAS: 1303–96–4 (Decahydrate); Chemical Formula: $Na_2B_4O_7\ 10H_2O$
1330–43–4 (Anhydrous); Chemical Formula: $Na_2B_4O_7$
12179–04–3 (Pentahydrate); Chemical Formula: $Na_2B_4O_7\ 5H_2O$
H.S. Nos. 1036, 1038, and 1037

OSHA has no exposure limits for the anhydrous or hydrated (penta- or decahydrate) forms of sodium tetraborate in the agricultural, construction, and maritime industries. The ACGIH TLV®–TWAs for these substances are 1 mg/m³ for the anhydrous and pentahydrate forms of sodium tetraborate and 5 mg/m³ for the decahydrate form. There is no NIOSH REL for these substances. OSHA is proposing a 10 mg/m³ 8-hour TWA limit for all forms of the sodium tetraborates in the agricultural, construction, and maritime industries. This is the limit recently established for this substance in general industry.

Anhydrous sodium tetraborate is a light gray, odorless solid; the pentahydrate and decahydrate forms are white, odorless, and crystalline (ACGIH 1986, p. 60). Borates are used in agriculture as fertilizers and herbicides; in soap and other cleaners; as fire retardants; and for other purposes (Grayson 1985, p. 78). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The sodium salts of the tetraborates (anhydrous, pentahydrate, and decahydrate (Borax)) are irritating to the skin and respiratory tract in humans. The oral LD_{50} in rats is 2660 mg/kg (RTECS 1990). Guinea pigs given three intratracheal injections of 50 mg each of borax dust (particle size below 5 μm) showed no reactions in the lung, the gastrointestinal tract, or any other organ (Clayton and Clayton 1981, p. 2986). Rats fed borax for 2 years at a dose equivalent to 1170 ppm borax (approximately 1 percent borax in the diet) showed growth suppression, gonadal degeneration, skin desquamation on paws and tails, testicular atrophy, and sterility, and dogs exposed to this dose also showed testicular atrophy. At a 350-ppm dose, no adverse effects on fertility, lactation, litter size, weight, or appearance were seen in rats. In 90-day feeding studies in dogs, a dose of 525 ppm in the diet was the no-effect level (Weir 1972).

Workers exposed to airborne concentrations of (borates) high enough to interfere with normal visibility complained of dermatitis, cough, nasal irritation, nose bleeds, and shortness of breath (Birmingham 1963). At a concentration of 31 mg/m³, boric acid dust causes atrophy of the respiratory mucosa (Clayton and Clayton 1981, p. 3959).

In the prior air contaminants rulemaking, commenters discussed two NIOSH health hazard evaluations (HHEs) relevant to occupational exposure to the borates. The first study (HHE 75–059–496, NIOSH 1978o) was conducted at the Kerr-McGee Chemical Corporation plant in Trona, California. NIOSH performed clinical examinations of nine employees exposed to tetraborates and collected total dust samples for each employee. Clinical examination revealed symptoms of eye irritation in five employees, nose irritation with bleeding in three workers, throat irritation in three employees, and chapping of the hands in four workers. Four of the nine dust samples exceeded 10 mg/m³, with the highest reading at 29.9 mg/m³. However, dust exposures at the California plant may have been well above the 10-mg/m³ level because employees at the plant commented that dust from "frequent [borate] windstorms" was the main problem at the plant. The NIOSH HHE did in fact report that dust levels at the plant were excessive and that the visibility of plant employees was impaired by this dust (Ex. 3–744, Attachment I).

The second NIOSH HHE (conducted in 1980) reported on a walk-through survey of the U.S. Borax and Chemical Corporation's Boron, CA operation. This HHE identified health complaints among employees, and its findings led to a larger, more comprehensive health survey in 1981 (HETA 80–109), a report of which was subsequently published in a peer-reviewed journal (Garabrant, Bernstein, Peters et al. 1985). Data on employees' respiratory symptoms were obtained by questionnaire, and total dust measurements were collected from historical data obtained between 1977 and 1981. The authors found no evidence of X-ray abnormalities or declines in pulmonary function among the 629 active employees examined. There was a dose-related and statistically significant increase in the frequency of reported symptoms, which included eye irritation, dry cough, nosebleeds, sore throat, shortness of breath, and chest tightness. Over 10 percent of employees having mean TWA tetraborate exposures of 8.6 mg/m³, measured as total tetraborate dust, reported experiencing nosebleeds, dry cough, eye irritation, and dryness of the

mouth, nose, or throat. At a mean exposure level of 14.6 mg/m³, between 15 and 30 percent of the employees examined reported these symptoms. The authors concluded that borax dust appears to act as a simple respiratory irritant and may cause small changes in pulmonary function among smokers who are also heavily exposed to borate dust.

Commenters (Ex. 3–744) in the earlier rulemaking took exception to the characterization of worker exposures in the Garabrant et al. paper (1985) on the grounds that exposures at the plant were much higher than reported in that paper and thus that the health effects observed were in fact associated with much higher borate concentrations than reported. Commenters (Ex. 3–744; Tr. 9–133) also stated that the available exposure data for the tetraborates were not adequate to define a dose-response relationship for these substances. As a consequence, U.S. Borax has been conducting a large epidemiologic study at its facility. The results of this study have become available since the conclusion of the prior rulemaking. This study examined the irritant response of 115 workers exposed to borate particulate. Detailed measurements of TWA and peak exposures to borates were made concurrent with medical assessments of the degree of irritation experienced by the exposed group of employees. The results indicate that, at TWA exposure levels between 1 and 4 mg/m³, there is less than a 5-percent probability that workers will experience moderate nose or eye irritation. At exposure levels between 5 and 9 mg/m³, there is an 18-percent probability that workers will experience moderate nasal irritation, but the probability of experiencing moderate eye irritation remains below 5 percent. At TWA exposure levels between 10 and 14 mg/m³, the probabilities of experiencing moderate nasal or eye irritation increased dramatically to 30 percent and 10 percent, respectively.

Based on this evidence, OSHA has preliminarily concluded that it is appropriate in the present rulemaking to propose an 8-hour TWA PEL of 10 mg/m³ for all forms of sodium tetraborate in construction, maritime, and agriculture. This PEL will make OSHA's limit for these substances consistent across all regulated sectors, which is the primary goal of this rulemaking. At the first PEL update rulemaking, OSHA will consider the evidence for these substances again to determine whether further action is warranted.

BORON TRIFLUORIDE

CAS: 7637–07–2; Chemical Formula: BF_3

H.S. No. 2020

OSHA's current PEL for boron trifluoride in general industry, construction, and maritime is 1 ppm as a ceiling limit; there is no current PEL in agriculture. The ACGIH TLV® for boron trifluoride is 1 ppm as a ceiling limit; the NIOSH recommendation for this substance is to reduce exposure to lowest feasible concentration. OSHA is proposing a ceiling of 1 ppm for boron trifluoride in agriculture. This is the limit recently established for this substance in general industry.

Boron trifluoride is used as a fumigant, a catalyst in organic synthesis, in soldering fluxes, and for neutron measurement (Proctor, Hughes and Fischman 1988, p. 100; ACGIH 1986, p. 63). It is a colorless gas that forms a dense white fume in moist air and has a persistent, pungent, and suffocating odor (Proctor, Hughes, and Fischman 1988, p. 100; Genium MSDS 1987, No. 91). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Boron trifluoride is a severe irritant to the lungs, eyes, and skin in both animals and humans (Proctor, Hughes, and Fischman 1988, p. 100; ACGIH 1986, p. 63). The gas reacts with moisture to form boron trifluoride dihydrate, which has a 4-hour LC_{50} of 436 ppm for rats; the symptoms exhibited included gasping, excessive oral and nasal discharge, and lacrimation (Proctor, Hughes, and Fischman 1988, p. 101). All animals exposed at 67 ppm, 6 hours/day, during a 14-day study died before the sixth exposure, and histopathology revealed necrosis and pyknosis of the proximal tubular epithelium of the kidneys; animals exposed to boron trifluoride concentrations of 24 ppm and 9 ppm experienced respiratory irritation, decreased body weight, and at autopsy showed increased lung weight as well as decreased liver weight (Proctor, Hughes, and Fischman 1988, p. 101). In humans, exposure for 30 to 60 minutes at a concentration of 50 ppm can cause massive fatal inflammation and congestion of the lungs. Boron trifluoride gas or fume can cause redness, pain, and serious burns to the eyes or skin.

Based on this evidence, OSHA preliminarily concludes that agricultural workers exposed to this substance are at significant risk of experiencing irritation of the eyes, skin, mucous membranes, and respiratory tract. The Agency believes that establishing a PEL of 1 ppm as a ceiling limit for boron trifluoride is necessary to protect workers in agriculture from these significant risks, which are material impairments of health within the meaning of the Act. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

BROMINE

CAS: 7726–95–6; Chemical Formula: Br_2

H.S. No. 1042

OSHA's current exposure limit for bromine in general industry, construction, and maritime is 0.1 ppm as an 8-hour TWA. There is no PEL for bromine in agriculture. The ACGIH TLV®–TWA for bromine is 0.1 ppm (0.66 mg/m³), and the TLV®–STEL is 0.3 ppm (2.0 mg/m³). NIOSH has no REL for this substance but concurs with the PEL being proposed (Ex. 8–47). OSHA is retaining its 8-hour TWA PEL of 0.1 ppm in construction and maritime and is proposing to add a STEL of 0.3 ppm for bromine in these two sectors; in agriculture, OSHA is proposing both an 8-hour TWA of 0.1 ppm and a STEL of 0.3 ppm. This action will make the PEL for bromine consistent with the limits recently established for this substance in general industry.

Bromine is a dark, reddish-brown, noncombustible, diatomic liquid that has irritating vapors. It is used for shrink proofing wools; as an intermediate for fumigants, e.g. methyl bromide; in flame retardants; in gasoline additives (ethylene dibromide); in hydraulic fluids; for water disinfection; in desizing of cotton; in bleaching of pulp and paper; and in many other uses (Grayson 1985, pp. 185–186; Hawley's 1987, p. 169).

Bromine is a severe irritant of the eyes, skin, mucous membranes, and lungs in both animals and humans. The LC_{50} in mice is 750 ppm for 9 minutes (RTECS 1990); death occurred within 30 days in nearly 50 percent of mice exposed to a 240-ppm concentration of bromine for 2 hours (Bitron and Aharonson 1978). At autopsy, guinea pigs and rabbits exposed to a 300-ppm concentration of bromine for 3 hours showed pulmonary edema, a pseudomembranous deposit on the trachea and bronchi, and hemorrhages of the gastric mucosa. Autopsy of animals that died several days after exposure revealed foci of bronchopneumonia and evidence of functional disturbances in the central nervous system (Lehmann and Hess 1987). Exposure of rats, mice, and rabbits to bromine concentrations of 0.2 ppm for 4 months resulted in respiratory, nervous, and endocrine system dysfunction; exposure of animals of the same species to a 0.02-ppm concentration for the same period produced no adverse effects (NRC 1981). Rats given 0.01 mg/kg bromine by oral administration daily for 6 months

exhibited changes in conditioned reflexes and in several blood indexes (NRC 1981).

Bromine causes lacrimation in humans at concentrations below 6.5 mg/m³ (approximately 1 ppm) (NRC 1980), and inhalation of high concentrations may cause fatal chemical burns of the lungs, lesions of the mucous membranes of the upper airways, inflammation and swelling of the tongue and palate, spasm of the glottis, and asthmatic bronchitis (Parmeggiani 1983, p. 327). Workers handling bromine may develop pustules and furuncles of the skin in exposed areas; if the liquid is not removed immediately, deep, painful ulcers may develop (Oppenheim 1915). Workers exposed regularly to bromine concentrations of approximately 0.3 to 0.6 ppm for 1 year experienced headache, pain in the joints, chest, and stomach, irritability, and loss of appetite (Parmeggiani 1983, p. 327).

The Agency preliminarily concludes that exposure to bromine poses a significant risk of severe eye, skin, and respiratory irritation and believes that both the TWA and short-term limit are necessary to substantially reduce the risk of experiencing these effects. Accordingly, OSHA is proposing a limit for bromine of 0.1 ppm as an 8-hour TWA (in the agriculture sector) and 0.3 ppm as a 15-minute STEL (in the agriculture, construction, and maritime sectors). These limits are consistent with the general industry limits recently established for bromine and are necessary to substantially reduce a significant risk of material health impairment.

BROMOFORM

CAS: 75–25–2; Chemical Formula: $CHBr_3$

H.S. No. 2021

In general industry, construction, and maritime, OSHA's current permissible exposure limit for bromoform is 0.5 ppm as an 8-hour TWA, with a skin notation. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.5 ppm, with a skin notation, for this substance; NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL in agriculture of 0.5 ppm, and a skin notation, for bromoform. This action will make OSHA's limit for bromoform consistent across all industry sectors.

Bromoform is a colorless liquid with an odor like that of chloroform. Bromoform is used as a chemical intermediate, reagent, and solvent, as an ingredient in gauge fluid and fire-resistant chemicals, and in geological assaying. It is also used in the aircraft, aerospace, and shipbuilding industries (HSDB 1984; ACGIH 1986, p. 67).

Bromoform is an irritant of the eyes and respiratory tract and, at high concentrations, a narcotic; chronic exposures of laboratory animals to this substance have resulted in liver and kidney damage. The oral LD_{50} for rats is 1147 mg/kg; the intraperitoneal LD_{50} for the same species is 414 mg/kg (RTECS 1990). Exposure of dogs to a 7000-ppm bromoform concentration caused anesthesia in 8 minutes and death in 60 minutes (Irish 1963). Instilled into the eyes of rabbits, bromoform caused moderate irritation, but healing was complete 2 days later. Moderate irritation resulted from repeated contact of this substance with rabbit skin (Torkelson and Rowe 1981). Exposure of rats to a 25-ppm concentration of bromoform for 4 hours/day for 2 months caused adverse effects on the liver and kidney (Torkelson and Rowe 1981).

Accidental ingestion of small amounts (unspecified doses) of bromoform caused central nervous system depression in humans (von Oettingen 1955), and inhalation of the vapor of this substance caused excessive salivation and irritation of the eyes (with lacrimation), respiratory tract, pharynx, and larynx (von Oettingen 1955). Workers exposed to a mixture of bromoform and bromoethane at approximately 100 mg/m³ experienced central nervous system and digestive disorders, chest pain, kidney dysfunction, and liver disease (Parmeggiani 1983, p. 328).

Based on this evidence, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing the eye and respiratory tract irritation and central nervous system depression associated with exposure to bromoform. The Agency believes that establishing an 8-hour TWA PEL of 0.5 ppm, and a skin notation, will substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

2-BUTANONE (METHYL ETHYL KETONE)

CAS: 78–93–3; Chemical Formula: $CH_3COCH_2CH_3$

H.S. No. 1045

OSHA's current exposure limit for 2-butanone in general industry, construction, and maritime is 200 ppm as an 8-hour TWA. There is no PEL for 2-butanone in agriculture. The ACGIH TLV®–TWA for 2-butanone is 200 ppm, with a STEL of 300 ppm. The NIOSH REL for 2-butanone is 200 ppm (590 mg/m³) as a 10-hour TWA, and NIOSH concurs with the PELs being proposed (Ex. 8–47). OSHA is proposing an 8-hour TWA PEL of 200 ppm and a 300-ppm STEL for 2-butanone in agriculture, construction, and maritime. This action will make the PEL for 2-butanone consistent with the limits recently established for this substance in general industry.

2-Butanone is a colorless, flammable liquid with an acetone-like odor (ACGIH 1986, p. 395). This substance is used as a solvent in resins, paint removers, cements, and coatings, and for many other purposes (ACGIH 1986, p. 395; Hawley's 1987, p. 769).

2-Butanone (also called methyl ethyl ketone) is irritating to the eyes, skin, and respiratory tract in both animals and humans; it is a central nervous system depressant and, in animals, a reproductive toxin. The oral LD_{50} in rats is 2737 mg/kg (RTECS 1990). The lowest lethal concentration in the same species is 2000 ppm for 4 hours, and the dermal LD_{50} in rabbits is 13 g/kg (RTECS 1990). A drop of 2-butanone in the eye of a rabbit caused moderate and reversible injury, graded 5 on an ascending severity scale of 1 to 10 (Smyth 1969). Guinea pigs exposed to a 10,000-ppm concentration of the vapor for 5 hours exhibited signs of narcosis and of severe irritation of the nose and eyes (Clayton and Clayton 1982, p. 4729). Rats exposed to an 800-ppm concentration of 2-butanone for 4 weeks showed increases in liver weights and in liver-to-body weight ratios at autopsy (Scand. J. Work Environ. Hlth 7(1):31, 1981). In a recent study (Takeuchi et al. 1983), rats exposed to a 200-ppm concentration of 2-butanone for 12 hours/day, 7 days/week for 24 weeks showed slight and reversible neurological effects after 4 weeks of exposure. Exposure of pregnant rats to 1000 or 3000 ppm 2-butanone for 7 hours/day on the sixth through fifteenth days of gestation resulted in skeletal abnormalities in the fetuses of rats in the 1000-ppm group and soft-tissue and skeletal abnormalities in the fetuses of rats in the 3000-ppm group (Schwetz, Leong, and Gehring 1974). A follow-up study confirmed 2-butanone's fetotoxicity but did not reveal teratogenic effects (Deacon, Pliny, John et al. 1981).

In humans, 2-butanone causes skin, eye, nose, and upper respiratory tract irritation; exposure to high concentrations may also cause narcosis. Brief exposure to concentrations of 33,000 or 100,000 ppm caused intolerable irritation in volunteers (Patty, Schrenk, and Yant 1935). Volunteers exposed briefly to a 100-ppm concentration reported slight nose and throat irritation; a 15-minute exposure to 200 ppm caused

irritation of the nose and throat; and concentrations greater than 300 ppm caused nausea and eye, nose, and throat irritation (Nelson, Enge, Ross, et al. 1943). Exposure to a 25-ppm concentration of 2-butanone for 5 minutes did not cause irritation (Nelson, Enge, Ross, et al. 1943). Workers exposed to 2-butanone via inhalation and skin contact have also reported experiencing dermatitis and numbness of the extremities (Smith and Mayers 1944). Two young women exposed concurrently to 2-butanone at concentrations of 298 to 560 ppm and to acetone at concentrations of 330 to 495 ppm exhibited signs of severe intoxication, including convulsions and loss of consciousness (Smith 1944).

In the recent air contaminants rulemaking in general industry, OSHA determined that an 8-hour TWA limit of 200 ppm and a 15-minute STEL of 300 ppm were necessary to protect workers in that sector from experiencing the significant irritation and narcotic effects associated with both full-shift and short-term exposures to high concentrations of 2-butanone. Accordingly, OSHA has preliminarily concluded that a 200-ppm 8-hour TWA limit and a 300-ppm 15-minute STEL are necessary to substantially reduce a significant risk of material health impairment among employees in the agricultural, construction, and maritime industries. This action will also make the PELs in these sectors consistent with those in general industry.

n-BUTYL ACETATE

CAS No. 123–86–4; Chemical Formula: $CH_3COO(CH_2)_3CH_3$

H.S. No. 1047

The current OSHA exposure limit for n-butyl acetate in general industry, construction, and maritime is 150 ppm, measured as an 8-hour TWA. There is no PEL for n-butyl acetate in agriculture. The ACGIH TLV®–TWA for n-butyl acetate is 150 ppm, with a STEL of 200 ppm. NIOSH has no REL for this substance but concurs with the PELs being proposed (Ex. 8–47). OSHA is retaining the 8-hour TWA PEL of 150 ppm in construction and maritime, proposing a STEL of 200 ppm in these sectors, and proposing both limits in agriculture. These are the limits recently established for n-butyl acetate in general industry.

n-Butyl acetate is a colorless liquid with a fruity odor. It is used as a larvicide and in the manufacture of lacquers, artificial leather, photographic films, plastics, safety glass, and for many other purposes (Clayton and Clayton 1981, p. 2277; ACGIH 1986, p. 72). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

n-Butyl acetate is an irritant of the eyes, skin, and respiratory system; at high concentrations, it is also a narcotic. The oral LD_{50} in rats is 14 g/kg, and the LC_{50} in the same species is 2000 ppm for 4 hours (RTECS 1987). Cats exposed for 6 hours to a concentration of 6100 ppm showed slight narcotic effects (Flury and Wirth 1933, as cited in ACGIH 1986/Ex. 1–3, p. 72). When the concentration of n-butyl acetate was reduced to 1600 ppm, the cats exhibited signs of mild eye irritation and salivated excessively (Flury and Wirth 1933, as cited in ACGIH 1986/Ex. 1–3, p. 72). A paper by Sayers, Schrenk, and Patty (1936/Ex. 1–802) reported that guinea pigs showed signs of eye irritation at 3300 ppm, became unconscious after 9 hours of exposure to 7000 ppm, and died after 4 hours of exposure to a 14,000-ppm concentration. Cats exposed to a 4200-ppm concentration 6 hours/day for 6 days lost weight, became weak, and showed blood changes; at a 12,000-ppm concentration, cats were anesthetized (Clayton and Clayton 1981, p. 2273). In contact with the skin of rabbits, n-butyl acetate caused moderate irritation (RTECS 1987); instilled into rabbit eyes, this substance produced injury graded 5 on an ascending severity scale of 1 to 10 (Smyth 1954).

Human volunteers complained of eye and throat irritation when exposed to a butyl acetate concentration of 200 ppm (RTECS 1990); these effects became quite severe at 300 ppm (Nelson, Enge, Ross, et al. 1943/Ex. 1–66). Several cases of vacuolar keratitis have been reported in workers exposed concurrently to a mixture of n-butyl acetate and isobutyl alcohol vapors; which compound was responsible for the damage is not known (Grant 1986, p. 163). A pharmaceutical manufacturing worker whose skin came into repeated contact with n-butyl acetate developed dermatitis (Roed-Peterson 1980, in HSDB 1985).

Based on this evidence in humans and animals, OSHA preliminarily concludes that workers are at significant risk of experiencing eye, skin, and respiratory irritation, in addition to narcotic effects, when exposed to n-butyl acetate in the workplace. Accordingly, OSHA is proposing the limits recently established in general industry, which are 150 ppm as an 8-hour TWA and 200 ppm as a 15-minute STEL, for workplaces in the construction, maritime, and agriculture industries. OSHA preliminarily concludes that these limits are necessary to substantially reduce a significant risk of material impairment of health among workers in these sectors.

n-BUTYL LACTATE

CAS: 138–22–7; Chemical Formula: $C_7H_{14}O_3$

H.S. No. 1053

OSHA has no limit for n-butyl lactate in the construction, maritime, and agricultural industries; however, the Agency recently established a 5-ppm 8-hour TWA for this substance in general industry. The ACGIH TLV®–TWA for n-butyl lactate is 5 ppm (30 mg/m^3). NIOSH has no REL for this substance but concurs with the PEL being proposed (Ex. 8–47). OSHA is proposing a PEL of 5 ppm as an 8-hour TWA for n-butyl lactate in the agricultural, construction, and maritime industries. Promulgation of this limit will make the PEL for n-butyl lactate consistent across all OSHA-regulated sectors.

Butyl lactate is a colorless ester of lactic acid (ACGIH 1986, p. 82). It is used as a solvent for nitrocellulose, ethyl cellulose, oils, dyes, natural gums, synthetic polymers, lacquers, and varnishes, as an antiskinning agent, and as an ingredient in perfumes, dry cleaning fluids, and adhesives. It is also a major component of many paints (Hawley's 1987, p. 188; ACGIH 1986, p. 82).

n-Butyl lactate causes irritation of the skin, eyes, and respiratory tract, as well as central nervous system depression, in both humans and animals. The subcutaneous LD_{50} in rats is 12 g/kg; acutely poisoned mice exhibited somnolence and dyspnea before death (RTECS 1989). Applied to the skin of rabbits for 24 hours, butyl lactate caused moderate irritation (RTECS 1989).

Workers exposed for unspecified but prolonged periods to n-butyl lactate at a concentration of approximately 7 ppm, with brief peak excursions to 11 ppm, experienced headache, irritation of the pharyngeal and laryngeal mucosa, and coughing; some workers reported experiencing nausea, vomiting, and sleepiness at this concentration (Zuidema and Pel 1969, as cited in ACGIH 1986). Headache, coughing, and irritation of the pharynx were sometimes associated with exposure to n-butyl lactate concentrations of 4 ppm; however, no adverse effects were reported by workers when the concentration was reduced to 1.4 ppm (Zuidema and Pel 1969). Studies employing improved sampling and analytic methods have subsequently reported that, although the odor of n-butyl lactate is discernible at the 7-ppm level, this concentration does not produce objectionable or injurious

effects (Turner 1972/as cited in ACGIH 1986).

Based on this evidence, OSHA is proposing a 5-ppm 8-hour TWA limit to protect workers in construction, maritime, and agriculture from the significant risks of irritation, headache, and nausea caused by exposure to higher concentrations of n-butyl lactate. The Agency considers these effects material impairments of health within the meaning of the Act. This is the limit recently established for this substance in general industry, and OSHA preliminarily concludes that this limit is necessary to substantially reduce a significant risk of material health impairment among workers in these sectors.

n-BUTYL MERCAPTAN

CAS: 109–79–5; Chemical Formula: $CH_3CH_2CH_2CH_2SH$

H.S. No. 1054

OSHA's current limit for n-butyl mercaptan in general industry, construction, and maritime is 0.5 ppm as an 8-hour TWA. There is no PEL for this substance in agriculture. The NIOSH REL for n-butyl mercaptan is 0.5 ppm as a 15-minute ceiling, and the ACGIH TLV®–TWA for this substance is 0.5 ppm (1.8 mg/m³). OSHA is retaining the 0.5 ppm PEL in the construction and maritime industries and is proposing this limit for agriculture; this is the limit recently established in general industry. This action will make the PEL for n-butyl mercaptan consistent across all OSHA-regulated sectors.

n-Butyl mercaptan is a colorless, flammable liquid that has a strong, obnoxious, garlic-like odor (ACGIH 1986, p. 83). It is used as a solvent, a chemical intermediate, and an odorant for natural gas (ACGIH 1986, p. 83; Hawley's 1987, p. 178).

n-Butyl mercaptan causes irritation and, at high concentrations, central nervous system depression. The LD_{50} in rats is 4020 ppm for 4 hours, and the oral LD_{50} in rats is 1500 mg/kg (RTECS 1990). Acutely poisoned animals showed signs of mucous membrane irritation, dyspnea, incoordination, staggering gait, weakness, partial muscle paralysis, light to severe cyanosis, and mild to heavy narcosis (Fairchild 1958). Application of a drop of butyl mercaptan to rabbit eyes caused momentary minor irritation and closure of the lids (Grant 1974, p. 208). Guinea pigs showed no dermal injury when 0.2 ml of a 20-percent solution of butyl mercaptan was applied to clipped skin for 10 days (NIOSH 1978 Criteria Document). Animals that survived near-lethal doses of n-butyl mercaptan by intraperitoneal injection or oral administration frequently showed liver and kidney damage when autopsied 20 days after exposure (Fairchild 1958, in Clayton and Clayton 1981, p. 2075).

Gobbato and Terribile (1968/Ex. 1–178) have reported that symptoms of CNS toxicity occurred in humans exposed for 1 hour to concentrations of n-butyl mercaptan believed to lie in the range of 50 to 500 ppm. Three of these workers became confused, and one went into a 20-minute coma (NIOSH 1978 Criteria Document on Thiols). Gobbato and Terribile also reported that mucosal irritation occurred in human volunteers exposed to a 4-ppm concentration of ethyl mercaptan, a closely related substance, and that irritation did not occur at exposures to 0.4 ppm.

Based on this evidence, OSHA is proposing a PEL of 0.5 ppm as an 8-hour TWA for n-butyl mercaptan in the construction, maritime, and agriculture industries; this is the limit recently established in general industry. The Agency believes that this PEL is necessary to substantially reduce the significant risks of irritation and CNS toxicity, which constitute material health impairments and are associated with exposure to this substance. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

BUTYLAMINE

CAS: 109–73–9; Chemical Formula: $CH_3(CH_2)_2CH_2NH_2$

H.S. No. 2025

In general industry, maritime, and construction, OSHA's current permissible exposure limit for butylamine is 5 ppm as a ceiling limit, with a skin notation. There is no limit in agriculture. The ACGIH has a TLV® of 5 ppm as a ceiling limit, with a skin notation; NIOSH has no REL. OSHA is proposing a PEL for butylamine in agriculture of 5 ppm as a ceiling limit, with a skin notation. This is the limit recently established for this substance in general industry.

Butylamine is used mainly as an intermediate in the synthesis of insecticides, emulsifying agents, tanning agents, pharmaceuticals, dyestuffs, corrosion inhibitors, petroleum specialties, and rubber chemicals (ACGIH 1986, p. 79; AIHA 1978). It is a volatile, colorless liquid with an odor similar to that of ammonia (ACGIH 1986, p. 79).

Butylamine is irritating to the eyes, skin, mucous membranes, and respiratory tract in both humans and animals. The oral LD_{50} for rats is 500 mg/kg, and the dermal LD_{50} for guinea pigs is 0.5 ml/kg (RTECS 1990). In addition to irritation of the respiratory tract, inhalation of the vapors can also cause pulmonary edema, central nervous system stimulation, depression, convulsions, and narcosis (Proctor, Hughes, and Fischman 1988, p. 108; AIHA 1978). Severe primary irritation and second-degree burns with vesiculation occur when the liquid contacts human skin. Irritation of the eyes, nose, and throat and, occasionally, headaches and facial skin flushing were experienced by workers exposed to butylamine at concentrations of 5 to 10 ppm on a daily basis. Exposure to concentrations above this (in the range of 10 to 25 ppm) were reported to be intolerable after a few minutes by some people, while exposures below 5 ppm resulted in no symptoms (Proctor, Hughes, and Fischman 1988, p. 108). The liquid tested on the eyes of experimental animals was severely damaging (extent of damage equivalent to the damage caused by ammonium hydroxide) (Grant 1986, p. 164; ACGIH 1986, p. 79). Exposure of rats to concentrations of butylamine ranging from 3000 to 5000 ppm for an unspecified time resulted in an immediate irritant response, with labored breathing and pulmonary edema; death occurred within minutes or hours after exposure (Proctor, Hughes, and Fischman 1988, p. 108).

Based on this evidence, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing eye, skin, mucous membrane, and respiratory tract irritation, pulmonary edema, and central nervous system damage. OSHA considers these effects material impairments of health within the meaning of the Act. The Agency believes that establishing a ceiling limit of 5 ppm and a skin notation will substantially reduce these risks.

CAMPHOR (SYNTHETIC)

CAS: 76–22–2; Chemical Formula: $C_{10}H_{16}O$

H.S. No. 1063

OSHA's current limit for synthetic camphor in general industry, construction, and maritime is 2 mg/m³ (0.3 ppm). There is no PEL in agriculture. The ACGIH TLV®s for camphor are a 2 ppm (12 mg/m³) TWA with a 3 ppm (18 mg/m³) STEL. NIOSH has no REL but concurs (Ex. 8–47) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 2 mg/m³ for synthetic camphor in agriculture. This is the limit recently established for this substance in general industry.

Synthetic camphor is a colorless or white crystalline substance with an aromatic odor. This substance is used as a plasticizer in explosives and lacquers,

an ingredient in insecticides, tooth powder, medicines, flavorings, and embalming fluids, and as a chemical intermediate (ACGIH 1986, p. 94).

In humans and animals, camphor is an irritant of the eyes, nose, and respiratory system. The LD_{50} in mice is 1310 mg/kg, and the lowest lethal concentration in the same species is 400 mg/m³ for 3 hours (RTECS 1991). Synthetic camphor is known to cause severe injuries in animals exposed for prolonged periods by inhalation to a level of 6 mg/m³. Exposure may cause convulsions, congestion, changes in the gastrointestinal tract, and damage to the kidneys and brain (Flury and Zernik 1931b/Ex. 1–996). Animal bioassays showed that camphor was not carcinogenic in rats injected subcutaneously; however, when the cancer promoter, croton oil, was concurrently applied to the skin of mice, 2 of 110 treated mice developed carcinomas (Graffi, Vlamynck, Hoffman, and Schultz 1953/Ex. 1–903).

In humans, there are reports of industrial exposure to camphor that resulted in coma, dyspnea, and headache; one fatality from inhalation of the vapor has been noted (Flury and Zernik 1931b/Ex. 1–996). A report by Gronka, Bobkoski, Tomchick, and Rakow (1969/Ex. 1–1043) evaluated airborne exposures and the health status of six employees in a synthetic-camphor-processing plant and reported that exposure for up to 10 months did not produce eye or nasal irritation if concentrations of camphor were maintained at or below 2 ppm. The health status of the six employees was determined before the plant installed local ventilation and improved handling procedures; at that time, camphor concentrations ranged from 24 to 43 mg/m³. Four of the six employees examined showed inflammation of the nose and throat, and one reported having occasional numbness in the fingers. After process improvements were installed, only two of the employees were still working in the camphor-processing area; the remaining four had been away from direct contact with camphor.

Based on this evidence, OSHA preliminarily concludes that the proposed 2 mg/m³ limit is necessary to protect workers in agriculture from the significant risk of headache, difficult breathing, and, if exposure is severe, coma and death. OSHA believes the proposed PEL will substantially reduce these risks. This is the limit recently established for this substance in general industry.

CAPROLACTAM (DUST)

CAS: 105–60–2; Chemical Formula: $C_6H_{11}NO$

H.S. No. 1064

OSHA has no permissible exposure limit for caprolactam dust in the agricultural, construction, or maritime industries. In the recent air contaminants rulemaking, OSHA established a 1-mg/m³ 8-hour TWA and a 3-mg/m³ STEL for this substance in general industry. The ACGIH TLV®–TWA for caprolactam dust is 1 mg/m³ as an 8-hour TWA. NIOSH has no REL for this substance but concurs with the limits being proposed (Ex. 8–47). OSHA is proposing a 1 mg/m³ 8-hour TWA and a 3-mg/m³ STEL for caprolactam dust in the agricultural, construction, and maritime industries; this action will make the PEL for caprolactam dust consistent across all OSHA-regulated sectors.

Caprolactam is a white crystalline solid with an unpleasant odor (ACGIH 1986, p. 95(89)). It is used in the production of Nylon 6 fibers and resins, carpets, clothing, tires, wire insulation, brush bristles, floor polishes, concrete patching cement, and dyes (IARC 1986, p. 250).

In addition to sensory irritation, caprolactam causes central nervous system, respiratory, and cardiovascular effects in experimental animals and humans. The oral LD_{50} in rats is 1210 mg/kg; acutely poisoned animals convulsed and showed signs of eye irritation before death (RTECS 1990). In animals, exposure to caprolactam by any route causes convulsions, tremors, mydriasis, opisthotonus (Elison, Lien, Zinger, et al. 1971/Ex. 1–1050; Lien, Lien, and Tong 1971/Ex. 1–1089), and salivation (Goldblatt, Farquharson, Bennett, and Askew 1954/Ex. 1–1044). Convulsions occurred in rats, rabbits, and cats given intraperitoneal or intravenous injections of caprolactam at doses greater than 100 mg/kg (Goldblatt, Farquharson, Bennett, and Askew 1954/Ex. 1–1044; Hohensee 1951). Cardiovascular and respiratory effects have been reported in rabbits and cats exposed to caprolactam; signs of poisoning included an initial increase, followed by a decrease, in blood pressure and an increase in respiratory rate (Goldblatt, Farquharson, Bennett, and Askew 1954/Ex. 1–1044). Weight loss and initial growth depression occurred in caprolactam-treated rats and mice (Morrison, Ross, and Ruth 1980/Ex. 1–1062). In a 90-day feeding study in dogs, Burdock, Kolwick, Alsakor, and Marshall (1984, as cited in ACGIH 1986/Ex. 1–3, p. 95) reported that animals given dietary doses of 0.1, 0.5, or 1.0 percent caprolactam showed weight losses at both the 0.5-percent and 1.0-percent levels. A 2-year carcinogenicity bioassay of caprolactam in rats and mice was negative (NCI/NTP 1982). The results of studies of caprolactam's teratogenicity in rats and rabbits indicate that it is not teratogenic even at doses as high as 1000 mg/kg/day (Gad, Powers, Robinson et al. 1984). Recent work on the genetic toxicity of caprolactam shows that this substance is not genotoxic in most short-term tests (Brady, Stack, and Waters 1989 (Toxline)). A three-generation study in rats involving dietary administration of caprolactam revealed no caprolactam-related effects on the reproductive performance of rats of either sex or on pup survival (Serota, Hoberman, Friedman, and Grad 1988, in Toxline).

In humans, caprolactam has been shown to be a convulsant, a dermal and respiratory irritant, and a dermal sensitizer. Studies of industrial exposures to caprolactam dust in Germany report severe irritation when workers inhale caprolactam as dust; these workers experienced a bitter taste, nervousness, nose bleeds, upper respiratory tract irritation, and dry and splitting skin on the lips and nose (Hohensee 1951). Direct contact with the solid form of caprolactam produces primary skin irritation (Ferguson 1972; Brief 1972).

Based on this evidence in humans and animals, OSHA preliminarily concludes that, in the absence of a limit for caprolactam dust in the agricultural, construction, and maritime industries, workers are at significant risk of experiencing respiratory irritation and adverse nervous system effects. Accordingly, OSHA is proposing a PEL of 1 mg/m³ (8-hour TWA) and a STEL of 3 mg/m³ for caprolactam dust in the agricultural, construction, and maritime industries. These are the limits recently established for caprolactam dust in general industry. OSHA preliminarily concludes that these limits are necessary to substantially reduce a significant risk of material impairment of health in exposed workers.

CAPROLACTAM (VAPOR)

CAS: 105–60–2; Chemical Formula: $C_6H_{11}NO$

H.S. No. 1065

OSHA currently has no permissible exposure limit for caprolactam vapor in the agriculture, construction, or maritime industries. In the prior air contaminants rulemaking, OSHA established an 8-hour TWA of 5 ppm (20 mg/m³) for caprolactam vapor, supplemented by a STEL of 10 ppm (40 mg/m³), for workplaces in general industry. The

ACGIH TLV®-TWA for this substance is 5 ppm, with a STEL of 10 ppm. There is no NIOSH REL for caprolactam vapor. OSHA is proposing these limits for the agricultural, construction, and maritime industries; this action will make the PEL for caprolactam vapor consistent across all OSHA-regulated sectors.

Caprolactam is a white crystalline solid with a low vapor pressure at room temperature; thus, high vapor concentrations are likely to occur only at elevated temperatures (ACGIH 1986, p. 95(89)). Caprolactam is used to make nylon, carpets, resins, clothing, tires, wire brushes, floor polishers, and many other products.

In addition to sensory irritation, caprolactam vapor can cause neurologic and respiratory effects in experimental animals and humans. The oral LD_{50} in rats is 1210 mg/kg, and the LC_{50} in the same species is 300 mg/m³ for 2 hours (RTECS 1990). Acutely poisoned animals convulsed and showed signs of eye irritation before death (RTECS 1990). A 1984 symposium on caprolactam reported that this substance causes convulsions, tremors, mydriasis, and salivation in experimental animals. Cardiovascular and respiratory effects were also seen in rabbits and cats; in addition, these animals showed an initial increase, followed by a decrease, in blood pressure and an increase in respiratory rate (ACGIH 1986, p. 95(89)). Injected at doses greater than 100 mg/kg, caprolactam caused convulsions in rats, rabbits, and cats (Goldblatt 1954).

The health effects of exposure to caprolactam vapor in humans are identical to those described for caprolactam dust, except that contact with the vapor is reported to be even more irritating (Hohensee 1951, as cited in ACGIH 1986/Ex. 1–3, p. 95). Workers exposed to the vapor at a concentration of approximately 12 ppm complained of a bitter taste in the mouth, nervousness, nose bleeds, upper respiratory tract congestion, and dry and splitting skin; other workers reported experiencing heartburn, flatulence, and a heavy feeling in the stomach (Hohensee 1951, as cited in ACGIH 1986/Ex. 1–3, p. 95). In another report of industrial exposure to the vapor, Ferguson and Wheeler (1973/Ex. 1–1108) reported that workers routinely exposed for 18 years to unspecified levels and occasionally to concentrations as high as 100 ppm reported severe discomfort from burning nose, throat, and eyes. This irritant response was dose-related, with no workers reporting effects at concentrations of 7 ppm or below, some experiencing transient upper respiratory tract irritation at levels above that, and others reporting eye irritation at concentrations of 25 ppm and above (Ferguson and Wheeler 1973/Ex. 1–1108). Ferguson (1972, as cited in ACGIH 1986/Ex. 1–3, p. 96.1) noted that a group of 143 workers, some of whom were exposed for as long as 17 years to vapor concentrations of 5 to 10 ppm, showed no evidence of adverse effects. At higher vapor exposures (13 to 130 ppm), all subjects experienced eye irritation (Ferguson 1972, as cited in ACGIH 1986/Ex. 1–3, p. 96.1). Human volunteers exposed at low relative humidities to concentrations of the vapor in the range of 10 to 100 ppm showed a dose-related response, but at higher relative humidities, no irritation was observed below a concentration of 14 ppm (Ferguson and Wheeler 1973/Ex. 1–1108).

Based on this evidence in humans and animals, OSHA is proposing the limits recently established in general industry—5 ppm as an 8-hour TWA and 10 ppm as a 15-minute STEL—for workplaces in the construction, maritime, and agriculture industries. OSHA believes that these PELs are necessary to substantially reduce the significant risk of eye, upper respiratory tract, and skin irritation associated with exposure to this substance. OSHA considers these exposure-related effects to be material impairments of health. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

CESIUM HYDROXIDE

CAS: 21351–79–1; Chemical Formula: CsOH

H.S. No. 1077

OSHA currently has no limit for cesium hydroxide in the agriculture, construction, or maritime industries. The ACGIH has a 2 mg/m³ 8-hour TWA for cesium hydroxide. NIOSH has no REL for this substance but concurs with the proposed limit (Ex. 8–47). The Agency is proposing a 2 mg/m³ 8-hour TWA PEL for cesium hydroxide in the construction, maritime, and agricultural industries; this is the limit recently established for this substance in general industry.

Cesium hydroxide is a colorless or yellowish fused crystalline mass; it is the strongest base known and is highly soluble in both water and alcohol (ACGIH 1986, p. 113). It is used as an electrolyte in alkaline storage batteries at subzero temperatures and as a catalyst in the polymerization of cyclic siloxanes (Hawley's 1987, p. 245; ACGIH 1986, p. 113)

In addition to causing sensory irritation, cesium hydroxide is corrosive to the eyes of humans and experimental animals. The oral LD_{50} in rats is 570 mg/kg (RTECS 1991). Acutely poisoned animals exhibited somnolence, muscle contractions or spasticity, and changes in respiration before death (RTECS 1991). Rabbits experienced severe eye irritation when 5 mg of a 5M solution of cesium hydroxide was instilled into their eyes and then rinsed out (RTECS 1991). In contact with the skin of rabbits, cesium hydroxide caused mild irritation (RTECS 1991). Studies indicate that cesium hydroxide has an acute oral toxicity of about one-third that of potassium hydroxide (Karpov 1971/Ex. 1–1115).

Inhalation of particles of cesium hydroxide or of mist containing cesium hydroxide is irritating to the eyes, nose, and upper respiratory tract in humans (Genium MSDS 1987, No. 201). If not removed promptly from the skin, this substance causes chemical burns (Genium MSDS 1987, No. 201).

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 2 mg/m³ for cesium hydroxide in the construction, maritime, and agriculture industries; this is the limit recently established for this substance in general industry. The Agency preliminarily concludes that this PEL is necessary to protect workers from the significant risk of irritation and corrosive burns associated with exposure to this substance. OSHA considers these adverse health effects to be material impairments of health within the meaning of the Act and believes that the proposed PEL will reduce these risks substantially.

CHLORINE

CAS: 7782–50–5; Chemical Formula: Cl_2

H.S. No. 1079

The current OSHA limit for chlorine in the construction and maritime industries is 1 ppm as a ceiling. There is no PEL for chlorine in agriculture. The 1987–1988 ACGIH TLV®-TWA for chlorine was 1 ppm (3 mg/m³), with a 3 ppm (9 mg/m³) STEL. The NIOSH REL for chlorine is 0.5 ppm as a 15-minute ceiling; however, NIOSH concurs with the limits being proposed, which are the same PELs as those recently established for chlorine in general industry. OSHA is proposing a PEL of 0.5 ppm as an 8-hour TWA and a 15-minute STEL of 1 ppm for chlorine in the agricultural, construction, and maritime industries. This action will make OSHA's limit for chlorine consistent across all industry sectors.

Chlorine is a greenish-yellow, noncombustible gas at normal temperatures; it has a suffocating odor. At −35°C, chlorine condenses to an

amber liquid (ACGIH 1986, p. 117.1(87)). Chlorine has a wide range of uses; it is an oxidizing and chlorinating agent, an ingredient in many chlorinated organic compounds, and is used to purify water and to shrink-proof wool. Chlorine also is used in flame retardant compounds, in lithium and zinc batteries, and in the processing of meat, fish, vegetables, and fruit (ACGIH 1986, p. 117.1(87)).

Chlorine is a severe irritant of the eyes, nose, lungs, and skin in humans and animals. The LC_{50} in rats is 293 ppm for 1 hour (RTECS 1991). Animals exposed to sublethal doses of chlorine for 15 to 193 days showed emphysema, bronchiolitis, and pneumonia at autopsy (Clayton and Clayton 1982, p. 2958).

The acute effects of chlorine exposure in humans have been well documented since World War I, when chlorine gas was used as a chemical warfare agent. Other severe exposures have resulted from the accidental rupture of chlorine tanks. These exposures have caused death, lung congestion, and pulmonary edema, pneumonia, pleurisy, and bronchitis. The lowest lethal concentration reported is 430 ppm for 30 minutes (Clayton and Clayton 1982, p. 2957); exposure to 15 ppm causes throat irritation, exposures to 50 ppm are dangerous, and exposures to 1000 ppm can be fatal, even if exposure is brief (Sax and Lewis 1989, p. 768). A study of 332 workers exposed to chlorine for an average of 10.9 years was published in 1970. All but six workers had exposures below 1 ppm; 21 had TWAs above 0.52 ppm. No evidence of permanent lung damage was found, but 9.4 percent of these workers had abnormal EKGs compared to 8.2 percent in the control group. The incidence of fatigue was greater among those exposed to chlorine above 0.5 ppm (ACGIH 1986, p. 117.1[873]). In 1981, a study was published involving 29 subjects exposed to up to 2.0 hours for 4- and 8-hour periods. Exposures of 1.0 ppm for 8 hours produced statistically significant changes in pulmonary function that were not observed at the 0.5 ppm exposure concentration. Six of 14 subjects exposed to 1.0 ppm for 8 hours showed increased mucus secretions from the nose and in the hypopharynx. Responses ranging from sensations of itching or burning of the nose and eyes to general discomfort were not severe but were perceptible, especially at 1.0 ppm exposure level. A 1983 study of pulmonary function in workers exposed to low concentrations of chlorine also found transient decreases in pulmonary function at the 1.0 ppm exposure level, but not at the 0.5 ppm level (ACGIH 1986, p. 117.1[87]-117.2[87]).

Based on this evidence in humans and animals, OSHA preliminarily concludes that workers in construction, maritime, and agriculture are at significant risk of experiencing the severe irritation associated even with brief exposure to this substance. OSHA considers these effects material impairments of health and believes that the proposed PELs of 0.5 (8-hour TWA) and 1 ppm (STEL) are necessary to substantially reduce this significant risk. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

CHLORINE TRIFLUORIDE

CAS: 7790–91–2

H.S. No. 2030; Chemical Formula: ClF_3

OSHA has no limit for chlorine trifluoride in agriculture; however, the Agency's current PEL for this substance in general industry, construction, and maritime is 0.1 ppm as a ceiling limit. The 1987–1988 ACGIH TLV® for chlorine trifluoride is 0.1 ppm as a ceiling limit; there is no NIOSH REL for this substance. OSHA is proposing a PEL of 0.1 ppm as a ceiling for chlorine trifluoride in agriculture. This is the limit recently established for this substance in general industry.

Chlorine trifluoride is a gas that is nearly colorless and has a sweet but irritating odor. This substance is easily pressurized to a pale green liquid; when frozen, it forms a white solid (ACGIH 1986, p. 119; Braker and Mossman 1980, p. 164). Chlorine trifluoride is used as an incendiary, as a fluorimetry agent, as a pyrolysis inhibitor for fluorocarbon polymers, as an ignitor and propellant for rockets, in cutting oil well tubes, and in the reprocessing of nuclear reactor fuels (Hawley's 1987, p. 280; Proctor, Hughes, and Fischman 1988, p. 131).

In animals, gaseous chlorine trifluoride causes lung damage and severe irritation of the eyes, skin, and respiratory tract; only limited data are available on human exposure to this substance (Proctor, Hughes, and Fischman 1988, p. 131; RTECS 1990). The LC_{50}s in monkeys, mice, and rats are, respectively: 230 ppm for 1 hour, 178 ppm for 1 hour, and 400 ppm for 30 minutes. In humans, the estimated LC_{50} is 50 ppm; no duration is specified (RTECS 1990). In one inhalation study, all rats exposed to 800 ppm for 15 minutes died, but most rats survived exposure to this concentration when the duration was reduced to 13 minutes. Acutely poisoned animals showed severe inflammation of the mucous membranes, lacrimation, ulceration of the cornea, and burns of the skin in exposed areas (Proctor, Hughes, and Fischman 1988, p. 131; ACGIH 1986, p. 119). Two dogs and 20 rats exposed to chlorine trifluoride at a concentration of approximately 1.17 ppm for 6 hours a day, 5 days/week for 6 months experienced severe pulmonary irritation, and some of the animals died (ACGIH 1986, p. 119). Although information on human exposure to chlorine trifluoride is lacking, it is believed that exposure would result in severe irritation or burns of the eyes and skin and that inhalation would cause pulmonary edema (Proctor, Hughes, and Fischman 1988, p. 131).

Based on this evidence, OSHA preliminarily concludes that, in the absence of an exposure limit, agricultural workers are at significant risk of experiencing irritation and burns of the eyes and skin, as well as pulmonary edema, if they are exposed to chlorine trifluoride even for a very brief time. The Agency believes that the proposed PEL of 0.1 ppm as a ceiling limit is necessary to reduce these significant risks among agricultural workers. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CHLOROACETALDEHYDE

CAS: 107–20–0; Chemical Formula: $ClCH_2CHO$

H.S. No. 2031

OSHA has no limit for chloroacetaldehyde in agriculture; however, the Agency's current PEL for this substance in general industry, construction, and maritime is 1 ppm as a ceiling limit. The 1987–1988 ACGIH TLV® for chloroacetaldehyde is 1 ppm as a ceiling limit; NIOSH has no REL for this substance. OSHA is proposing to establish a ceiling limit of 1 ppm for chloroacetaldehyde in agriculture. This is the limit recently established for this substance in general industry.

Chloroacetaldehyde is used as a fungicide, to help facilitate the removal of bark from trees, and as a chemical intermediate in the manufacture of 2-aminothiazole and other compounds (Hawley's 1987, p. 280; Proctor, Hughes, and Fischman 1988, p. 132). Chloroacetaldehyde is a clear, colorless liquid with a very sharp, irritating odor (Hawley's 1987, p. 280). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Chloroacetaldehyde is corrosive and severely irritating to the eyes, skin, and mucous membranes of both humans and animals (Hawley's 1987, p. 280; ACGIH 1986, p. 120). The oral LD_{50} in rats is 75 mg/kg and the dermal LD_{50} in rabbits is

224 mg/kg (RTECS 1990). A solution of 30 percent chloroacetaldehyde in water produced severe damage when applied to the skin and eyes of rabbits (Proctor, Hughes, and Fischman 1988, p. 132). An inhalation study in rats showed that exposure to a 5-ppm concentration of chloroacetaldehyde (for an unspecified time) caused eye and nasal irritation (ACGIH 1986, p. 120).

In humans, repeated or prolonged exposures to concentrations that cause even slight irritation may result in injury (ACGIH 1986, p. 120). If a strong solution of chloroacetaldehyde contacts the eye, it is likely to cause permanent tissue damage and visual impairment (Proctor, Hughes, and Fischman 1988, p. 132; ACGIH 1986, p. 120). Severe burns may result from a single brief contact of the skin with a 40-percent aqueous solution of chloroacetaldehyde (ACGIH 1986, p. 120).

Based on this evidence, OSHA preliminarily concludes that agricultural workers exposed to chloroacetaldehyde at the levels permitted by the absence of a limit are at significant risk of experiencing irritation of the eyes, skin, and mucous membranes. The Agency believes that establishing a PEL of 1 ppm as a ceiling limit is necessary to protect workers in agriculture from these significant risks, which represent material impairments of health within the meaning of the Act. This is the limit recently established for this substance in general industry.

alpha-CHLOROACETOPHENONE

CAS: 523–27–4; Chemical Formula: $C_6H_5COCH_2Cl$

H.S. No. 2032

OSHA's PEL for alpha-chloroacetophenone in general industry, construction, and maritime is 0.05 ppm as an 8-hour time-weighted average (TWA); there is no PEL in agriculture. The 1987–1988 ACGIH TLV®–TWA for alpha-chloroacetophenone is 0.05 ppm as an 8-hour TWA; there is no NIOSH REL for this substance. OSHA is proposing an 8-hour TWA PEL of 0.05 ppm for alpha-chloroacetophenone in agriculture. This is the limit recently established for this substance in general industry.

alpha-Chloroacetophenone is a crystalline substance that is colorless to grey in appearance; at very low concentrations, it has a floral (apple blossom-like) odor (ACGIH 1986, p. 121; Gosselin, Smith, and Hodge 1984, p. II–96). It is a chemical warfare agent and is the main ingredient in the chemical mace (used for riot control and personal protection). This substance is also used as an intermediate in the pharmaceutical industry and formerly found use as an alcohol denaturant (Proctor, Hughes, and Fischman 1988, p. 132; HSDB 195; Hawley's 1987, p. 261).

alpha-Chloroacetophenone is a severe irritant and lacrimator in both animals and humans; exposure to high concentrations also can cause pulmonary edema (ACGIH 1986, p. 121). The oral LD_{50} in rats is 50 mg/kg; the lowest lethal concentration reported in the same species is 417 mg/m³ (approximately 70 ppm) for 15 minutes (RTECS 1990). Rabbits exposed dermally to a 12-percent solution of alpha-chloroacetophenone exhibited moderate skin irritation (RTECS 1990). When applied either topically or intradermally, alpha-chloroacetophenone caused contact sensitization in guinea pigs (HSDB 1985). Instilled into rabbit eyes, this substance produced mild to severe irritation, depending on the amount dropped into the eye (RTECS 1990). The eyes of laboratory animals exposed to high (not further specified) concentrations of alpha-chloroacetophenone vapor showed permanent opacification, ulceration with vascularization, and perforations (Gosselin, Smith, and Hodge 1984, p. II–97).

Humans acutely exposed to alpha-chloroacetophenone (200 to 340 mg/m³, or approximately 32 to 54 ppm) for 30 seconds experienced a burning sensation in the eyes, tearing, blurred vision, nasal irritation, runny nose, and a burning sensation in the throat (Proctor, Hughes, and Fischman 1988, p. 132). Contact with the eye can result in permanent partial opacity, reversible corneal haziness, and ocular pain (ACGIH 1986, p. 121; Gosselin, Smith, and Hodge 1984, p. II–96). Contact of this substance with the skin may result in burning and irritation (especially if the skin is moist), allergic contact dermatitis, and skin sensitization (ACGIH 1986, p. 121; Proctor, Hughes, and Fischman 1988, p. 132). Pulmonary edema caused the death of one individual exposed by inhalation to a high concentration (not further specified) of alpha-chloroacetophenone (Proctor, Hughes and Fischman 1988, p. 132). The use of alpha-chloroacetophenone as a riot-control agent has caused several deaths; the estimated lethal concentration in humans is 850 mg/m³ (approximately 134 ppm) for 10 minutes (ACGIH 1986, p. 121; Gosselin, Smith, and Hodge 1984, p. II–96).

Based on this evidence, OSHA preliminarily concludes that agricultural workers exposed to alpha-chloroacetophenone at the levels permitted by the absence of an exposure limit are at significant risk of experiencing eye, skin, and respiratory tract irritation, dermatitis, and contact sensitization. Workers exposed to high concentrations may also be at risk of experiencing pulmonary edema. The Agency believes that establishing a PEL of 0.05 ppm as an 8-hour TWA will protect workers in agriculture from these significant occupational risks which constitute material health impairments within the meaning of the Act. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CHLOROACETYL CHLORIDE

CAS: 79–04–9; Chemical Formula: $ClCH_2COCl$

H.S. No. 1083

There is no PEL for chloroacetyl chloride in the agriculture, construction, or maritime industries. The 1987–1988 ACGIH TLV®–TWA for this substance was 0.05 ppm (0.23 mg/m³). There is no NIOSH REL for chloroacetyl chloride; however, NIOSH concurs with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 0.5 ppm for chloroacetyl chloride in the agricultural, construction, and maritime industries; this is the limit recently established by OSHA for this substance in general industry.

Chloroacetyl chloride is a colorless liquid with a pungent odor (ACGIH 1986, p. 123(89)). It is used in the production of chloroacetophenone and tear gas (ACGIH 1986, p. 123(89); Hawley's 1987, p. 261).

In addition to sensory irritation, chloroacetyl chloride may cause burns of the eyes and skin. The LD_{50} in rats is 120 mg/kg, and the LC_{50} in the same species is 1000 ppm for 4 hours (RTECS 1989). The dermal LD_{50} in rabbits ranges from 316 to 501 mg/kg (Torkelson 1956). The minimum concentration of chloroacetyl chloride lethal to mice exposed for 2 hours is 2500 ppm. Before death, acutely poisoned animals became agitated and showed signs of severe eye and respiratory tract irritation (Herzog 1959). Inhalation of 4 ppm for 5 to 10 minutes caused respiratory problems in rats; however, no effect was observed in these animals when they inhaled 2.5 ppm for a period of 7 hours (Dow Chemical Company 1977a, as cited in ACGIH 1986/Ex. 1–3, p. 122). Thirty-day inhalation studies with rats, mice, and hamsters showed that these animals developed eye and respiratory irritation at 2.5 ppm but showed no effects at a 0.5-ppm concentration (Dow Chemical Company 1977a, as cited in ACGIH 1986/Ex. 1–3, p. 122).

The acute effects associated with exposure to chloroacetyl chloride in

humans include mild to moderate skin burns and erythema, eye burns and tearing, cough, and dyspnea. Eye and respiratory irritation occurred in an industrial setting characterized by a chloroacetyl chloride concentration of 0.009 to 0.017 ppm, with excursions as high as 0.14 ppm (Dow Chemical Company 1977a, as cited in ACGIH 1986/Ex. 1–3, p. 122). A worker accidentally drenched with a mixture containing chloroacetyl chloride developed extensive first- and second-degree burns and pulmonary edema and experienced three episodes of cardiac arrest, followed by coma and anoxia-induced brain damage (Pagnotto 197, as cited in ACGIH 1986/Ex. 1–3, p. 122). Other ingredients of the mixture involved in the accident included xylidine, benzene, and sodium carbonate. Rescuers of this victim experienced hand blisters, chest tightness, and nausea for two days.

Based on this evidence, OSHA preliminarily concludes that an 8-hour TWA limit of 0.05 ppm for chloroacetyl chloride is necessary to protect employees in the agriculture, construction, and maritime industries from the significant risk of eye, skin, and respiratory irritation and burns of exposed tissue associated with exposure to this substance. OSHA determined in the recent general industry rulemaking that this limit will substantially reduce this risk of material health impairment and believes that the same PEL is necessary to protect workers in agriculture, construction, and maritime. Accordingly, OSHA is proposing a PEL of 0.05 ppm as an 8-hour TWA for chloroacetyl chloride in these sectors. Promulgation of this PEL will make OSHA's limit for this substance consistent across all regulated sectors.

o-CHLOROBENZYLIDENE MALONONITRILE

CAS: 2698–41–1; Chemical Formula: $ClC_6H_4CH=C(CN)_2$

H.S. No. 1084

OSHA's PEL for o-chlorobenzylidene malononitrile (OCBM) in the construction and maritime industries is 0.05 ppm as an 8-hour TWA. There is no PEL for this substance in agriculture. The ACGIH TLV®–TWA for o-chlorobenzylidene malononitrile is 0.05 ppm as a ceiling, with a skin notation. There is no NIOSH REL for this substance. OSHA is proposing a ceiling limit of 0.05 ppm, with a skin notation, for OCBM in the agricultural, construction, and maritime industries. NIOSH concurred with this limit when the Agency recently established it in general industry.

o-Chlorobenzylidene malononitrile is a white, crystalline solid with a pepper-like odor (ACGIH 1986, p. 124). It is used as an incapacitating agent by military and law enforcement personnel (ACGIH 1986, p. 124; Hawley's 1987, p. 263).

In addition to sensory irritation, this substance causes dermatitis and skin sensitization in humans. The oral LD_{50} in rats is 187 mg/kg, and the lowest lethal concentration in the same species is 1808 mg/m³ (225 ppm) for 45 minutes (RTECS 1991). In animals, OCBM is metabolized by the body into cyanide (Frankenberg and Sorbo 1973/Ex. 1–480). Death caused by inhalation exposure is a result of lung damage, with broncho pneumonia and asphyxiation. Twenty rats exposed to a 2700-mg/m³ (337 ppm) concentration of OCBM for 1 hour died (Proctor, Hughes, and Fischman 1988, p. 134). Short-term exposures to high levels of OCBM did not cause carcinogenic, teratogenic, or embryolethal effects in animals (McNamara et al. 1973). Applied to rabbit eyes, OCBM causes severe irritation (RTECS 1991).

In humans, OCBM has extremely irritating properties. It causes intense eye and skin irritation, coughing, difficulty in breathing, chest tightness, rhinorrhea, dizziness, nausea, and vomiting. These effects are evident on exposure to OCBM concentrations between 12 and 20 mg/m³ (1.5 to 2.5 ppm), and they become incapacitating within 20 seconds of the onset of such exposures; the effects persist for approximately 5 to 10 minutes after the exposed individual has been moved to fresh air (Military Chemistry and Chemical Agents 1963, as cited in ACGIH 1986/Ex. 1–3, p. 124). Three of four human volunteers exposed to a 1.5-mg/m³ (0.19-ppm) concentration of OCBM aerosol for 90 minutes developed headaches, and one showed mild eye and nose irritation. Headache persisted for 24 hours in two subjects. At a concentration of 4 to 5 mg/m³ (0.5 to 0.6 ppm), subjects' problem-solving abilities were affected and they developed conjunctivitis, eye tearing, and skin burns (Punte, Owens, and Gutentag 1963/Ex. 1–353). Other researchers observed no persistent clinical abnormalities in seven subjects exposed to OCBM at concentrations ranging from 1 to 13 mg/m³ (0.13 to 1.6 ppm) over a 15-day period. Twenty-five of 28 workers in an OCBM production plant reported having had repeated episodes of dermatitis of the arms and neck, and two of these workers had positive patch tests when challenged with OCBM (Shmunes and Taylor 1973/Ex. 1–370).

Based on this evidence, OSHA is proposing a PEL of 0.05 ppm as a ceiling, with a skin notation, for o-chlorobenzylidene malononitrile; this is the limit recently established for this substance in general industry. The Agency preliminarily concludes that this limit is necessary to substantially reduce a significant risk of severe eye and upper respiratory tract irritation, skin sensitization, and dermatitis among workers in construction, maritime, and agriculture. OSHA considers these exposure-related health effects material impairments of health within the meaning of the Act. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CHLOROPICRIN

CAS: 76–06–2; Chemical Formula: CCl_3NO_2

H.S. No. 2037

OSHA has no limit for chloropicrin in agriculture; however, the Agency's current PEL for this substance in construction, general industry, and maritime is 0.1 ppm as an 8-hour time-weighted average (TWA). The 1987–1988 ACGIH TLV®–TWA is also 0.1 ppm; there is no NIOSH REL for this substance. The Agency is proposing an 8-hour TWA of 0.1 ppm for chloropicrin in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all industry sectors.

Chloropicrin is an oily, colorless to faint-yellow liquid with an intense odor (HSDB 1985). Chloropicrin is used as an insecticide, fungicide, herbicide in soil, and as a stored grain fumigant to control insects and rodents. It is also used as a tear gas, in the synthesis of methyl violet, and as an additive to increase the odor warning properties of other chemicals (HSDB 1985; ACGIH 1986, p. 134; Hawley's 1987, p. 271). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Chloropicrin is a severe irritant of the eyes, mucous membranes, skin, and upper respiratory tract (HSDB 1985; ACGIH 1986, p. 134; Proctor, Hughes, and Fischman 1988, p. 149). The oral LD_{50} in rats is 250 mg/kg; the LC_{50} in mice is 1600 mg/kg (approximately 230 ppm) for 10 minutes (RTECS 1990; ACGIH 1986, p. 134). Acutely poisoned animals showed congestion, hemorrhage, lung edema, and inflammation before death (ACGIH 1986, p. 134). Necrosis of the liver, kidneys, and skeletal muscles was seen in these animals after chronic exposure (ACGIH 1986, p. 134). Exposure to a 9-ppm concentration of chloropicrin

caused a 50-percent decrease in respiratory rate; at autopsy, ulceration and necrosis of the respiratory tract lining, pulmonary edema, and moderate lung damage were seen (Proctor, Hughes, and Fischman 1988, p. 149). A carcinogenicity bioassay of chloropicrin found no significant incidence of tumors in rats or mice (HSDB 1985).

In humans, the lethal dose is estimated to be 119 ppm for 30 minutes; death in these cases is caused by pulmonary edema (Proctor, Hughes, and Fischman 1988, p. 149). Exposure to chloropicrin vapor concentrations ranging from 0.30 to 0.37 ppm causes painful irritation and tearing of the eyes in 3 to 30 seconds, and inhalation of a 7.5-ppm concentration for 10 minutes or of 15 ppm for 1 minute is described by exposed individuals as intolerable (Clayton and Clayton 1981, p. 4165; ACGIH 1986, p. 134). Accidental ingestion of the liquid causes gastroenteritis, severe nausea, vomiting, colic, and diarrhea (HSDB 1985).

Based on this evidence, OSHA preliminarily concludes that agricultural workers exposed to chloropicrin at the levels permitted by the absence of a limit are at significant risk of experiencing irritation of the eyes, skin, mucous membranes, and lungs. The Agency believes that establishing a PEL for chloropicrin of 0.1 ppm as an 8-hour TWA is necessary to substantially reduce these risks in agricultural workplaces. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

COPPER (DUSTS AND MISTS)
CAS: 7440–50–8; Chemical Formula: Cu
H.S. No. 2041

In general industry, construction, and maritime, OSHA's permissible exposure limit for copper dusts and mists is 1 mg/m^3 as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA for copper dusts and mists of 1 mg/m^3 as an 8-hour limit. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing to establish an 8-hour TWA limit of 1 mg/m^3 for copper dusts and mists in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Copper is an odorless, reddish brown, ductile metal or powder. Copper is widely used as a structural metal in electric wiring, switches, heating, plumbing, roofing, building construcyion, alloys, coins, insecticides, chemical and pharmaceutical machinery, electroplated protective coatings and undercoats, antifouling paints, and as a catalyst (Genium MSDS 1990, No. 162; ACGIH 1986, p. 146).

Exposure to copper dust or mist causes eye, skin, and respiratory tract irritation, as well as liver, kidney, and hematological effects. The lowest toxic oral dose for copper dust in rats is 1210 mg/kg (RTECS 1990). In mice, the intra peritoneal LD_{50} for the dust is 3.5 mg/kg (Clayton and Clayton 1981, p. 1623). Signs of acute copper poisoning in animals include nausea, vomiting, salivation, purgation, tachycardia, convulsions, paralysis, and collapse, followed by death. Autopsy of acutely poisoned animals shows gastroenteritis and congestion of the spleen, liver, and kidneys (Clarke 1981, p. 45, in HSDB 1985). Chronic ingestion of copper by animals leads to a pigmentary cirrhosis of the liver (Mallory and Parker 1931; Wiederanders et al. 1968, in Gosselin 1984, p. III–122). The offspring of rats given copper orally several weeks before mating showed embryotoxic and developmental effects (RTECS 1990).

In humans, exposure to copper dusts and mists is associated with irritation of the eyes and respiratory tract. If the exposure is prolonged or severe, nasal ulceration and perforation of the septum may occur. The lowest toxic oral dose of copper in humans is reported to be 120 μg/kg; at this dose, gastrointestinal effects were seen (RTECS 1990). Acute copper poisoning causes jaundice and tenderness in the area of the liver (Webster 1930; Chuttani et al. 1965, in Gosselin 1984, p. III–121). Greenish discoloration of the skin and hair have been reported in workers exposed to copper dust and mist (Saltzer and Wilson 1968; Stokinger 1963, in Proctor and Hughes 1978, p. 181). Allergic contact dermatitis is rare among copper workers, but this condition has been reported occasionally (Adams, R. M., ed. Occupational Skin Disease 1983). The main effects from industrial exposure to copper dust and mist involve the respiratory tract; symptoms of acute overexposure include chills, fever, aching muscles, dry mouth and throat, and headache (Clayton and Clayton 1981, p. 1628). Three men who were exposed to fine copper dust at concentrations of 0.075 to 0.120 mg/m^3 suffered from apparent metal fume fever (Proctor and Hughes 1978, p. 181). Seven cases of copper fever were reported in workers in a paint factory where copper oxide was being pulverized (Shiötz 1949, in Clayton and Clayton 1981, p. 1628). Copper dust exposure may also cause systemic effects similar to those caused by other heavy metal exposure such as capillary damage, kidney and liver injury, and central nervous system excitation followed by depression (Gosselin 1984, p. III–121). Chronic exposure to copper may cause anemia, widespread damage to the small blood vessels, and injury to the liver and kidney (Gosselin, Smith, and Hodge 1984, p. III–121; ACGIH 1986, p. 146).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to copper dusts and mists causes eye, skin, and respiratory tract irritation, kidney and liver damage, and circulatory system effects in humans and animals. OSHA therefore believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed limit for copper dusts and mists of 1 mg/m^3 as an 8-hour TWA in agriculture is necessary to significantly reduce these risks of material health impairment. Promulgation of this limit also will make the PEL for copper dust and mist consistent across all OSHA-regulated sectors.

COPPER (FUME)
CAS: 7440–50–8; Chemical Formula: Cu
H.S. No. 1101

OSHA's limit for copper fume in general industry, construction, and maritime is 0.1 mg/m^3 as an 8-hour TWA. The ACGIH TLV® is 0.2 mg/m^3 as an 8-hour TWA. NIOSH has no REL but concurs (Ex. 8–47, Table N1) that the proposed 0.1-mg/m^3 limit in agriculture is appropriate. Promulgation of this PEL will make OSHA's limit for copper fume consistent across all regulated sectors.

There are few toxicological data available on copper fume's effects in experimental animals. The oral LD_{50} for cupric oxide (finely divided copper dust) is 470 mg/kg (Clayton and Clayton 1981, p. 1624).

In humans, exposure to copper fume causes metal fume fever. The signs and symptoms of this syndrome include fever lasting for as long as 48 hours, chills, aching muscles, and headaches. Welders exposed chronically to copper fumes experienced sneezing, cough, fever, atrophy and ulceration of the nasal mucosa, and a metallic taste (Clayton and Clayton 1981, p. 1628). Workers exposed to copper fume at a concentration of 1 to 3 mg/m^3 experienced an altered taste response (ACGIH 1986, p. 146). When exposures were reduced to between 0.02 and 0.04 mg/m^3, this effect disappeared (ACGIH 1986, p. 146).

Based on this evidence, OSHA preliminarily concludes that exposure to copper fume is associated with metal fume fever; OSHA considers this condition a material impairment of health. The Agency believes that the

proposed PEL of 0.1 mg/m³ will substantially reduce the risk that workers in agriculture will experience metal fume fever. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CYANOGEN

CAS: 460–19–5; Chemical Formula: $(CN)_2$
H.S. No. 1105

OSHA currently has a TWA PEL of 10 ppm for cyanogen in general industry and in the construction and maritime industries. The Agency has no PEL for cyanogen in agriculture. The ACGIH has a TLV®–TWA of 10 ppm for this substance; there is no NIOSH REL, although NIOSH concurs with the limit being proposed in agriculture, which is 10 ppm as an 8-hour TWA. This action will make the PEL for cyanogen consistent across all OSHA-regulated sectors.

Cyanogen is a colorless gas with an almond-like odor. It is used as a fumigant and chemical intermediate, an ingredient in rocket and missile propellants, and in the welding and cutting of heat-resistant metals (Braker and Mossman 1980, p. 1986). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Cyanogen is an irritant of the eyes, mucous membranes, and respiratory tract in both animals and humans; severe exposure may cause pulmonary edema (ACGIH 1986, p. 154). Cyanogen's effects resemble those of other cyanides (Clayton and Clayton 1981, p. 4858). The LC_{50} in rats is 350 ppm for 1 hour; acutely poisoned animals showed eye and pulmonary system effects and behavioral changes (RTECS 1991). The acute toxicity of cyanogen in various animal species is high (Flury and Zernik 1931d). Exposure to a 100-ppm concentration was fatal to cats in 2 to 3 hours, and exposure to 400 ppm was fatal to rabbits in less than 2 hours. Cats exposed to a 50-ppm concentration were severely affected but subsequently recovered (Flury and Zernik 1931d). Experiments in rats suggest that cyanogen is approximately 10 times less acutely toxic than hydrogen cyanide (McNerney and Schrenk 1960/Ex. 1–426). Monkeys and rats exposed to cyanogen at concentrations of 11 or 25 ppm for 6 hours/day, 5 days/week for 6 months showed a reduction in lung moisture content (in both groups of monkeys) and in body weight (rats in the 25-ppm group); the authors concluded that subchronic exposure to 25 ppm is marginally toxic (Lewis, Anger, and TeVault 1984, in J. Environ. Path. Tox. Oncol. 5:151–163).

Human studies showed that volunteers experienced almost immediate eye and nasal irritation on exposure to a cyanogen concentration of 16 ppm for 6 minutes (McNerney and Schrenk 1960/Ex. 1–426). When the concentration was reduced to 8 ppm, no effects were reported (McNerney, Schrenk 1960/Ex. 1–426).

OSHA is proposing an hour TWA limit of 10 ppm for cyanogen in the agriculture industry. The Agency preliminarily concludes that this limit is necessary to protect workers in this sector against the significant risk of irritation and systemic effects associated with exposure to cyanogen. OSHA believes that this limit will protect workers in this sector from experiencing these material impairments of health. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CYANOGEN CHLORIDE

CAS: 506–77–4; Chemical Formula: ClCN
H.S. No. 1106

OSHA has no limit for cyanogen chloride in the construction, agriculture, or maritime industries. The ACGIH has a TLV® of 0.3 ppm as a ceiling; NIOSH has no REL for this substance but concurs (Ex. 8–47) with the limit of 0.3 ppm (ceiling) being proposed for cyanogen chloride in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Cyanogen chloride is a colorless liquid or gas (depending on temperature and pressure) that has a pungent odor. It is used in organic synthesis and has been used by the military as a poison gas (ACGIH 1986, p. 155). Cyanogen chloride is also used as a fumigant, as an ingredient in metal cleaners, in the refining of ore, and in the production of synthetic rubber (Sittig 1985, p. 276). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Cyanogen chloride is an irritant of the eyes, mucous membranes, and respiratory tract in both humans and animals; severe exposure may cause pulmonary edema and central nervous system effects (ACGIH 1986, p. 155). The lethal effects of cyanogen chloride are caused by the formation of hydrocyanic acid (Aldridge and Evans 1946/Ex. 1–708). The LC_{50} in monkeys is 4400 mg/m³ for 1 minute (RTECS 1989). Flury and Zernik (1931d) observed the effects of exposure to cyanogen chloride in five animal species. In mice, exposure to a concentration of approximately 500 ppm was fatal within 3 minutes; in cats, 120 ppm was fatal in 3.5 minutes; 48 ppm was fatal to dogs in 6 hours; in goats, a 1000-ppm exposure for 3 minutes caused death after 70 hours; and 1200 ppm was fatal to the rabbit. Other studies have demonstrated that animals exposed to cyanogen chloride develop pulmonary edema; interference with cellular metabolism is also an effect of exposure (Jandorf and Bodansky 1946/Ex. 1–334; Aldridge and Evans 1946/Ex. 1–708).

Human data indicate that 1 ppm is the lowest irritant concentration of cyanogen chloride that can be tolerated for a 10-minute exposure; exposure to a 2-ppm concentration was intolerable for this time period, and 4 ppm was fatal in 30 minutes (Prentiss 1937/Ex. 1–1164). The Michigan Department of Health (1977, as cited by ACGIH 1986/Ex. 1–3, p. 155) reported that exposure to a cyanogen chloride concentration of about 0.7 ppm caused eye and nasal irritation severe enough to force workers to evacuate the area. Workers in a cyanogen chloride manufacturing facility reported experiencing the following symptoms of acute overexposure: Dizziness, blurred vision, and nausea (Reed 1920). The signs and symptoms of chronic exposure to this substance included weight loss, muscular weakness, lassitude, skin irritation, and lung congestion (Reed 1920).

OSHA is proposing a 0.3-ppm ceiling limit for cyanogen chloride in the construction, agriculture, and maritime industries; this is the limit recently established in general industry. The Agency preliminarily concludes that this limit is necessary to substantially reduce the significant risk of material health impairment (irritation and systemic poisoning) potentially associated with exposure to this substance in agriculture. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CYCLOHEXANE

CAS: 110–82–7; Chemical Formula: C_6H_{12}
H.S. No. 2046

In general industry, construction, and maritime, OSHA currently has an hour time-weighted average (TWA) limit of 300 ppm for cyclohexane. The Agency has no permissible exposure limit (PEL) for this substance in agriculture. The 1987–1988 ACGIH TLV®–TWA is 300 ppm as an 8-hour TWA; NIOSH has no REL. OSHA is proposing an 8-hour TWA of 300 ppm for cyclohexane in agriculture. Promulgation of this limit

will make the PEL for cyclohexane consistent across all regulated sectors.

Cyclohexane is a colorless, mobile liquid at room temperature, and it has a pungent odor that is detectable at a concentration of 300 ppm (Hawley's 1987, p. 335; ACGIH 1986, p. 156). Cyclohexane is used in fungicidal formulations and as a solvent for lacquers, resins, synthetic rubber, fats, and waxes. It is also used as an intermediate in the manufacture of a variety of chemicals and in the industrial recrystallization of steroids (HSDB 1987). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Cyclohexane causes moderate irritation of the eyes and mucous membranes; at high concentrations it causes central nervous system depression (narcosis) in laboratory animals. In animals, the acute toxicity of this substance is low; rabbits exposed to a concentration of 18,500 ppm for 8 hours survived (ACGIH 1986, p. 156). A 1-hour exposure to 28,752 ppm was lethal to all rabbits; acutely poisoned animals showed narcosis, tremor, and, in some animals, tetanic spasm before death (Proctor, Hughes, and Fischman 1988, p. 170). The oral LD_{50} in rats is 12,705 mg/kg (RTECS 1990). At autopsy, generalized vascular damage and severe degenerative changes in the heart, lungs, liver, kidneys, and brain were seen (Proctor, Hughes, and Fischman 1988, p. 170).

Humans exposed to airborne cyclohexane concentrations of 300 ppm report experiencing eye and mucous membrane irritation (ACGIH 1986, p. 156). Repeated contact of the skin with cyclohexane causes defatting (Proctor, Hughes, and Fischman 1988, p. 170). There have been no reports of systemic poisoning in humans as a result of cyclohexane exposure (Gosselin, Smith, and Hodge 1984, p. II–151).

Based on this evidence, OSHA preliminarily concludes that, in the absence of a PEL, workers in agriculture are at significant risk of experiencing eye and mucous membrane irritation and that they may also be at significant risk of experiencing central nervous system depression. The Agency believes that establishing an 8-hour TWA of 300 ppm for cyclohexane in agriculture is necessary to substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CYCLOHEXENE
CAS: 110–83–8
H.S. No. 2047

In general industry, construction, and maritime, OSHA's permissible exposure limit for cyclohexene is 300 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV–TWA* of 300 ppm for this substance; NIOSH has no REL. OSHA is proposing to apply an 8-hour TWA PEL of 300 ppm for cyclohexene in agricultural workplaces. This is the limit recently established for this substance in general industry.

Cyclohexene is a colorless liquid that is used in organic synthesis, in oil extraction, and as a solvent (ACGIH 1986, p. 160). This substance also finds use as a stabilizer for high-octane gasoline (Clayton and Clayton 1982, p. 3233).

Cyclohexene is an eye, skin, and respiratory tract irritant; at high concentrations, this substance also causes central nervous system depression in laboratory animals (Sittig 1985, p. 281; Proctor, Hughes, and Fischman 1988, p. 172). In direct contact with the skin, liquid cyclohexene is a defatting agent (Sittig 1985, p. 281; Proctor, Hughes, and Fischman 1988, p. 172). Dogs exposed by inhalation to an unspecified concentration of cyclohexene showed muscular quivering and staggering gait (Clayton and Clayton 1982, p. 3233). Mice lost their righting reflex after exposure to a cyclohexene concentration of about 9000 ppm, and a single exposure to 15,000 ppm was lethal (Proctor, Hughes, and Fischman 1988, p. 172). In a 6-month inhalation study of rats, rabbits, and guinea pigs that involved repeated exposures to 75, 150, 300, or 600 ppm cyclohexene, all rats showed an increase in alkaline phosphatase, and the high-dose rats gained weight more slowly than did controls; no other significant exposure-related effects were seen (Clayton and Clayton 1982, p. 3233; Proctor, Hughes, and Fischman 1988, p. 172). Cyclohexene is believed to have relatively low toxicity in mammals because it is rapidly metabolized and eliminated (Clayton and Clayton 1982, p. 3233).

Although no toxic effects have been reported in humans exposed to cyclohexene, this substance is regarded as a mild respiratory irritant and central nervous system depressant based on effects seen in animals (Proctor, Hughes, and Fischman 1988, p. 172; ACGIH 1986, p. 160).

Based on this evidence, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at a significant risk of experiencing the eye, skin, and respiratory irritation associated with exposure to cyclohexene and that they may also be at significant risk of experiencing central nervous system depression. The Agency believes that establishing an 8-hour TWA PEL of 300 ppm will substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CYCLOPENTADIENE
CAS: 542–92–7; Chemical Formula: C_5H_6
H.S. No. 2048

OSHA's current PEL for cyclopentadiene in general industry, construction, and maritime workplaces is 75 ppm as an 8-hour time-weighted average (TWA). OSHA has no PEL for this substance in agriculture. The 1987–1988 ACGIH TLV®–TWA was 75 ppm for this substance; there is no NIOSH REL for cyclopentadiene. OSHA is proposing an 8-hour TWA of 75 ppm for cyclopentadiene in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all regulated sectors.

Cyclopentadiene is a volatile, colorless liquid that polymerizes easily on standing to the dimer dicyclopentadiene. This substance has an irritating odor that is similar to that of terpene (ACGIH 1986, p. 163). Cyclopentadiene is used as a starting material for chlorinated insecticides, synthetic prostaglandin, and the formation of sandwich compounds by chelation. Cyclopentadiene is also used in the manufacture of resins and in organic synthesis (HSDB 1986; Hawley's 1987, p. 338).

Cyclopentadiene causes irritation, dermatitis, and sensitization in humans; in laboratory animals, it has been reported to cause injury to the liver and kidneys as well as irritation and narcosis. The oral LD_{50} for the dimer in rats is 0.82 g/kg, and the LC_{50} in the same species is 39 g/m^3 for the monomer (Clayton and Clayton 1981, p. 3239; RTECS 1990). The dermal LD_{50} in rabbits is 6.7 ml/kg (Clayton and Clayton 1982, p. 3239). Subcutaneous injection of 3.0 ml cyclopentadiene caused narcosis and fatal convulsions in rabbits (Clayton and Clayton 1982, p. 3239). Mild injury of the liver and kidneys was seen at autopsy in rats exposed to a 500-ppm concentration of cyclopentadiene for 7 hours/day for 35 days (over a period of 53 days) (Proctor, Hughes, and Fischman 1988, p. 174). No ill effects were noted by observation, clinical tests, or histologic examination in dogs exposed to a 400-ppm concentration for 6 hours, for 39 episodes, followed by 15 exposures to 00

ppm (Proctor, Hughes, and Fischman 1988, p. 174).

Humans exposed to cyclopentadiene at concentrations of 250 and 500 ppm experienced a sensory response described as distinctly unfavorable (ACGIH 1986, p. 163). Cyclopentadiene has caused contact dermatitis and sensitization in humans (Grant 1974, p. 343).

Based on this evidence, OSHA preliminarily concludes that agricultural workers exposed to this substance at the levels permitted by the absence of a limit are at significant risk of experiencing irritation and of developing dermatitis and, in some cases, sensitization. The Agency believes that establishing a PEL of 75 ppm as an 8-hour TWA will protect workers in agriculture from these significant risks, which are material impairments of health within the meaning of the Act. In addition, promulgation of this limit with make OSHA's Pel for this substance consistent across all regulated sectors.

DIACETONE ALCOHOL

CAS: 123–42–2; Chemical Formula: $(CH_3)_2C(OH)CH_2COCH_3$

H.S. No. 2052

OSHA's PEL for diacetone alcohol in general industry, construction, and maritime workplaces is 50 ppm as an 8-hour time-weighted average (TWA); there is no PEL in agriculture. The 1987–1988 ACGIH TLV®–TWA was 50 ppm, and this is also the NIOSH REL. OSHA is proposing an 8-hour TWA PEL of 50 ppm for diacetone alcohol in agriculture. Promulgation of this limit will make the PEL for diacetone alcohol consistent across all regulated sectors.

Diacetone alcohol is a colorless liquid with a faint and pleasant odor (HSDB 1990). It is used as a chemical intermediate in organic synthesis and as a solvent for cellulose compounds, fats, oils, waxes, resins, and certain pigments. In addition, it is used in some antifreeze solutions, hydraulic fluids, photographic films, and quick-drying inks and also finds use as a preservative in pharmaceuticals (HSDB 1990).

Diacetone alcohol is primarily an irritant of the eyes, mucous membranes, and respiratory tract; however, at high concentrations, it causes narcosis in laboratory animals. The oral LD_{50} in rats is 4000 mg/kg; the dermal LD_{50} in rabbits is 13,500 mg/kg; other studies indicated that an oral dose of 2 ml/kg produced transient liver damage in rats and that a dose of 4 ml/kg killed the animals (RTECS 1991; Clayton and Clayton 1982, p. 4755). Narcosis was produced in rabbits by single doses ranging from 2.4 to 4.0 ml/kg, and a dose of 5 ml/kg was lethal (Clayton and Clayton 1982, p. 4755). Exposure to 2100 ppm for 1 to 3 hours caused animals to exhibit signs of restlessness, mucous membrane irritation, and drowsiness (Proctor, Hughes, and Fischman 1988, p. 179). Application of pure diacetone alcohol to the skin of rabbits produced mild skin irritation, but moderate to severe eye irritation and transient corneal damage occurred when this substance was dropped into rabbit eyes (RTECS 1991; Clayton and Clayton 1982, p. 4755).

Most human subjects experienced eye, nose, and throat irritation during an exposure to a 100-ppm concentration of diacetone alcohol for 15 minutes. At the 100-ppm concentration, humans also experienced headaches, nausea, and vomiting (RTECS 1990). At a concentration of 400 ppm, these volunteers reported chest discomfort (RTECS 1991; Proctor, Hughes, and Fischman 1988, p. 178). Repeated or prolonged skin contact may cause defatting of the skin and dermatitis, and eye contact will cause moderate to marked irritation and transient corneal damage (Proctor, Hughes, and Fischman 1988, p. 178).

Based on this evidence, OSHA preliminarily concludes that agricultural workers exposed to this substance at the levels permitted by the absence of a limit are at significant risk of experiencing irritation of the eyes, mucous membranes, and respiratory tract. At high concentrations, they may also be at risk of experiencing narcotic effects. The Agency believes that establishing a PEL of 50 ppm as an 8-hour TWA for diacetone alcohol is necessary to protect workers in agriculture from these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DIAZOMETHANE

CAS: 334–88–3; Chemical Formula: CH_2N_2

H.S. No. 2053

In general industry, construction, and maritime, OSHA's permissible exposure limit for diazomethane is 0.2 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.2 ppm for this substance; NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL in agriculture of 0.2 ppm for diazomethane. This is the limit recently established for this substance in general industry.

Diazomethane is a yellow gas with a musty odor. Its only use is as a methylating agent in chemical laboratories (HSDB 1984; Clayton and Clayton 1981, p. 2784).

Exposure to diazomethane causes severe eye and respiratory tract irritation and skin and respiratory sensitization in both animals and humans; this substance has also produced cancer and teratogenic effects in animals. Inhalation exposure caused pulmonary edema, hemorrhagic emphysema, and bronchopneumonia in exposed cats, rabbits, and guinea pigs (IARC 1974, p. 226). Cats exposed to a 175-ppm concentration of diazomethane for 10 minutes died within 3 days of pulmonary edema and hemorrhage of the lungs (Clayton and Clayton 1981, p. 2785). In guinea pigs, repeated applications of diazomethane in a cottonseed oil or dioxane vehicle caused skin sensitization (Clayton and Clayton 1981, p. 2785). In skin painting and inhalation bioassays in mice and rats, diazomethane caused an increased incidence of lung tumors (IARC 1974, p. 228). Diazomethane is mutagenic in bacterial test systems (RTECS 1990).

In humans, diazomethane is a severe respiratory tract irritant and allergen that causes, even in very low concentrations, the following effects: coughing, fever, chest pains, cyanosis, fulminating pneumonia, and, occasionally, death. Exposure can lead to hypersensitivity, which is manifested as asthma and fever (Parmeggiani 1983, pp. 620–621). Two persons exposed to unspecified concentrations of diazomethane developed dizziness, headache, chest pain, fever, and severe asthmatic symptoms beginning about 5 hours after exposure. Other exposures to this substance have resulted in cyanosis, pulmonary edema, weakness, chest pain, pneumonia, and death (ACGIH 1986, p. 173). Hepatic enlargement and hemolysis also are reported to result from exposure to diazomethane (Clayton and Clayton 1981, p. 2785). Diazomethane is irritating to the eyes and could cause serious ocular damage, and skin contact with this substance leads to denudation of the skin and mucous membranes (Clayton and Clayton 1981, p. 2785).

Based on this evidence in humans and animals, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing the respiratory tract and other irritation associated with exposure to diazomethane. The Agency believes that establishing an 8-hour TWA PEL of 0.2 ppm is necessary to substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DIBUTYL PHOSPHATE

CAS: 107–66–4; Chemical Formula: $(n\text{-}C_4H_9O)_2(OH)PO$
H.S. No. 1119

OSHA currently has an 8-hour TWA PEL of 1 ppm for dibutyl phosphate in the construction and maritime industries. The Agency has no PEL in agriculture for this substance. The ACGIH has a TLV®–TWA of 1 ppm and a TLV®–STEL of 2 ppm for dibutyl phosphate; there is no NIOSH REL, but NIOSH concurs with the PELs being proposed (Ex. 8–47). The Agency is retaining the 8-hour TWA PEL in construction and maritime, is proposing a 2 ppm STEL in these two sectors, and is proposing both limits in agriculture. These are the limits recently established for this substance in general industry.

Dibutyl phosphate is a pale amber liquid that is used as an organic catalyst in the manufacture of phenolic and urea resins and as an antifoaming agent (ACGIH 1986, p. 175; Hawley's 1987, p. 372).

Dibutyl phosphate is an irritant of the eyes, mucous membranes, skin, and upper respiratory tract. The oral LD_{50} in rats is 3.2 g/kg (Proctor, Hughes, and Fischman 1988, p. 183). In a personal communication to the ACGIH, Mastromatteo reported that workers exposed to relatively low concentrations of dibutyl phosphate developed respiratory tract irritation and headache (Mastromatteo 1964a, as cited in ACGIH 1986/Ex. 1–3, p. 236). Workers exposed to an unspecified concentration of dibutyl phosphate also reportedly experienced headaches (ACGIH 1986, p. 175). Repeated contact of dibutyl phosphate with the skin causes dermatitis (Genium MSDS 1987, No. 613).

OSHA preliminarily concludes that both a TWA and a STEL are necessary to protect workers in construction, maritime, and agriculture from the significant risk of eye, skin, and respiratory tract irritation and headaches reported to occur among workers exposed to low concentrations of this substance. The Agency considers these exposure-related effects to be material impairments of health within the meaning of the Act. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

O–DICHLOROBENZENE
CAS: 95–50–1; Chemical Formula: $C_6H_4Cl_2$
H.S. No. 2057

In general industry, construction, and maritime, OSHA's current permissible exposure limit for o-dichlorobenzene is 50 ppm as a ceiling. There is no OSHA limit for o-dichlorobenzene in agriculture. The ACGIH has a ceiling limit for this substance of 50 ppm, with a skin notation; NIOSH has no REL. OSHA is proposing a 50 ppm ceiling limit for o-dichlorobenzene in agriculture; this action will ensure consistency in the exposure limit for this substance across all OSHA-regulated sectors.

o-Dichlorobenzene is a colorless to pale yellow liquid with a pleasant odor (Hazardous Substance Fact Sheet 1985, p. 1). There are many uses for o-dichlorobenzene; it is used as an insecticide against termites and locust borers; a fumigant; a degreasing agent; a heat transfer medium; to remove sulfur from illuminating gas; and in the manufacture of dyes (Merck 1983, p. 444). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In both humans and animals, o-dichlorobenzene is an irritant of the eyes, nose, upper respiratory tract, and skin; at high concentrations, this substance also causes central nervous system depression. In humans, exposure to high concentrations of o-dichlorobenzene has also caused blood dyscrasias. The oral LD_{50} in rats is 500 mg/kg (RTECS 1991). The lowest reported lethal concentration in rats is 4946 mg/m³ (821 ppm) for 7 hours (Hollingsworth et al. 1958, in IARC 1982, Vol. 29, p. 228). Rabbits experienced mild eye irritation after 100 mg of o-dichlorobenzene had been instilled into their eyes and was left for 30 seconds before being rinsed out (RTECS 1991). Repeated application of o-dichlorobenzene to the skin of rats caused death, indicating that this substance can be absorbed through the skin in lethal amounts (U.S. EPA 1985, in Proctor, Hughes, and Fischman 1988, p. 184). Rats exposed to a 977-ppm concentration of o-dichlorobenzene for 2 hours survived; however, exposure to the same concentration for 7 hours was fatal to these animals. No deaths occurred among rats exposed for 3 hours to a 539-ppm concentration of o-dichlorobenzene, but autopsy revealed marked centrilobular necrosis of the liver and cloudy swelling of the tubular epithelium of the kidney (Hollingsworth et al. 1958, in IARC 1982, Vol. 29). Liver damage was also reported in a study involving exposure to o-dichlorobenzene concentrations of 50 to 800 ppm for a few hours (Cameron and Thomas 1937, in ACGIH 1986, p. 178). Several species of experimental animals (rats, rabbits, and guinea pigs) showed no adverse effects from exposure to this substance at a concentration of 93 ppm for 7 hours/day for 6 or 7 months (Hollingsworth et al. 1958).

In humans, exposure to an o-dichlorobenzene concentration of 25 or 30 ppm causes eye irritation (AIHA 1964). After prolonged contact with the skin, o-dichlorobenzene causes blistering, and the skin may subsequently become pigmented; dermal sensitization has also been reported (Hollingsworth et al. 1958). Ten of 28 volunteers accidentally exposed to unspecified concentrations of o-dichlorobenzene for hours/day for 4 days experienced dizziness, severe headache, fatigue, and nausea, in addition to eye, nose, and throat irritation (Zapata-Gayon et al. 1982, in Proctor, Hughes, and Fischman 1988, p. 184). This study also reported significant alterations in the leukocytes of these individuals; these changes were not evident 6 months after exposure ended. A 40-year-old worker exposed for 22 years to o-dichlorobenzene (concentration unspecified) during the preparation of dyestuffs suffered from purpura, anemia, hepatomegaly, and splenic enlargement; death was attributed to o-dichlorobenzene-related proliferating myelosis (Tolot et al. 1969, in Clayton and Clayton 1981, p. 3615). Another 40-year-old worker developed chronic lymphatic leukemia after 10 years of occupational exposure to a solvent containing 80 percent o-, 15 percent p-, and 2 percent m-dichlorobenzene (Clayton and Clayton 1981, p. 3615).

Based on this evidence in humans and animals, OSHA preliminarily concludes that o-dichlorobenzene causes irritation of the eyes, nose, and skin, central nervous system depression, liver and kidney damage, and blood dyscrasias. OSHA therefore believes that, in the absence of a limit for o-dichlorobenzene, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed ceiling limit of 50 ppm is necessary to substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

1,3-DICHLORO-5,5-DIMETHYL HYDANTOIN
CAS: 118–52–5; Chemical Formula: $C_5H_6Cl_2N_2O_2$
H.S. No. 1122

OSHA currently has a limit of 0.2 mg/m³ as an 8-hour TWA for 1,3-dichloro-5,5-dimethyl hydantoin (DCDMH) in the construction and maritime industries. The Agency has no PEL for DCDMH in agriculture. The ACGIH has a TLV®–

TWA of 0.2 mg/m³ and a TLV®–STEL of 0.4 mg/m³ for this substance; there is no NIOSH REL. The Agency is proposing PELs of 0.2 mg/m³ as an 8-hour TWA and 0.4 mg/m³ as a 15-minute STEL for DCDMH in the construction, agriculture, and maritime industries. OSHA recently established these limits in general industry, and NIOSH (Ex. 8–47) concurs that these limits are appropriate for DCDMH. Promulgation of the PELs will make OSHA's limits for DCDMH consistent across all regulated sectors.

1,3-Dichloro-5,5-dimethyl hydantoin is a white powder with a slight chlorine-like odor. It is used as a chlorinating agent, a chemical intermediate in the production of insecticides, drugs, and amino acids, and as a stabilizer for vinyl chloride polymers (ACGIH 1986, p. 183).

1,3-Dichloro-5,5-dimethyl hydantoin produces systemic toxicity in laboratory animals and irritation of the respiratory tract in humans (Proctor, Hughes, and Fischman 1988, p. 188). The acute oral LD_{50} in rats of both sexes is 542 ±84 mg/kg when a 10-percent aqueous suspension of DCDMH is administered. At autopsy, rats dying within 48 hours of administration showed gastrointestinal hemorrhages (Industrial Bio-Test Laboratories 1961 and 1962, as cited in ACGIH 1986/Ex. 1–3, p. 183). The lowest lethal concentration in rats is 20 g/m³ for 4 hours (RTECS 1990). Applied to the skin of rabbits (100 mg) for 24 hours, DCDMH caused severe injury (RTECS 1990).

Limited exposure data for humans were reported by Baier, who stated that individuals experienced extreme respiratory irritation at an average DCDMH concentration of 1.97 mg/m³, but that some experienced this degree of irritation even at a concentration of 0.7 mg/m³ (Baier 1964, as cited in ACGIH 1986/Ex. 1–3, p. 183). In contact with the eyes, this substance forms hypochlorous acid and therefore causes severe irritation (Proctor, Hughes, and Fischman 1988, p. 188).

The 0.2-mg/m³ 8-hour TWA PEL and 0.4-mg/m³ 15-minute STEL that the Agency is proposing for workplaces in the construction, maritime, and agriculture industries are based on evidence of DCDMH's systemic toxicity in laboratory animals and respiratory irritation at low exposure concentrations in human subjects. The Agency preliminarily concludes that both a TWA and a STEL are necessary to substantially reduce the significant risk of material health impairment that is associated with exposure to this substance. The proposed limits are those recently established for this substance in general industry.

DICHLOROETHYL ETHER

CAS: 111–44–4; Chemical Formula: $(CH_2ClCH_2)_2O$

H.S. No. 1127

OSHA currently has a 15-ppm ceiling limit, with a skin notation, for dichloroethyl-ether in the construction and maritime industries. The Agency has no PEL for this substance in agriculture. The ACGIH has a TLV®–TWA of 5 ppm and a TLV®–STEL of 10 ppm, with a skin notation, for dichloroethyl ether. NIOSH has no REL for this substance but concurs with the limits being proposed. The Agency is proposing a 5 ppm TWA and a 10 ppm STEL for dichloroethyl ether in agriculture, construction, and maritime. These are the limits recently established for dichloroethyl ether in general industry.

Dichloroethyl ether is a colorless, flammable liquid with a nauseating odor. It is used as a soil fumigant, a solvent, a dewaxing agent, a chemical intermediate, a wetting agent, and as a penetrant (ACGIH 1986, p. 186). Dichloroethyl ether also finds use as an ingredient of paints, paint removers, dry-cleaning fluids, lacquers, and varnishes (IARC 1975, Vol. 9, p. 119). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Dichloroethyl ether causes severe irritation of the eyes, mucous membranes, skin, and respiratory tract in both animals and humans; at high concentrations, it causes narcosis. This substance has also been shown to cause cancer in mice. The oral LG_{50} in rats is 75 mg/kg; the dermal LG_{50} in rabbits is 720 mg/kg; and the LC_{50} in rats is 330 mg/m³ for 4 hours (RTECS 1991). Schrenk, Patty, and Yant (1933/Ex. 1–665) reported that guinea pigs exposed to the vapor of dichloroethyl ether at a concentration of 500 ppm experienced immediate and severe eye and nose irritation, respiratory disturbances after 1.5 to 3 hours, and death after 5 to 8 hours; lung, kidney, liver, and brain damage were observed in these animals at autopsy. Exposure to a reduced concentration of 105 ppm caused death after 10 hours of continuous exposure, while a 18-hour exposure to the same concentration caused irritation only (Carpenter, Smyth, and Pozzani 1949/Ex. 1–722). At 35 ppm, for an unspecified duration, irritation but no other adverse effects were observed (Schrenk, Patty, and Yant 1933/Ex. 1–665). Rats responded similarly; 4-hour exposures to a concentration of 250 ppm proved lethal to these animals (Carpenter, Smyth, and Pozzani 1949/Ex. 1–722). Repeated exposures to 69 ppm (7 hours/day, 5 days/week for 130 days) caused no serious injury in rats or guinea pigs; only mild stress-related effects were noted (Kosyan 1967/Ex. 1–914). However, other studies of guinea pigs have shown mild primary irritative effects on the skin, and fatalities occurred when 300 mg/kg was applied dermally as a pure liquid for 24 hours (Smyth and Carpenter 1948/Ex. 1–375). Direct contact of dichloroethyl ether with the eye causes moderate pain, conjunctival irritation, and transient corneal injury (Carpenter and Smyth 1946/Ex. 1–859). In oral carcinogenicity bioassays, dichloroethyl ether has caused an increased incidence of hepatomas in two strains of mice (IARC 1975, Vol. 9, p. 121). By subcutaneous administration, this substance has also caused a low incidence of injection-site tumors in mice (IARC 1975, Vol. 9, p. 121).

Humans exposed briefly to dichloroethyl ether at concentrations above 550 ppm experienced intolerable eye and nasal irritation, with coughing, nausea, and retching. Exposure to concentrations between 100 and 260 ppm were irritating but tolerable; however, the odor of dichloroethyl ether continued to be nauseating even at 35 ppm (Schrenk, Patty, and Yant 1933/Ex. 1–665). Eye irritation has been reported from industrial exposure to a concentration of dichloroethyl ether of 2.5 ppm (Bell and Jones 1958/Ex. 1–714). A single fatality, presumably from inhalation of dichloroethyl ether vapor, has been reported but not documented (Elkins 1959c, as cited in ACGIH 1986/Ex. 1–3, p. 186).

OSHA is proposing a 5 ppm TWA and 10 ppm STEL, with a skin notation, for this substance in construction, maritime, and agriculture. The Agency preliminarily concludes that these limits are necessary to substantially reduce the significant risk of irritation, lung injury, and nausea associated with occupational exposure to elevated levels of dichloroethyl ether. OSHA considers these exposure-related effects material impairments of health. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

2,2-DICHLOROPROPIONIC ACID

CAS: 75–99–0; Chemical Formula: CH_3CCl_2COOH

H.S. No. 1130

OSHA has no limit for 2,2-dichloropropionic acid in the construction, maritime, or agriculture industries. The ACGIH has a TLV®–

TWA of 1 ppm for this substance; there is no NIOSH REL. The Agency is proposing a 1 ppm 8-hour TWA for 2,2-dichloropropiónic acid in the construction, agriculture, and maritime industries. This is the limit recently established for this substance in general industry, and NIOSH (Ex. 8–47) concurs that it is appropriate.

2,2-Dichloropropionic acid is a colorless liquid. It is used as a herbicide both in agricultural and non-crop applications (ACGIH 1986, p. 190; Sittig 1985, p. 332). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

2,2-Dichloropropionic acid is corrosive to the skin and may cause permanent eye injury (ACGIH 1986, p. 190). The oral LD_{50} in rats is 970 mg/kg (RTECS 1991). Percutaneous absorption for this substance is reported to be negligible (Gosselin, Smith, and Hodge 1984, p. II–341). Application of 100 $\mu g/m^3$ to the skin of rabbits for 24 hours caused irritation (RTECS 1991). Seven-hour exposures to a saturated atmosphere of the acid vapor caused no ill effects in rats, and a 120-day study of dietary exposure in rats showed a no-effect level of 15 mg/kg/day (ACGIH 1986, p. 190).

Acute human exposures to 2,2-dichloropropionic acid have been reported to cause mild to moderate skin, eye, and respiratory irritation. Minimal respiratory irritation was observed in workers exposed to concentrations of between 2 and 7 ppm (ACGIH 1986, p. 190). Repeated or prolonged contact may cause mild skin burns, and eye contact causes transient corneal injury (HSDB 1985). The signs and symptoms of systemic poisoning caused by 2,2-dichloropropionic acid include anorexia, nausea, and sweating (Hazardous Substance Fact Sheet 1986).

The Agency preliminarily concludes that a 1-ppm TWA limit for 2,2-dichloropropionic acid will protect workers in construction, maritime, and agriculture from the significant risk of eye, skin, and respiratory irritation associated with exposure to this substance. The Agency considers these exposure-related effects to be material impairments of health. The proposed PEL is the limit recently established for this substance in general industry.

DIETHYLAMINE
CAS: 109–89–7; Chemical Formula: $(C_2H_5)_2NH$
H.S. No. 1137

OSHA's current limit for diethylamine in the construction and maritime industries is 25 ppm as an hour TWA. The Agency has no PEL for this substance in agriculture. The ACGIH has a TLV®–TWA of 10 ppm and a TLV®–STEL of 25 ppm for diethylamine; there is no NIOSH REL for this substance. OSHA is proposing a PEL of 10 ppm as an 8-hour TWA and a STEL of 25 ppm for diethylamine in the construction, agriculture, and maritime industries. These limits were recently established for this substance in general industry, and NIOSH (Ex. 8–47) concurred that these limits were appropriate for diethylamine.

Diethylamine (DEA) is a colorless liquid with an ammonia-like odor. It is used in pharmaceuticals and dyes, as a flotation agent, and in the rubber and petroleum industries (ACGIH 1986, p. 197). DEA also finds use as a chemical intermediate in the synthesis of rubber processing chemicals and other substances (HSDB 1985).

Diethylamine is a strong irritant of the eyes, skin, and mucous membranes in both animals and humans; multiple exposures of laboratory animals to sublethal concentrations of DEA cause tracheitis, bronchitis, pneumonitis, and pulmonary edema (Proctor, Hughes, and Fischman 1988, p. 199; ACGIH 1986/Ex. 1–3, p. 197). The oral LD_{50} in rats is 540 mg/kg, the dermal LD_{50} in rabbits is 820 mg/kg, and the 48-hour LC_{50} in rats is 4000 ppm (RTECS 1991). Instillation of 1 percent or greater solutions of DEA into the eyes of rabbits causes corneal opacity (injury graded 10 on an ascending severity scale of 1 to 10) (RTECS 1991; Grant 1986, p. 333; Sutton 1963/Ex. 1–1101), and direct contact of the skin with diethylamine causes necrosis (ACGIH 1986, p. 197). Rabbits exposed 7 hours/day, 5 days/week for 6 weeks to a 50- or 100-ppm concentration of diethylamine survived; those exposed to a 50-ppm concentration showed marked lung and corneal irritation, and, occasionally, myocardial degeneration (Brieger and Hodes 1951/Ex. 1–408). In the animals exposed to 100 ppm, these changes were more severe, and degeneration of the heart muscle was marked (Brieger and Hodes 1951/Ex. 1–408).

In humans, inhalation of high concentrations of diethylamine causes severe cough and chest pain; if exposure is repeated or prolonged, pulmonary edema may occur. One individual was splashed in the eye with diethylamine and subsequently experienced intense pain; despite treatment, some permanent visual impairment occurred (Proctor, Hughes, and Fischman 1988, p. 199). Vesiculation and necrosis of the skin occurs if liquid diethylamine contacts the skin (AIHA Hygienic Guide Series 1960).

Based on this evidence in humans and animals, OSHA preliminarily concludes that the limits being proposed in construction, maritime, and agriculture—10 ppm as an 8-hour TWA and 25 ppm as a 15-minute STEL—are necessary to reduce the significant risk of material impairment of health associated with exposure to DEA. These are the limits recently established for this substance in general industry.

DIETHYLAMINOETHANOL
CAS: 100–37–8; Chemical Formula: $(C_2H_5)_2NCH_2CH_2OH$
H.S. No. 2062

In general industry, construction, and maritime, OSHA's current permissible exposure limit for 2-diethylaminoethanol is 10 ppm as an 8-hour TWA, with a skin notation. The ACGIH has a TLV®–TWA of 10 ppm, also with a skin notation, for this substance; there is no NIOSH REL. OSHA is proposing that a 10-ppm 8-hour TWA PEL, and a skin notation, apply in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all regulated sectors.

2-Diethylaminoethanol is a colorless liquid with a weak odor similar to that of ammonia (Sittig 1985, p. 343). This substance is used as a curing agent for resins, an emulsifying agent, a fabric softener, a chemical intermediate for petroleum and gas- processing chemicals, cosmetics, and surface coatings, and in pharmaceutical manufacture (HSDB 1985; Sittig 1985, p. 343; ACGIH 1986, p. 198).

2-Diethylaminoethanol is an irritant of the eyes, skin, and mucous membranes. The liquid is a severe irritant of the eyes and skin and causes skin sensitization in laboratory animals; however, data on the toxic effects of exposure to this substance are very limited for humans, although nausea and vomiting have been reported. The oral LD_{50} in rats is 1300 mg/kg, and the dermal LD_{50} in rabbits is 1260 mg/kg (RTECS 1990). Instilled into rabbit eyes, 5 milligrams produced severe irritation (RTECS 1990). Marked eye and nasal irritation was exhibited by rats on the first day of exposure to a 500-ppm concentration for 6 hours. Mild tremors of the forelegs and head occurred throughout the 5-day study and, by the third exposure, corneal opacity was noted in several animals. All rats showed weight loss, and four of 20 died by the fifth exposure. Acutely poisoned animals showed purulent bronchitis and bronchopneumonia at autopsy (ACGIH 1986, p. 198; Proctor, Hughes, and Fischman 1988, p. 200). In guinea pigs, the liquid has been shown to be a severe irritant of the skin and to cause skin

sensitization (Proctor, Hughes, and Fischman 1988, p. 200).

A reported human exposure to this substance involved the brief inhalation of a 100-ppm concentration of diethylamino-ethenol and resulted in nausea and vomiting within 5 minutes of the onset of exposure (Proctor, Hughes, and Fischman 1988, p. 200).

Based on this evidence, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing eye, skin, and respiratory irritation and nausea associated with exposure to diethylaminoethanol. The Agency believes that establishing an 8-hour TWA of 10 ppm, with a skin notation, is necessary to substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DIISOBUTYL KETONE

CAS: 108–83–8; Chemical Formula: $[(CH_3)_2CHCH_2]_2CO$

H.S. No. 1140

OSHA currently has an hour TWA limit of 50 ppm for diisobutyl ketone in the construction and maritime industries. The Agency has no PEL for this substance in agriculture. The ACGIH has a TLV®–TWA of 25 ppm for diisobutyl ketone, which is also the NIOSH REL. OSHA is proposing a PEL of 25 ppm as an 8-hour TWA for diisobutyl ketone in the construction, agriculture, and maritime industries. This is the limit recently established for this substance in general industry.

The primary health effects associated with exposure to diisobutyl ketone are eye, nose, and throat irritation, although experimental animals have shown some systemic effects and at high concentrations have shown narcotic effects. The oral LD_{50} in rats is 5750 mg/kg, the lowest lethal concentration in the same species is 2000 ppm for 4 hours, and the dermal LD_{50} in rabbits is 16 g/kg (RTECS 1990). Rats and guinea pigs survived single exposures lasting for 7.5 to 16 hours to essentially saturated vapor (McOmie and Anderson 1949/Ex. 1–918). Smyth, Carpenter, and Weil (1949/Ex. 1–528) reported that five of six rats were narcotized and died after exposure to a concentration of 2000 ppm for hours. At autopsy, acutely poisoned animals showed damage of the lungs, liver, and kidneys (Carpenter, Pozzani, and Weil 1953). Direct application of diisobutyl ketone to rabbit skin caused only mild irritation, and no eye irritation was reported after instillation of this substance into rabbit eyes (Smyth, Carpenter, and Weil 1949/Ex. 1–528). Carpenter and Smyth (1946/Ex. 1–859) reported a no-effect level for diisobutyl ketone of 125 ppm in rats and guinea pigs given 30 7-hour exposures. At a concentration of 250 ppm on the same regimen, the liver and kidney weights of female rats increased and the liver weights of male guinea pigs decreased; at concentrations of 530 and 920 ppm, rats showed increased liver and kidney weights at autopsy, and an increase in mortality was noted when the concentration reached 1650 ppm (Carpenter and Smyth 1946/Ex. 1–859). Daily diisobutyl ketone doses of 4000 mg/kg administered by gavage killed all rats after 2 or 3 days. Severe central nervous system depression, hepatotoxicity, and dehydration were the cause of death in these acutely poisoned animals (Clayton and Clayton 1982, p. 4770).

Silverman, Schulte, and First (1946/Ex. 1–142) reported that volunteers experienced eye irritation and complained of objectionable odor when exposed to concentrations of diisobutyl ketone above 25 ppm. At a 100-ppm concentration for 3 hours, these subjects experienced lacrimation, throat irritation, headaches, and dizziness (Silverman, Schulte, and First (1946/Ex. 1–142).

Based on this evidence, OSHA preliminarily concludes that the proposed 25-ppm limit is necessary to reduce the significant risk of irritation, headaches, and dizziness associated with workplace exposures to diisobutyl ketone. This is the limit recently established for this substance in general industry. The Agency considers these exposure-related effects material impairments of health and believes that the proposed PEL will substantially reduce these risks for workers in construction, maritime, and agriculture.

DIMETHYLAMINE

CAS: 124–40–3; Chemical Formula: $(C_2H_5)_2NH$

H.S. No. 2066

In general industry, construction, and maritime, OSHA's permissible exposure limit for dimethylamine is 10 ppm as an 8-hour time-weighted average (TWA). There is no limit in agriculture for this substance. The 1987–1988 ACGIH TLV®–TWA for this substance is 10 ppm; NIOSH has no REL for dimethylamine. OSHA is proposing an 8-hour TWA PEL of 10 ppm for dimethylamine in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all regulated sectors.

Dimethylamine is a gas at room temperature and has a strong ammonia-like odor (HSDB 1985; Hawley's 1987, p. 409). It is used as a propellant for pesticides, a chemical intermediate, an accelerator in vulcanizing rubber, and as an attractant for boll weevils (to facilitate their extermination). Dimethylamine is also used as an acid gas absorbent, a dehairing agent, a gasoline stabilizer, and an ingredient in missile fuels, rocket propellants, and pharmaceuticals (HSDB 195; Hawley's 1987, p. 410).

Dimethylamine is an irritant of the eyes, mucous membranes, and respiratory tract in both animals and humans (Proctor, Hughes, and Fischman 1988, p. 207; ACGIH 1986, p. 206). The 6-hour LC_{50} in rats is 4540 ppm, and the oral LD_{50}s in mice, rats, and guinea pigs are 316 mg/kg, 698 mg/kg, and 240 mg/kg, respectively (RTECS 1990). Severe irritation of the gastric mucosa, accompanied by hemorrhage in the stomach wall, was noted in animals acutely poisoned by oral administration (ACGIH 1986, p. 206). Signs of eye irritation, gasping, tearing, salivation, and bloody discharge from the nose were noted in rats that died after exposure for 1 hour to a 4540-ppm concentration. Corneal opacity occurred 3 hours after the exposure began, and death was usually preceded by convulsions; at autopsy, severe congestion, ulceration, and necrosis of the nasal turbinates were observed (Steinhagen et al. 1982). At concentrations greater than 2500 ppm, emphysema, bronchopneumonia, liver cell death, and corneal ulceration occurred in acutely poisoned animals (Steinhagen et al. 1982). Animals of several species were repeatedly exposed to concentrations of approximately 100 to 200 ppm of dimethylamine for 18 to 20 weeks and developed marked irritation of the respiratory tract and pulmonary edema and also sustained injury to the liver (McNulty 1983). Among the guinea pigs and rabbits exposed on this regimen, corneal injury was observed after 9 days of exposure; degenerative changes of the testicles occurred in one monkey and one of ten rats in these experiments (ACGIH 1986, p. 206). Continuous exposure to a 5 ppm dimethylamine concentration for 90 days resulted in no overt signs of toxicity in experimental animals of several species, but autopsy showed inflammatory changes in the lungs (Coon et al. 1970). Skin contact with dimethylamine results in necrosis, and eye contact causes severe corneal injury or permanent corneal opacity (Proctor, Hughes, and Fischman 1988, p. 207; Grant 1986, p. 348).

In humans, contact of the eyes with this substance causes intense pain, severe damage, and may cause

permanent corneal opacity (Grant 1986, p. 348). Skin contact with the liquid may cause minor irritation on short contact; on longer contact, necrosis of the skin is possible (Genium MSDS 1986, No. 588). Dermatitis and conjunctivitis have both been reported to occur in chemical workers chronically exposed to this substance (Proctor, Hughes, and Fischman 1988, p. 207). By analogy with effects seen in animals, sublethal doses could result in delayed tracheitis, bronchitis, pulmonary edema, and pneumonitis, and ingestion could lead to gastric irritation and hemorrhage (ACGIH 1986a, p. 207).

Based on this evidence, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing the eye, mucous membrane, and respiratory tract irritation associated with exposure to dimethylamine. The Agency believes that establishing an 8-hour TWA PEL of 10 ppm is necessary to substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DIMETHYLPHTHALATE

CAS: 131–11–3; Chemical Formula: $C_6H_4(COOCH_3)_2$

H.S. No. 1136

OSHA's PEL for dimethylphthalate in general industry, construction, and maritime is 5 mg/m³ as an 8-hour TWA; there is no PEL in agriculture. The ACGIH TLV®–TWA for dimethylphthalate is 5 mg/m³ as an 8-hour TWA; there is no NIOSH REL for this substance. OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ for dimethylphthalate in agriculture. This action will make the PEL for this substance consistent across all OSHA-regulated sectors.

Dimethylphthalate is a colorless, viscous liquid with a slightly aromatic, ester-like odor (Clayton and Clayton 191, p. 2343; HSDB 1991). It is primarily used as a solvent and plasticizer. Since World War II, dimethylphthalate has been used as an insect repellant; this substance is also an effective leech repellant. Dimethylphthalate also finds use as an ingredient in solid rocket propellants, lacquers, coating agents, safety glass, molding powders, and perfumes (HSDB 1987).

Dimethylphthalate is an irritant of the eyes and mucous membranes in both animals and humans; embryotoxic and fetotoxic effects have been reported in animals. The oral LD_{50}s in rats and mice are 6800 mg/kg (both species); the lowest lethal concentration in cats is 9630 mg/m³ for 6 hours (RTECS 1991). Applied to 10 percent of the body surface of rabbits for 90 days at a dose of 4.0 mg/kg, dimethylphthalate caused death in some animals; at autopsy, pulmonary edema and slight kidney damage were seen in these animals (Draize J. et al. 1948, in ACGIH 1986, p. 211). Cats exposed to dimethylphthalate vapors exhibited signs of intense irritation of the mucous membranes, excessive salivation, and restlessness at a concentration of 250 ppm; exposure to a concentration of 1250 ppm caused the death of one cat and signs of nervous system depression in the surviving animals (LeFaux 1968, p. 136, in ACGIH 1986, p. 211). A chronic feeding study in female rats involving doses of 2 to 8 percent dimethylphthalate in the diet showed growth effects in animals dosed at the 4 and 8 percent levels and nephritic changes in those dosed at the 8 percent level (Clayton and Clayton 1982, p. 2343). Pregnant rats given intraperitoneal injections of dimethylphthalate at various doses on days 5 through 15 or 3 through 9 of gestation had litters with an increased number of skeletal abnormalities or experienced an increase in the number of resorptions (RTECS 1991; Singh, Lawrence, Autian 1972, J. Pharmacol. Sci. 61:51–55; J. Dairy Sci 55, 1972).

In humans, dimethylphthalate is an irritant of the eyes and mucous membranes; this substance is not readily absorbed through the skin (Windholz M and Budavari S, 1976). Ingestion of dimethylphthalate causes gastrointestinal irritation, hypotension, and coma. In one fatal case of a suicidal ingestion of a mixture containing dimethylphthalate and ketone peroxides, the principal toxic symptoms were marked esophagitis and gastritis with hemorrhage (NIOSH/OSHA Guidelines 1981, p. 1). Direct contact of dimethylphthalate with the eyes has caused chemical burns that subsequently healed completely (McLaughlin RS, 1946, in Clayton and Clayton 1981, p. 2343).

Based on this evidence, OSHA preliminarily concludes that agricultural workers exposed to this substance at the levels permitted by the absence of a limit are at significant risk of experiencing irritation of the eyes and mucous membranes. The Agency considers these effects material health impairments and believes that establishing a PEL of 5 mg/m³ as an 8-hour TWA is necessary to protect workers in agriculture from these significant risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

EPICHLOROHYDRIN

CAS: 106–89–; Chemical Formula: C_3H_5ClO

H.S. No. 1158

OSHA has an 8-hour TWA limit of 5 ppm, with a skin notation, for epichlorohydrin in the construction and maritime industries. The Agency has no PEL for this substance in agriculture. The ACGIH has a TLV®-TWA of 2 ppm, with a skin notation, for epichlorohydrin. NIOSH considers this substance a potential human carcinogen and recommends that occupational exposure be minimized to the extent possible. OSHA is proposing a 2 ppm TWA, with a skin notation, for epichlorohydrin in the construction, agriculture, and maritime industries. This is the limit recently promulgated for this substance in the air contaminants standard for general industry.

Epichlorohydrin is an unstable liquid with an odor like that of chloroform. It is used as a solvent, as the raw material for the manufacture of epoxy and phenoxy resins and of elastomers, in the synthesis of glycerol, and in the rubber and paper industries (ACGIH 1986, p. 233).

In animals, epichlorohydrin causes irritation and is systemically toxic by all routes of exposure (Shell Chemical Corporation 1958, as cited in ACGIH 1986/Ex. 1–3, p. 233). Chronic exposure causes nephrotoxicity (IARC 1987, Suppl. 7, p. 202). This substance also causes cancer and has reproductive effects in experimental animals. The oral LD_{50} in rats ranges from 90 mg/kg to 260 mg/kg, and the dermal LD_{50} in rabbits ranges from 515 mg/kg to 755 mg/kg (Lawrence, Malik, Turner, and Autian 1972/Ex. 1–1058; RTECS 1987). Acutely poisoned animals show respiratory and nervous system effects before death. In mice, single 30-minute exposures to an 8300-ppm concentration of epichlorohydrin vapor caused muscular paralysis and death from respiratory failure; similar results have been reported in rats after dermal application of a 0.5-ml/kg dose and in mice after repeated oral administration of 0.1 mg/kg doses (Shell Chemical Corporation 195, as cited in ACGIH 1986/Ex. 1–3, p. 233). When exposed to an epichlorohydrin concentration of 32 ppm (7 hours/day, 5 days/week) for 91 days, rats failed to gain weight nor mally; at a concentration of 16 ppm they developed enlarged kidneys (ACGIH 1986/Ex. 1–3, p. 233). Gage (1959/Ex. 1–1052) confirmed these findings and demonstrated lung, liver, and kidney injury in rats exposed repeatedly for 6 hours to epichlorohydrin concentrations ranging from 17 to 120 ppm; no effects

were observed when the concentration was reduced to 9 ppm. Epichlorohydrin has been tested for carcinogenicity in rats by oral administration and inhalation and in mice by skin application and subcutaneous and intraperitoneal injection. The results of both bioassays in rats were positive (papillomas and carcinomas of the forestomach and nasal cavity, respectively). Results of the skin painting study in mice were negative; however, epichlorohydrin caused injection-site tumors when administered to mice subcutaneously and was active in a mouse-lung tumor bioassay by intraperitoneal injection (IARC 1987, Suppl. 7, p. 202). Based on this evidence, the International Agency for Research on Cancer has concluded that the evidence for the carcinogenicity of epichlorohydrin in animals is sufficient and has assigned this substance to Category 2A (IARC 1987, Suppl. 7, p. 202). Administration of 15 mg/kg/day to rats caused infertility within 7 days; cessation of exposure restored fertility in these animals (IARC 1976, Vol. 11, p. 135). In a recent experiment, fertility in male rats given 50 mg/kg/day for 21 days before mating was totally impaired; dose-dependent trends in all sperm-motion parameters were observed in this study (Toth, Zenick, and Smith 1989, in Silver Platter printout).

In humans, exposure to epichlorohydrin causes moderate-to-severe irritation of the eyes and respiratory tract, skin burns and sensitization, and central nervous system, blood, and kidney effects. Exposure to an epichlorohydrin concentration of 20 ppm for 1 hour caused burning of the eyes and nasal mucosa (NIOSH 1976c/Ex. 1–972). At a concentration of 40 ppm, irritation persisted for 48 hours after exposure (NIOSH 1976c/Ex. 1–972). A worker exposed to high (not further specified) concentrations of epichlorohydrin experienced eye and upper respiratory tract irritation, nausea, vomiting, headache, and difficult breathing; this individual continued to show signs of pulmonary and liver damage and of hypertension 2 years after the incident (NIOSH 1976c/Ex. 1–972). In contact with the skin, epichlorohydrin causes redness and itching, as well as burns that may have a delayed onset (NIOSH 1976c/Ex. 1–972). Six workers in the Netherlands developed skin sensitization reactions to this substance after repeated contact with it in the workplace (van Joost 1988, Contact Derm: 19). NIOSH (1976c/Ex. 1–972) reports that humans exposed to concentrations of epichlorohydrin above 100 ppm for brief periods have subsequently developed lung edema and kidney damage. A group of epichlorohydrin-exposed workers showed blood and cytogenetic effects, including increased incidences in the percentage of lymphocytes with chromatid breaks, damaged blood cells, and abnormal cells (Picciano 1979, Mutat. Res. 66:169–173). A laboratory assistant severely overexposed to epichlorohydrin developed eye and lung irritation, followed by chronic asthmatic bronchitis. Serial biopsies of the liver in this worker established that a substantial amount of fatty infiltration had occurred (Schultz 1964, in Deutsche med. Wochenschrift 89:1342–1344). The results of epidemiological studies of epichlorohydrin-exposed workers have been inconclusive. One study showed a slight excess of lung cancer in two cohorts of U.S. workers (Enterline 1982, in Ann. N.Y. Acad. Sci. 381:344–349); another study in a European cohort yielded inconclusive results (Tassignon, Bos, Craigen, Jacquet, Kueng, Lanouziere-Simon, Pierre 1983, in Int. Arch. Occup. Environ. Hlth. 51:325–336). A recent mortality study in dye and resin workers (Delzell, Macaluso, Cole 1989, in JOM 31) has shown a slight excess of lung cancer, although the number of observed cases was small in this study.

OSHA is proposing an hour TWA limit of 2 ppm, with a skin notation, for epichlorohydrin in the construction, agriculture, and maritime industries. These are the limits recently established for this substance in general industry. The skin notation is proposed because of this substance's capacity to penetrate the skin and cause systemic toxicity. The Agency preliminarily concludes that this limit and the skin notation will protect workers in these sectors from the significant risk of dermal, respiratory, liver, and kidney effects that are potentially associated with exposure to epichlorohydrin at elevated concentrations. OSHA considers these exposure-related effects material impairments of health.

ETHYL ACETATE

CAS: 141–78–6; Chemical Formula: $CH_3COOC_2H_5$

H.S. No. 2075

In general industry, construction, and maritime, OSHA's current permissible exposure limit for ethyl acetate is 400 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 400 ppm for this substance; there is no NIOSH REL. OSHA is proposing to apply an 8-hour TWA PEL of 400 ppm to ethyl acetate in agricultural workplaces. Promulgation of this limit will make the PEL for ethyl acetate consistent across industry sectors.

Ethyl acetate is a colorless liquid with a fruity odor. It is used as a solvent for varnishes, lacquers, and nitrocellulose; and in the manufacture of artificial silk and leather, perfumes, photographic film, and artificial fruit essences (ACGIH 1986, p. 239; Clayton and Clayton 1982, p. 2267).

Ethyl acetate is an irritant of the eyes, mucous membranes, and respiratory tract in both animals and humans; at high concentrations it causes central nervous system depression in laboratory animals (ACGIH 1986, p. 239; Proctor, Hughes, and Fischman 1988, p. 234). The oral LD_{50} in rats is 5620 mg/kg; the subcutaneous LD_{50} in cats is reported to be 3000 mg/kg (RTECS 1990). Cats exhibited irritation and labored breathing following an 8-hour exposure to a 9000-ppm concentration of ethyl acetate; deep narcosis resulted from exposure to 20,000 ppm for 45 minutes, and exposure of these animals to 43,000 ppm for about 15 minutes was fatal. Autopsy revealed pulmonary edema with hemorrhage and hyperemia of the respiratory tract (Proctor, Hughes, and Fischman 1988, p. 234). Rabbits exposed repeatedly to 4450 ppm showed secondary anemia with leukocytosis and hyperemia and, at autopsy, liver damage (Proctor, Hughes, and Fischman 1988, p. 234). A study by Smyth and Smyth (1928) stated that animals could survive 65 exposures lasting 4 hours each at a concentration of 2000 ppm without suffering any ill effects.

Exposure of unacclimated human subjects to a 200-ppm concentration of ethyl acetate resulted in complaints about its strong, objectionable odor, and irritation of the eyes, nose, and throat was experienced at a concentration of 400 ppm (ACGIH 1986, p. 239). However, no adverse effects were noted in workers exposed for several months to ethyl acetate concentrations ranging from 375 to 1500 ppm (Proctor, Hughes, and Fischman 1988, p. 234; ACGIH 1986, p. 239). Exposure to ethyl acetate may, in rare instances, cause skin sensitization and result in inflammation of the mucous membranes and eczematous eruptions (Proctor, Hughes, and Fischman 1988, p. 234). There is one report of a fatality that resulted when a tank painter inhaled very high concentrations (level unspecified) of ethyl acetate (ACGIH 1986, p. 239).

Based on this evidence, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant

risk of experiencing the eye, mucous membrane, and respiratory tract irritation potentially associated with exposure to ethyl acetate and that they may also be at significant risk of experiencing central nervous system depression. The Agency believes that establishing an 8-hour TWA of 400 ppm for ethyl acetate in agriculture is necessary to substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ETHYL AMYL KETONE

CAS: 541–85–5; Chemical Formula: $CH_3CH_2COCH_2CH_2CHCH_2CH_3$

H.S. No. 2080

In general industry, construction, and maritime, OSHA's permissible exposure limit for ethyl amyl ketone is 25 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 25 ppm for ethyl amyl ketone. NIOSH has no REL but concurs with the PEL being proposed. OSHA is proposing an 8-hour TWA PEL in agriculture of 25 ppm for ethyl amyl ketone. Promulgation of this limit will make the PEL for ethyl amyl ketone consistent across all OSHA-regulated sectors.

Ethyl amyl ketone, which is also called 5-methyl-3-heptanone, is a colorless liquid with a strong, pungent odor that is similar to that of apricots and peaches. Ethyl amyl ketone is used as a solvent for nitrocellulose and vinyl resins and in the manufacture of perfume (Hawley's 1987, p. 478; Clayton and Clayton 1982, p. 4767).

Ethyl amyl ketone is an irritant of the eyes, mucous membranes, and skin in humans and animals. At very high concentrations, it also causes central nervous system depression (Proctor, Hughes, and Fischman 1988, p. 237; Clayton and Clayton 1982, p. 4767). The oral LD_{50}s for rats, mice, and guinea pigs are 3500 mg/kg, 300 mg/kg, and 2500 mg/kg, respectively (RTECS 1991). In contact with the skin of rabbits, a 500-mg dose of ethyl amyl ketone caused mild irritation (RTECS 1991). The lowest lethal concentrations in rats and mice are 3484 ppm over 8 hours and 3484 ppm over 4 hours, respectively (RTECS 1988). Mice and rats rapidly developed signs of eye and respiratory tract irritation when exposed to a single dose of vapor-saturated air (an ethyl amyl ketone concentration of approximately 3000 ppm) for 4 hours. Three of six mice died from this exposure, but there were no deaths among the rats (Clayton and Clayton 1982, p. 4768). Four of the six rats died after exposure to a 6000-ppm concentration of ethyl amyl ketone for 8 hours (Clayton and Clayton 1982, p. 476). Other signs of overexposure, including ataxia, prostration, and respiratory distress, were observed in these animals and were followed by narcosis (Clayton and Clayton 1982, p. 4768; Proctor, Hughes, and Fischman 1988, p. 237).

In humans, eye contact with ethyl amyl ketone causes transient corneal injury, and repeated contact with the skin results in drying and cracking (Proctor, Hughes, and Fischman 1988, p. 237). Individuals exposed to a 25-ppm concentration of ethyl amyl ketone developed irritation of the eyes and respiratory tract; exposure to 100 ppm caused symptoms of mucous membrane irritation, headache, and nausea that were too severe to be tolerated for more than a few minutes (Krasavage 1982, in Proctor, Hughes and Fischman 1988, p. 237).

Based on this evidence, OSHA preliminarily concludes that ethyl amyl ketone causes irritation of the eyes and mucous membranes, dermatitis, and narcosis in exposed workers. OSHA therefore preliminarily finds that, in the absence of a limit for ethyl amyl ketone, workers in agriculture are at significant risk of experiencing these material impairments of health and that the proposed 8-hour TWA PEL of 25 ppm is necessary to substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ETHYL BENZENE

CAS: 100–41–4; Chemical Formula C_8H_{10}

H.S. No. 1162

OSHA's limit for ethyl benzene in the construction and maritime industries is 100 ppm as an 8-hour TWA. The Agency has no PEL in agriculture for this substance. The ACGIH has a TLV®–TWA of 100 ppm and a TLV®–STEL of 125 ppm for ethyl benzene; there is no NIOSH REL. Based on the irritant properties associated with exposure to ethyl benzene, OSHA is proposing permissible exposure limits for this substance in the construction, agriculture, and maritime industries of 100 ppm as an 8-hour TWA and 125 ppm as a 15-minute STEL. The Agency recently established these limits for ethyl benzene in general industry, and NIOSH (Ex. 8–47) concurred that these limits were appropriate for this substance.

Ethyl benzene is a colorless, flammable liquid with an aromatic odor. It is used as an intermediate in synthetic rubber and styrene production and as a solvent (ACGIH 1986, p. 244). This substance also finds use as a component of fuel (HSDB 1985).

Ethyl benzene is an irritant of the eyes, skin, and mucous membranes in both animals and humans; at high concentrations, this substance causes central nervous system depression (Proctor, Hughes, and Fischman 1988, p. 237). The oral LD_{50} in rats is 3500 mg/kg, and the dermal LD_{50} in rabbits is reported to range from 5000 to 17,800 mg/kg (Clayton and Clayton 1982, p. 3306; RTECS 1991). The lowest lethal concentration in rats is 4000 ppm for 4 hours (RTECS 1991).

A comment submitted by the Arco Chemical Company in OSHA's recent air contaminants rulemaking for general industry summarizes the available information on ethyl benzene's toxicity in animals, as reported in several recent studies (ECETOC 1986; Dynamac Corporation 1986) and in a personal communication from the National Toxicology Program's Chemical Manager for Ethyl Benzene. (Arco's comment summarizing these findings is in the docket as Ex. 3–638.) These investigators found that ethyl benzene causes: (1) Moderate dermal irritation on intact and abraded rabbit skin after a 24-hour application; (2) mild conjunctival irritation (without corneal effects) from direct instillation of undiluted ethyl benzene in rabbit eyes; (3) erythema and edema with superficial necrosis, resulting in exfoliation of large patches of skin, following repeated and prolonged application of the undiluted material to rabbit skin; (4) "a slight, cloudy swelling of hepatocytes" in animals subchronically exposed to the vapor; (5) congestion of the lungs, nasal mucosa, liver, and kidneys in mice and rats exposed 6 hours/day for 4 consecutive days to ethyl benzene concentrations of 2360 ppm and in mice exposed to 1190 ppm; and (6) lacrimation and salivation in rats exposed to 400 or 800 ppm concentrations of ethyl benzene for 6 hours/day, 5 days/week (ECETOC 1986 and Dynamac Corporation 1986, both as cited in Ex. 3–638). Arco (Ex. 3–638) stressed the fact that, except at very high concentrations, significant systemic toxicity does not appear to be a manifestation of ethyl benzene exposure.

Brief exposures of human subjects to a 5000-ppm concentration of ethyl benzene caused intolerable irritation, and exposure to 2000 ppm resulted in lacrimation, nasal irritation, and vertigo; exposure to a 1000-ppm concentration produced eye irritation, although volunteers rapidly developed a tolerance to this concentration. Acute exposure to the vapor at a concentration of 200 ppm causes mild eye irritation

(Wolf et al. AMA Arch. Ind. Hlth. 14:387, 1956). Chronic exposure to ethyl benzene concentrations above 100 ppm has caused fatigue, sleepiness, headache, and eye and respiratory tract irritation (Bardodej and Bardodegova 1970). Redness and inflammation may occur on contact of the skin with this substance (HSDB 1985).

Based on this evidence in humans and animals, OSHA preliminarily concludes that workers in construction, maritime, and agriculture who are exposed to concentrations of ethyl benzene above 100 ppm, even briefly, are at significant risk of experiencing eye and upper respiratory tract irritation. The Agency believes that the proposed limits are necessary to substantially reduce this risk of material health impairment. Accordingly, OSHA is proposing in construction, maritime, and agriculture a 15-minute short-term limit of 125 ppm for ethyl benzene to supplement the proposed 100-ppm TWA limit for this substance in these sectors. Promulgation of the proposed STEL will make OSHA's PELs for ethyl benzene consistent across all industry sectors within OSHA's jurisdiction.

ETHYL BUTYL KETONE

CAS: 106–35–4; Chemical Formula: $C_7H_{14}O$

H.S. No. 2077

In general industry, construction, and maritime, OSHA's permissible exposure limit for ethyl butyl ketone is 50 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 50 ppm for this substance; NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL in agriculture of 50 ppm for ethyl butyl ketone. Promulgation of this limit will make the PEL for this substance consistent across all regulated sectors.

Ethyl butyl ketone is a colorless liquid with a strong, fruity odor. It is used primarily as an organic solvent and a chemical intermediate but also finds use as a synthetic flavoring agent (Clayton and Clayton 1982, p. 4761; HSDB 1984).

Ethyl butyl ketone is an irritant of the eyes, mucous membranes, and skin in laboratory animals; at high concentrations, it causes central nervous system depression. The oral LD_{50} in rats is 2760 mg/kg; the dermal LD_{50} for rabbits is greater than 16.4 g/kg (20 ml/kg) (RTECS 1990; ACGIH 1986, p. 246). Rats exposed to a 4000-ppm concentration for 4 hours died, but all rats survived an exposure to 2000 ppm for 4 hours (ACGIH 1986, p. 246). A 68-hour exposure to a 5800-ppm ethyl butyl ketone concentration killed all rats, but exposure to 420 or 3000 ppm for the same period of time was not lethal; however, ataxia, prostration, and narcosis were noted in these animals at the higher exposures (Clayton and Clayton 1982, p. 4762). Rats administered 2 g/kg/day by gavage, 5 days/week for 14 weeks exhibited hind-leg weakness and tail drag. Neuropathology revealed central-peripheral-distal axonopathy characterized by neurofilamentous hyperplasia and giant axonal swelling (O'Donoghue et al. 1984). No signs of systemic toxicity or neurotoxicity were observed in rats exposed to a 700-ppm concentration of ethyl butyl ketone for 72 hours/week for 24 weeks (Clayton and Clayton 1982, p. 4762; Proctor, Hughes, and Fischman 1988, p. 239). Ethyl butyl ketone causes mild-to-moderate irritation when applied to the skin of rabbits. Contact of ethyl butyl ketone with the eyes of rabbits caused slight ocular irritation (Clayton and Clayton 1982, p. 4761).

Information on the effects of human exposure to ethyl butyl ketone is limited. Applied to the skin of volunteers for 48 hours under an occluded patch, a 4-percent solution of ethyl butyl ketone failed to cause skin irritation or skin sensitization (Clayton and Clayton 1982, p. 4762).

Based on this evidence, OSHA preliminarily concludes that in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing the eye, mucous membrane, and skin irritation associated with exposure to ethyl butyl ketone and that they may also be at significant risk of experiencing central nervous system depression. The Agency believes that establishing an 8-hour TWA PEL of 50 ppm is necessary to substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ETHYL ETHER

CAS: 60–29–7; Chemical Formula: $C_2H_5OC_2H_5$

H.S. No. 1164

OSHA's limit for ethyl ether in the construction and maritime industries is 400 ppm as an 8-hour TWA. The Agency has no PEL for this substance in agriculture. The ACGIH has a TLV®–TWA of 400 ppm and a TLV®–STEL of 500 ppm for ethyl ether; there is no NIOSH REL. OSHA is proposing a 400 ppm 8-hour TWA and a 500 ppm STEL for ethyl ether in the construction, agriculture, and maritime industries. These are the limits recently established in general industry, and their promulgation will make OSHA's PELs for this substance consistent across all OSHA-regulated sectors.

Ethyl ether is a colorless, volatile, mobile liquid with a distinct odor and a burning, sweet taste. It is extremely flammable and is a severe fire and explosion hazard when exposed to heat or flame. Ethyl ether is used as a reagent in organic synthesis and is also used as a solvent for waxes, fats, oils, perfumes, alkaloids, and gums (ACGIH 1986, p. 259). Ethyl ether also finds use as an anesthetic in human and animal medicine and as a refrigerant and motor fuel additive (ITI 1986, p. 178; NIOSH/OSHA Occupational Health Guideline 1981, p. 3).

Ethyl ether is an irritant of the eyes, skin, and respiratory tract in both animals and humans; exposure to high concentrations causes central nervous system depression and anesthesia. The oral LD_{50} in rats is 1215 mg/kg, and the LC_{50} in the same species is 73,000 ppm for 2 hours (RTECS 1991). The lethal concentration for a single exposure in the monkey is reportedly between 71,600 ppm and 192,500 ppm ethyl ether by volume (Clayton and Clayton 1982, pp. 2507–2510). Exposure to a 6.4 percent (64,000 ppm) concentration of ethyl ether caused deep anesthesia in mice, and respiratory arrest occurred at a concentration of 12.8 percent (128,000 ppm) (Clayton and Clayton 1981, p. 2507–2510). Contact of the eyes of rabbits with ethyl ether has produced slight, reversible injury (Grant 1986, p. 411). Chronic exposure of rats to a 2000-ppm concentration of ethyl ether for more than 30 weeks caused no adverse effects on body weight, food consumption, hematology, urinalysis, or histopathology; however, liver enzymes were elevated in these animals despite the fact that no liver damage was identified at autopsy by microscopic examination (Clayton and Clayton 1982, pp. 2507–2510). Mice exposed to ethyl ether during pregnancy have had an increased number of offspring with abnormalities (RTECS 1991; Friedman 1988). Ethyl ether is mutagenic both in bacterial and mammalian test systems (RTECS 1991).

Concentrations of 3.6 to 6.5 percent ethyl ether in air (36,000 to 65,000 ppm) cause anesthesia in humans; exposure to 7- to 10-percent concentrations (70,000 to 100,000 ppm) causes respiratory arrest, and exposure to concentrations greater than 10 percent (100,000 ppm) can be fatal (ACGIH 1986/Ex. 1–3, p. 259). Repeated workplace exposures to ethyl ether can cause narcosis, exhaustion, headache, dizziness, sleepiness, excitation, and other psychic disturbances (Hake and Rowe 1963a/Ex. 1–1152). Albuminuria and polycythemia may be caused by repeated exposure,

and nephritis has occasionally been reported (Clayton and Clayton 1981, p. 2507–2510). Prolonged or repeated contact may cause defatting of the skin; irritation of the mucous membranes and eyes occurs on contact with the liquid or after exposure to high concentrations of the vapor (Hake and Rowe 1963a/Ex. 1–1152). Nelson and co-workers (1943/Ex. 1–66) reported that workers began to experience nasal irritation at a concentration of 200 ppm (Nelson, Enge, Ross et al. 1943/Ex. 1–66). However, Armor (1950, as cited in ACGIH 1986/Ex. 1–3, p. 259) observed that exposure effects occur in humans only at 500 ppm or higher concentrations of ethyl ether.

OSHA recently established in general industry the limits being proposed here for the construction, maritime, and agriculture industries. The Agency preliminarily concludes that both a TWA and a STEL are necessary to protect exposed workers in construction, maritime, and agriculture against the significant risk of irritation and narcosis potentially associated with excursions above the 8-hour TWA level. OSHA believes that the irritation and narcosis caused by excessive exposure to ethyl ether constitute material impairments of health and that the proposed limits are necessary to substantially reduce these risks.

ETHYL FORMATE

CAS: 109–94–4; Chemical Formula: $HCOOC_2H_5$

H.S. No. 2079

In general industry, construction, and maritime, OSHA's permissible exposure limit for ethyl formate is 100 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 100 ppm for this substance; NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL in agriculture of 100 ppm for ethyl formate. Promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

Ethyl formate is a colorless, combustible liquid with a pleasant odor. It is used as a fungicide and larvicide for tobacco crops, dried fruits, cereals, and other crops. It is also used as a flavoring agent, a solvent, and in organic synthesis (ACGIH 1986, p. 260; Clayton and Clayton 1982, p. 2263). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Ethyl formate is an irritant of the eyes, skin, and upper respiratory tract; at high concentrations, it causes central nervous system depression in laboratory animals. The oral LD_{50} in rats is 1850 mg/kg, and the dermal LD_{50} in rabbits is 20 g/kg (RTECS 1990; HSDB 1985). Exposure to an 8000-ppm concentration of ethyl formate for 4 hours killed 5 of 6 rats, but exposure to a 4000-ppm concentration for 4 hours was not lethal (Flury and Zernik 1931, p. 375; Proctor, Hughes, and Fischman 1988, p. 254). Deep narcosis resulted when cats were exposed to a 10,000-ppm concentration for 80 minutes, and these animals subsequently died. Exposure to a 10,000-ppm concentration for 4 hours caused pulmonary edema and death in dogs (Flury and Zernik 1931, p. 375; Fasset 1963, p. 1865; Proctor, Hughes, and Fischman 1988, p. 254). Cats exposed for 20 minutes to a 5000-ppm concentration exhibited signs of eye irritation. Applied to the skin of mice, ethyl formate did not cause tumors (Clayton and Clayton 1982, p. 2264; Flury and Zernik, 1931, p. 375; Proctor, Hughes, and Fischman 1988, p. 254).

Slight eye irritation and rapidly increasing nasal irritation were reported in humans exposed to a 330-ppm concentration of ethyl formate (Flury and Zernik 1931, p. 375; Proctor, Hughes, and Fischman 1988, p. 254), but no systemic effects have been reported.

Based on this evidence, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing the eye, skin, and respiratory tract irritation associated with exposure to ethyl formate and that they may also be at significant risk of experiencing central nervous system depression. The Agency believes that establishing an 8-hour TWA PEL of 100 ppm for this substance will substantially reduce this risk for workers in this sector. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ETHYL MERCAPTAN

CAS: 75–08–1; Chemical Formula: C_2H_5SH

H.S. No. 1165

OSHA has an 8-hour TWA PEL of 0.5 ppm for ethyl mercaptan in general industry and in the construction and maritime industries. The Agency has no PEL for ethyl mercaptan in agriculture. The ACGIH has a TLV®–TWA of 0.5 ppm for this substance, and the NIOSH REL is 0.5 ppm as a 15-minute ceiling. OSHA is proposing an 8-hour TWA limit of 0.5 ppm for ethyl mercaptan in the agriculture industry. Promulgation of this limit will make the PEL for ethyl mercaptan consistent across all OSHA-regulated sectors.

Ethyl mercaptan is a colorless liquid with a very strong, unpleasant odor. It is used in the manufacture of insecticides, defoliants, pharmaceuticals, adhesives, antioxidants, and plastics and as an odorant for natural gas (ACGIH 1986, p. 262; HSDB 1985). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Ethyl mercaptan is an irritant of the mucous membranes; at high concentrations, it causes central nervous system depression in both animals and humans. The oral LD_{50} in rats is 682 mg/kg, and the LC_{50} in the same species is 4420 ppm for 4 hours (RTECS 1989). Acutely poisoned animals showed behavioral and pulmonary effects before death (RTECS 1989). Maximal sublethal intraperitoneal doses have been reported to induce deep sedation, with higher exposures causing restlessness, muscular incoordination, skeletal muscle paralysis, cyanosis, respiratory depression, coma, and death. Although inhalation exposure caused no noticeable pathology in rats, intraperitoneal injection caused lymphatic infiltration of the liver, with occasional necrosis (Fairchild and Stokinger 1958, Ex. 1–415). One drop applied to rabbit eyes caused only slight irritation, but 15 minutes of exposure to high concentrations of the vapor caused closing and rubbing of the eyes, both indications that the animals were experiencing considerable irritation (Fairchild and Stokinger 1958/Ex. 1–415). In chronic inhalation studies in rabbits, rats, and mice, a five-month exposure to a 40-ppm concentration caused minimal cardiovascular and other systemic effects (Blinova 1965/Ex. 1–603).

Human volunteers exposed to a 4-ppm concentration of ethyl mercaptan for three hours daily for 5 to 10 days reported the following adverse effects: altered taste and olfactory reations, periodic nausea, mucous membrane irritation, and fatigue. Exposure of volunteers to a 0.4-ppm concentration of this substance produced no adverse signs or symptoms (ACGIH 1986/Ex. 1–3, p. 262).

Based on this evidence in humans and animals, OSHA preliminarily concludes that the limit recently established in general industry and currently in effect in all OSHA-regulated sectors other than agriculture is necessary to protect workers in the agricultural sector. OSHA considers the exposure-related effects of nausea, fatigue, and irritation to be material impairments of health. The Agency preliminarily concludes that the proposed 8-hour TWA limit of 0.5 ppm is necessary to substantially reduce

these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ETHYLAMINE

CAS: 75–04–7; Chemical Formula: $C_2H_5NH_2$

H.S. No. 2081

In general industry, construction, and maritime, OSHA's permissible exposure limit for ethylamine is 10 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 10 ppm for this substance; NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL in agriculture of 10 ppm for ethylamine. Promulgation of this limit will make the PEL for this substance consistent across all regulated sectors.

Ethylamine is a colorless liquid or gas, depending on the ambient temperature (boiling point: 16.6°C). It has a strong, unpleasant odor similar to that of ammonia (ACGIH 1986, p. 243; Genium MSDS 1985, No. 540). Ethylamine is used as a chemical intermediate for triazine herbicides (such as atrazine), a corrosion inhibitor, in resin chemistry, in organic chemistry, and as a stabilizer for rubber latex (HSDB 1986; ACGIH 1986, p. 243).

Ethylamine is an irritant of the eyes, mucous membranes, and skin; in laboratory animals, it has also been shown to cause systemic toxic effects. The oral LD_{50} in rats is 400 mg/kg (RTECS 1990; Clayton and Clayton 1981, p. 3145). The LC_{50} in rats is 3000 ppm for 4 hours, and the dermal LD_{50} in rabbits is 390 mg/kg (RTECS 1990). Two of six rats exposed to an 8000-ppm concentration of ethylamine for 4 hours died (Smyth et al. 1954, p. 61). Rabbits exposed repeatedly to a 100-ppm concentration developed corneal and pulmonary irritation, and damage to the liver and kidneys was seen at autopsy. Exposure to a 50-ppm concentration of ethylamine vapors for 2 weeks caused corneal injury in rabbits, and myocardial degeneration was seen at post-mortem (Brieger and Hodes 1951, p. 287). One drop of a 70 percent solution of ethylamine instilled into rabbit eyes caused damage rated as severe (Grant 1986, p. 413). Direct contact of the skin of guinea pigs with a 70-percent aqueous solution caused necrosis and deep scarring (ACGIH 1986, p. 243).

Exposure to ethylamine causes eye, skin, and upper respiratory tract irritation in humans. Workers have reported experiencing temporary blue, hazy vision after exposure to ethylamine; this effect is believed to be related to corneal edema (Grant 1986, p. 413). Conjunctivitis and tearing also are associated with exposure to the vapors of ethylamine (Clayton and Clayton 1982, p. 3149). Inhalation causes respiratory irritation, coughing, and difficulty in breathing. Direct contact of the eyes or skin with the liquid may cause permanent eye damage and skin burns. Systemic poisoning may occur and be manifested as headache, nausea, faintness, and anxiety (Clayton and Clayton 1982, p. 3149).

Based on this evidence in humans and animals, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing the eye, skin, and mucous membrane irritation associated with exposure to ethylamine. The Agency believes that establishing an 8-hour TWA PEL of 10 ppm is necessary to substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ETHYLENE GLYCOL

CAS: 107–21–1; Chemical Formula: CH_2OHCH_2OH

H.S. No. 1169

OSHA has no limit for ethylene glycol in the construction, agriculture, or maritime industries; however, the proposed limit of 50 ppm, as a ceiling, was recently established for this substance in general industry. The ACGIH has a TLV® of 50 ppm for ethylene glycol, as a ceiling limit; there is no NIOSH REL for this substance. The Agency is proposing a ceiling limit of 50 ppm for ethylene glycol in the construction, agriculture, and maritime industries. Promulgation of this limit will make the PEL for ethylene glycol consistent across all OSHA-regulated industries.

Ethylene glycol is used in antifreeze and coolant mixtures for cars and other vehicles; it is also an ingredient of hydraulic fluids and heat exchangers and a chemical intermediate (Clayton and Clayton 1982, p. 3818). Ethylene glycol is a colorless, viscous liquid that has no odor (Clayton and Clayton 1982, p. 3818).

Ethylene glycol causes eye irritation, central nervous system depression, and kidney and liver damage in animals. The oral LD_{50}s for various species range from 4700 mg/kg in rats to 7500 mg/kg in mice; the dermal LD_{50} in rabbits is 9530 mg/kg (RTECS 1990). Rats and rabbits exposed to an ethylene glycol concentration of 12 mg/m³ (approximately 5 ppm) 24 hours/day for 90 days showed signs of moderate to severe eye irritation and corneal damage (Coon, Jones, Jenkins, and Siegel 1970/Ex. 1–84). Another study involving 30-day or 16-week exposures to concentrations as high as 350 to 400 mg/m³ (70 to 0 ppm), however, has shown no adverse effects in rats, guinea pigs, rabbits, dogs, and monkeys (Wiley, Hueper, and von Oettingen 1936, in ACGIH 1986, p. 253). Applied to the eyes of rabbits, ethylene glycol caused pain and reversible conjunctival injury but no corneal damage (Grant 1986, p. 417). In contact with the skin, ethylene glycol caused mild irritation in rabbits (RTECS 1990). Ingestion of ethylene glycol caused depression, ataxia, vomiting, seizures, and coma in cats and dogs; at autopsy, calcium oxalate crystals were found in the renal tubules and blood vessels of the brain (HSDB 1987). Rats fed diets containing 1 or 2 percent ethylene glycol for 2 years had shortened life spans, calcium oxalate stones in the bladder, severe kidney damage, and degeneration of the liver (Clayton and Clayton 1982, p. 3820). Recent studies in rats and mice have shown that inhalation or oral administration of ethylene glycol during pregnancy causes developmental abnormalities and fetotoxic effects (RTECS 1990; NIEHS 1988; Bushy Run Center 1988).

In humans, exposure to ethylene glycol causes eye and upper respiratory tract irritation. Ingestion of ethylene glycol causes narcosis, severe kidney and liver damage, and pulmonary edema (Proctor, Hughes, and Fischman 1988, p. 246). The lowest oral dose reported to be lethal in humans is 786 mg/kg (RTECS 1990). Exposure to an aerosol of ethylene glycol at a concentration of 12 ppm for 20 to 22 hours/day for 4 weeks caused headaches and throat irritation in human volunteers (Wills et al. 1974/Ex. 1–582). When the concentration was increased to 80 ppm, the irritation was intolerable (Wills et al. 1974/Ex. 1–582). Rowe (1963/Ex. 1–865) concluded that daily exposure to a 100-ppm concentration of the vapor did not cause systemic or eye injuries, although Troisi (1949/Ex. 1–598) described nystagmus in overexposed workers (concentrations not reported). In the prior air contaminants rulemaking, one commenter (Ex. 3–830) was of the opinion that the 50-ppm ceiling limit should apply only to those workplaces where ethylene glycol is used at elevated temperatures. In response to this commenter, OSHA reiterated the Agency's long-standing policy, which is to base its permissible exposure limits on scientific evidence that exposure to a substance at a given concentration or dose is associated with a health risk and that promulgating a PEL will reduce that risk. Thus, a relationship between

exposure level and degree of risk is established and is deemed applicable in all workplaces and situations where the substance is present. If the characteristics of a process are such that employee exposure to a substance is nonexistent or is well below the levels associated with a health risk, the promulgation of a limit on employee exposure will have little or no effect on the operation or process and will therefore impose no additional burden on the employer. Therefore, in the specific case of ethylene glycol, OSHA saw no reason to limit application of the 50-ppm ceiling limit to those processes where exposure to airborne ethylene glycol is most likely. Accordingly, the air contaminants rule for general industry established a 50-ppm ceiling limit for ethylene glycol that is in effect in all general industry workplaces.

Based on the toxicity evidence described above in humans and animals, OSHA is proposing a ceiling limit of 50 ppm for ethylene glycol in the construction, agriculture, and maritime industries; this concentration is just below the level at which clinical signs and symptoms have been noted in humans. OSHA considers these signs and symptoms, which include throat and respiratory irritation and headache, to be material impairments of health. The Agency preliminarily concludes that this limit is necessary to substantially reduce these significant risks for workers in the construction, maritime, and agriculture industries. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ETHYLIDENE NORBORNENE

CAS: 16219–75–3; Chemical Formula: C_9H_{12}

H.S. No. 1171

OSHA has no limit for ethylidene norbornene in the construction, agriculture, and maritime industries. The ACGIH has a TLV® of 5 ppm as a ceiling limit; NIOSH has no REL for this substance but concurs (Ex. 8–47) with the limit being proposed. The Agency is proposing a 5-ppm ceiling limit for ethylidene norbornene in the construction, agriculture, and maritime industries; this is the limit for ethylidene norbornene recently established in general industry.

Ethylidene norbornene is a colorless liquid with a turpentine-like odor. It is used in the manufacture of pesticides and pharmaceuticals (ACGIH 1986, p. 261; HSDB 1987; Hazardous Substance Fact Sheet 1985, p. 1).

Ethylidene norbornene is an irritant of the eyes and nose in humans and causes systemic injury in laboratory animals. The oral LD_{50} in rats is 2527 mg/kg, the 48-hour LC_{50} in the same species is 1246 ppm, and the dermal LD_{50} in rabbits is 8189 mg/kg (RTECS 1989). Applied to the skin of rabbits, ethylidene caused a mild degree of irritation (RTECS 1989). In a range-finding study, five of six rats died following a 4-hour exposure to a 4000-ppm concentration of ethylidene norbornene (Smyth, Carpenter, Weil et al. 1969/Ex. 1–442). Exposure to a 237-ppm concentration of ethylidene norbornene for 7/hours/day, 5 days/week for 88 days was lethal to 21 of 24 rats; at autopsy, liver lesions, testicular atrophy, and hydrothorax were seen (Kinkead, Pozzani, Geary, and Carpenter 1971/Ex. 1–606). No deaths occurred in rats repeatedly exposed to a concentration of 90 ppm, but renal lesions and enlarged livers were observed in these animals at autopsy (Kinkead, Pozzani, Geary, and Carpenter 1971/Ex. 1–606). Beagle dogs exposed on the same regimen to a 93-ppm concentration of ethylidene norbornene for 89 days survived but showed such post-mortem effects as testicular atrophy, hepatic lesions, and slight blood changes; less pronounced effects were seen after dogs were exposed to a 61-ppm concentration, and no effects were seen at 22 ppm (Kinkead, Pozzani, Geary, and Carpenter 1971/Ex. 1–606).

Human volunteers exposed to ethylidene norbornene concentrations of 11 ppm for 30 minutes experienced eye and nose irritation; even at a concentration of 6 ppm, transient eye irritation occurred (ACGIH 1986/Ex. 1–3, p. 261).

Based on this evidence in humans and animals, OSHA is proposing a 5-ppm ceiling limit for this substance in the construction, agriculture, and maritime industries. The Agency preliminarily concludes that this limit is necessary to minimize the risk of material health impairment in the form of irritation that has been documented to occur during occupational exposures to ethylidene norbornene concentrations as low as 6 ppm for 30 minutes. The Agency preliminarily concludes that the promulgation of this limit will reduce this risk substantially and will additionally make OSHA's PEL for this substance consistent across all OSHA-regulated sectors.

FLUORINE

CAS: 7782–41–4; Chemical Formula: F

H.S. No. 1179

OSHA's PEL for fluorine in general industry, construction, and maritime is 0.1 ppm as an 8-hour TWA; NIOSH has no REL for fluorine. There is no PEL in agriculture. The ACGIH TLV®–TWA is 1 ppm and TLV®–STEL is 2 ppm. OSHA is proposing an 8-hour TWA PEL of 0.1 ppm for fluorine in agriculture. Promulgation of this limit will make the PEL for fluorine consistent across all regulated sectors.

Fluorine is a pale yellow gas with a pungent irritating odor.

Fluorine is a severe caustic and irritant of the eyes, nose, upper respiratory tract, and skin in humans and animals. The LC_{50} in rats is 185 ppm for 1 hour; acutely poisoned animals showed signs of severe eye and nose irritation before death and, at autopsy, showed pulmonary congestion and hemorrhage (RTECS 1991; Ricca 1970). On sublethal exposure, mice showed liver and kidney damage at autopsy (Keplinger and Suissa 1968).

In contact with moisture, fluorine reacts to form ozone and hydrofluoric acid. Volunteers exposed to a 50 ppm concentration of fluorine found the exposure intolerable. Subjects could tolerate a brief exposure to 25 ppm, but subsequently developed sore throats and pain in the chest that lasted for 6 hours (AIHA 1956). Pulmonary edema may follow overexposure to fluorine (Parmeggiani 1983, pp. 891–894). A recent study (Nemeth and Zsogon 1989) reports that long-term occupational exposure to fluorine causes osteosclerosis, especially of the spine and pelvic bones, and calcification of the spinal ligaments.

Based on this evidence in humans and animals, OSHA preliminarily concludes that workers in agriculture who are exposed to fluorine are at significant risk of severe irritation of the eyes, nose, and respiratory tract. The Agency considers these effects material impairments of health and believes that the proposed PEL of 0.1 ppm as an 8-hour TWA is necessary to substantially reduce the risk of these effects. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

FORMIC ACID

CAS: 64–18–6; Chemical Formula: HCOOH

H.S. No. 2086

In general industry, construction, and maritime, OSHA's permissible exposure limit for formic acid is 5 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 5 ppm for this substance; NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL in agriculture of 200 ppm for formic acid. Promulgation of this limit will make the PEL for this substance consistent across all regulated sectors.

Formic acid is a colorless, fuming liquid with a pungent, penetrating odor. It is used as a fumigant, an animal feed additive, a food preservative, a flavor adjunct, and in the manufacture of several substances, including insecticides, refrigerants, solvents, lacquers, and decalcifiers. Formic acid also is used in the textile, paper, and leather industries (HSDB 1986; ACGIH 1986, p. 279). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Formic acid is irritating and corrosive to the skin, eyes, mucous membranes, and respiratory tract; it has caused narcosis in laboratory animals and methemoglobinemia in dogs. The oral LD_{50}s in rats, dogs, and mice are 1100 mg/kg, 4000 mg/kg, and 700 mg/kg, respectively, and the intraperitoneal LD_{50} for mice is 940 mg/kg (RTECS 1990; Clayton and Clayton 1982, p. 4906). Rabbits exposed intravenously to formic acid in doses ranging from 0.46 to 1.25 mg/kg suffered central nervous system depression, vasoconstriction, and diuresis. Increasing the dose to approximately 4 g/kg caused convulsions and death in rabbits, and dogs given this dose showed methemoglobinemia (Clayton and Clayton 1982, p. 4906). Administration of formic acid to monkeys has produced the same type of toxicity to the optic nerves as is seen in systemic methanol poisoning (Grant 1986, pp. 447–448). Instilled into the eyes of rabbits, formic acid caused severe burns (RTECS 1990). Reduction in body weight gain and organ size was seen in young rats that had been given 0.5 to 1.0 percent formic acid in their food or drinking water for 6 weeks. Rats given 360 mg/kg formic acid in their drinking water for 2 to 27 weeks had a reduced rate of body weight gain and a reduced food intake but did not show other adverse effects (Clayton and Clayton 1982, p. 4906).

In an industrial accident that resulted in a fatality, a worker splashed in the face with hot formic acid experienced marked dyspnea with dysphagia; death occurred 6 hours following exposure and was caused by pulmonary edema (Proctor, Hughes, and Fischman 1988, p. 262). Workers exposed to a mixture of formic and acetic acid at 15 ppm concentrations of each acid complained of nausea and considerable irritation (ACGIH 1986, p. 279; Clayton and Clayton 1982, p. 4906). Ingestion of formic acid causes salivation, vomiting, a burning sensation in the mouth and pharynx, and severe pain; circulatory collapse and death may follow (Clayton and Clayton 1982, p. 4906). In contact with the skin, formic acid causes burns with vesiculation, and keloid formation is common (Proctor, Hughes, and Fischman 1988, p. 262).

Based on this evidence in humans and animals, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing irritation and burning of the skin, eyes, mucous membranes, and respiratory tract associated with exposure to formic acid. The Agency believes that establishing an 8-hour TWA PEL of 5 ppm in agriculture is necessary to provide agriculture workers with the same degree of protection being afforded workers in the other sectors covered by OSHA.

FURFURAL

CAS: 98–01–1; Chemical Formula: $C_5H_4O_2$

H.S. No. 1183

OSHA's PEL for furfural in the construction and maritime industries is an -hour TWA limit of 5 ppm, with a skin notation. The Agency has no PEL for this substance in agriculture. The ACGIH has a TLV®–TWA of 2 ppm, with a skin notation, for furfural; there is no NIOSH REL for this substance. OSHA is proposing a TWA PEL of 2 ppm, with a skin notation, for furfural in the construction, agriculture, and maritime industries; this is the limit recently established for this substance in general industry.

Furfural, also called furfuraldehyde, is a colorless, oily liquid that darkens to reddish-brown on exposure to light and air. Furfural is used as an extraction solvent in butadiene and lubricating oil production, as an intermediate in the production of furfuryl alcohol, and as a solvent for synthetic and natural resins (HSDB 1986). It is also used as a laboratory reagent, weed killer, fungicide, and as a flavoring agent in foods (HSDB 1986). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Furfural is a severe irritant of the eyes, mucous membranes, and respiratory tract; in contact with the skin, this substance may cause dermatitis, skin sensitization, and photosensitization (Proctor, Hughes, and Fischman 1988, p. 263). Furfural is also a carcinogen in experimental animals. The oral LD_{50} in rats is 65 mg/kg, the 48-hour LC_{50} in the same species is 153 ppm, and the lowest lethal dermal dose in rabbits is 620 mg/kg (RTECS 1991). Rats acutely poisoned by oral administration of furfural showed weakness, ataxia, and coma before death (Proctor, Hughes, and Fischman 1988, p. 263). Exposure to a 260-ppm concentration of furfural was fatal to rats but not to mice or rabbits (AIHA 1978). Rabbits given subcutaneous injections of furfural developed hypochronic anemia, with leukopenia, and had deficits in liver and kidney function (Gosselin, Smith, and Hodge 1984, p. II–187). A 4-week exposure of dogs to a 130 ppm concentration of furfural for 6 hours/day caused fatty degeneration of the liver, but no adverse effects were observed when dogs were exposed to 63 ppm on the same regimen (AIHA 1978). A 2-year carcinogenicity bioassay showed that furfural is clearly carcinogenic in male mice and suggested that this substance is also carcinogenic in male rats and female mice (NTP 1990; Fifth Annual Report). Furfural also has induced mutations in the somatic cells of hamster ovaries (RTECS 1991).

Bugyi and Lepold (1949/Ex. 1–1077) described numbness of the tongue and oral mucosa, absence of a sense of taste, and labored breathing in workers exposed to furfural (at unspecified levels) in a poorly ventilated facility. Korenman and Resnik (1930, as cited in ACGIH 1986/Ex. 1–3, p. 280) reported that workers inhaling furfural concentrations of from 1.9 to 14 ppm developed headache, scratchy throat, and eye irritation; Kuhn (1944/Ex. 1–3) reported that exposure to furfural also damages the eyesight of some exposed individuals. NIOSH (1975e/Ex. 1–1183) described widespread eye and respiratory tract irritation among workers at a grinding wheel plant who were exposed to furfural vapors at concentrations ranging from 5 to 16 ppm. However, a study by Dunlop and Peters (1953/Ex. 1–1189) reports that 15 years of furfural use in the synthetic resin industry did not cause adverse health consequences in exposed workers if the facility was adequately ventilated; this study also showed that furfural occasionally causes sensitization reactions.

Based on this evidence in humans and animals, OSHA preliminarily concludes that the current 8-hour TWA limit of 5 ppm is not sufficient to protect workers in the construction, maritime, and agriculture industries from eye and respiratory tract irritation; this fact is clearly shown by the NIOSH study (1975e/Ex. 1–1183), in which widespread irritation was reported among workers exposed to furfural concentrations ranging from 5 to 16 ppm. Therefore, to protect workers in these sectors from experiencing eye and respiratory tract

irritation, OSHA is proposing a PEL for furfural of 2 ppm as an -hour TWA in the construction, agriculture, and maritime industries. OSHA is also retaining the skin notation in construction and maritime and proposing it in agriculture because furfural readily penetrates the skin to cause toxic effects. OSHA preliminarily concludes that the proposed limit is necessary to substantially reduce a significant risk of material health impairment in exposed workers. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

FURFURYL ALCOHOL

CAS: 98–00–0; Chemical Formula: $C_5H_6O_2$

H.S. No. 1184

OSHA's limit for furfuryl alcohol in the construction and maritime industries is 50 ppm as an 8-hour TWA. The Agency has no PEL in agriculture for this substance. The ACGIH has a TLV®–TWA of 10 ppm and a TLV®–STEL of 15 ppm, with a skin notation, for furfuryl alcohol; the NIOSH REL is 50 ppm as a 108-hour TWA. OSHA is proposing a 10 ppm 8-hour TWA and a 15 ppm 15-minute STEL, with a skin notation, for this substance in the construction, agriculture and maritime industries. These are the limits recently established for furfuryl alcohol in general industry.

Furfuryl alcohol is a colorless liquid that turns red or brown on exposure to light and air. It is used as a wetting agent, as a solvent for dyes, gums, and resins, and as an ingredient in flavorings, foundry cores, polymers, sealants, and cements (ACGIH 1986, p. 281). Furfuryl alcohol is also a chemical intermediate and a liquid propellant for missiles (HSDB 1984).

Furfuryl alcohol causes irritation of the eyes and respiratory tract in both animals and humans; exposure to high concentrations also causes central nervous system depression. The oral LD_{50} in rats is 88 mg/kg, the LC_{50} in the same species is 233 ppm for 4 hours, and the dermal LD_{50} in rabbits is 400 mg/kg (RTECS 1991). Rats exposed to a 760-ppm concentration of furfuryl alcohol showed signs of eye irritation and narcosis, and rats exposed daily to a concentration of approximately 19 ppm exhibited signs of moderate respiratory irritation (Comstock 1952). Contact of the liquid with the eyes of rabbits caused corneal injury, with opacity and reversible inflammation (NIOSH 1979). Experiments on laboratory animals indicated that percutaneous absorption of the liquid can cause toxic effects (NIOSH 1979).

Furfuryl alcohol exposure has been shown to cause sensory irritation in exposed workers. Apol (1973/Ex. 1–1180) reported that workers exposed to a 10.8-ppm concentration of furfuryl alcohol experienced no discomfort but that severe lacrimation occurred at a concentration of 15.8 ppm. Formaldehyde was also present at a concentration of 0.33 ppm. Burton and Rivera (1972/Ex. 1–944) found no irritation, headache, or dizziness among workers exposed to -hour TWA furfuryl alcohol concentrations of 5 and 6 ppm, with excursions up to 16 ppm. A recent study by Cockcroft et al. (1980, as cited in Ex. 150) reports that a 50-year-old moldmaker developed asthma after working with a mixture containing furfuryl alcohol, paraformaldehyde, xylene, and a catalyst containing sulfuric acid, phosphoric acid, and butyl alcohol. The patient's bronchial response to inhaled histamines was two to three times more severe following exposure to furfuryl alcohol mixed with butyl alcohol.

In the recent air contaminants rulemaking for general industry, one commenter (Ex. 3–349) stated that, in his opinion, the proposed 10 ppm and 15 ppm TWA/STEL limits for furfuryl alcohol were too low. This commenter believed that the irritation reported in the Apol et al. study was caused not by furfuryl alcohol but by formaldehyde, which was present at a concentration of 0.33 ppm. In response to this commenter, OSHA noted that severe irritation and lacrimation occur in most individuals only when the formaldehyde levels reach 3 ppm or above; at levels between 0.1 and 0.5 ppm, slight eye irritation may occur in some individuals (52 *FR* 46235). In the foundry study by Apol (1973/Ex. 1–1180), formaldehyde was present at a concentration of 0.33 ppm, about 10 times below the level associated with severe eye irritation. Therefore, OSHA concluded in the rulemaking that these workers' exposure to furfuryl alcohol at a concentration of about 16 ppm was most likely to be the cause of the lacrimation reported by Apol (1973/Ex. 1–1180).

Based on this evidence in humans and animals, OSHA preliminarily concludes that severe eye irritation is associated with exposure to concentrations of about 16 ppm furfuryl alcohol, and that furfuryl alcohol is capable of inducing more serious asthmatic responses in at least some workers. OSHA believes that the severe eye irritation and asthma caused by exposure to furfuryl alcohol are material impairments of health and functional capacity, and the Agency is accordingly proposing PELs for this substance in the construction, maritime, and agriculture industries of 10 ppm as an 8-hour TWA and 15 ppm as a 15-minute STEL, with a skin notation, to substantially reduce these significant risks among exposed employees. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

GLUTARALDEHYDE

CAS: 111–30–8; Chemical Formula: $OCH(CH_2)_3CHO$

H.S. No. 1187

OSHA has no limit for glutaraldehyde in the construction, agriculture, and maritime industries. The ACGIH has a TLV® for this substance of 0.2 ppm as a ceiling; there is no NIOSH REL. The Agency is proposing a ceiling limit of 0.2 ppm for glutaraldehyde in the construction, agriculture, and maritime industries. This is the limit recently established for glutaraldehyde in general industry, and NIOSH (Ex. 8–47) concurred that this is an appropriate limit for this substance.

Glutaraldehyde is a colorless aqueous solution with a pungent odor. It is used as a disinfectant in cold sterilization of medical equipment, as an intermediate and fixative for tissues, to crosslink polyhydroxy materials and proteins, as an embalming fluid, and in the tanning industry (ACGIH 1986, p. 285; HSDB 1985). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Glutaraldehyde is strongly irritating to the nose, eyes, and skin and can cause allergic contact dermatitis, dermal sensitization, and asthma from occasional or incidental occupational exposure (Jordan, Dahl, and Albert 1972/Ex. 1–1056). The oral LD_{50} in rats is 134 mg/kg, the LC_{50} in the same species is 5000 ppm for 4 hours, and the dermal LD_{50} in rabbits is 2560 mg/kg (RTECS 1991). Instilled into the eyes of rabbits, a 25 percent solution of glutaraldehyde caused severe injury (graded 9 on an ascending severity scale of 1 to 10) (Grant 1986, p. 462). In contact with the skin of rabbits, glutaraldehyde causes a moderate degree of irritation (Clayton and Clayton 1981, p. 2654). A dose-dependent contact hypersensitivity response to glutaraldehyde has recently been demonstrated in guinea pigs and mice (Stern, Holsapple, McCay, and Munson 1989). Mice exposed to alkalinized glutaraldehyde at concentrations of 8 or 33 ppm for 24 hours have shown marked nervous behavior, with panting and compulsive washing of the face and limbs; those

exposed to 33 ppm exhibited signs of toxic hepatitis at autopsy (Varpela, Otterstrom, and Hackman 1971/Ex. 1–1072).

There is considerable evidence that occupational exposure to glutaraldehyde causes contact dermatitis, allergic skin sensitization, and respiratory sensitization. The groups primarily affected are health care workers and morticians. In a study of a cold-sterilizing operation in which the operator was exposed for 12 minutes to an activated 2-percent aqueous solution of glutaraldehyde, a measurement of 0.38 ppm glutaraldehyde was taken in the operator's breathing zone; the operator and the investigators experienced severe eye, nose, and throat irritation as well as sudden headache at the end of the sterilization procedure (Schneider and Blejer 1973, as cited in ACGIH 1986/Ex. 1–3, p. 285). A series of recent studies (Wiggins, McCurdy, and Zeidenberg 1989; Fowler 1989; Burge 1989; Stern, Holsapple, and Munson 1989; Norback 1988; Nethercott and Holness 19; Nethercott, Holness, and Page 1988) has demonstrated that occupational exposure to glutaraldehyde causes chest tightness, headaches, eye and throat irritation, contact dermatitis, nausea, airway obstruction, and asthma; patch tests confirmed the allergenicity of glutaraldehyde in many of these cases.

Based on this human and animal evidence, OSHA preliminarily concludes that glutaraldehyde clearly presents a significant risk of irritation of the eyes, nose, and throat, respiratory symptoms, nausea, headache, skin sensitization, and asthma to exposed workers. OSHA considers these exposure-related effects to be material impairments of health. Accordingly, OSHA believes that the proposed 0.2-ppm ceiling limit is necessary to reduce these significant risks among workers in construction, maritime, and agriculture. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

HEXACHLOROCYCLOPENTADIENE
CAS: 77–47–4; Chemical Formula: C_5Cl_6
H.S. No. 1196

OSHA has no PEL for hexachlorocyclopentadiene in the construction, agriculture, and maritime industries. The ACGIH has a TLV®–TWA of 0.01 ppm for this substance; there is no NIOSH REL. The Agency is proposing a TWA PEL of 0.01 ppm for hexachlorocyclopentadiene in the construction, agriculture, and maritime industries. OSHA recently established this PEL for hexachlorocyclopentadiene in general industry, and NIOSH (Ex. 8–47) concurred that this limit was appropriate for this substance.

Hexachlorocyclopentadiene is a yellow to amber-colored nonflammable liquid with a pungent odor. It is used primarily in the manufacture of chlorinated pesticides and flame retardants (ACGIH 1986, p. 300; HSDB 1985). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Hexachlorocyclopentadiene is an irritant of the eyes, skin, mucous membranes, and respiratory system; in laboratory animals, it has also been shown to cause systemic toxicity. The oral LD_{50} in rats is 113 mg/kg, the LC_{50} in the same species is 1600 ppb for 4 hours, and the dermal LD_{50} in rabbits is 430 mg/kg (RTECS 1991). Applied to the skin of monkeys, 10 mg of hexachlorocyclopentadiene caused severe irritation (RTECS 1991). Rabbits died after inhaling a 1.5-ppm concentration of hexachlorocyclopentadiene for 7 hours, rats died after five 7-hour exposures to a 1-ppm concentration, and guinea pigs died after two 7-hour exposures to 3.2 ppm; however, animals of all species survived 150 7-hour exposures to a hexachlorocyclopentadiene concentration of 0.15 ppm (Treon, Cleveland, and Cappel 1955/Ex. 1–497). Acutely poisoned animals showed tearing, labored breathing, and, at high concentrations, tremors (Treon, Cleveland, and Cappel 1955/Ex. 1–497). Treon and associates (1955/Ex. 1–497) noted degenerative changes in the brain, heart, liver, adrenal glands, and kidneys, and signs of pulmonary damage in these animals at autopsy, even in those animals exposed to the lowest concentration of 0.15 ppm. In animals exposed to higher concentrations, pulmonary edema, hyperemia, necrotizing bronchitis, and bronchiolitis were observed (Treon, Cleveland, and Cappel 1955/Ex. 1–497).

In humans, there are few data concerning hexachlorocyclopentadiene's toxicity. Irritation is known to occur, but the intolerable odor and eye irritation associated with exposure to this substance have discouraged prolonged exposures (McGilvray 1971, as cited in ACGIH 1986/Ex. 1–3, p. 300). In a group of 145 sewage treatment plant workers exposed to hexachlorocyclopentadiene after a large amount of this substance was dumped into the sewage system, 59 percent reported experiencing eye irritation, 45 percent developed headaches, and 27 percent experienced throat irritation (Clayton and Clayton 1981, p. 3751). Clinical examination of these workers 3 days after the incident revealed proteinuria and elevated serum lactic dehydrogenase levels; these effects were no longer present three weeks later (Clayton and Clayton 1981, p. 3751).

The TWA PEL of 0.01 ppm that OSHA is proposing for the construction, maritime, and agriculture industries for this severely toxic substance is about 10 times below the level associated with systemic damage and pulmonary irritation in experimental animals. This is the limit recently established for hexachlorocyclopentadiene in general industry. OSHA preliminarily concludes that employees in construction, maritime, and agriculture are at significant risk of experiencing intense eye and pulmonary irritation, headaches, and potential organ damage and that the proposed PEL is necessary to substantially reduce these risks.

HEXACHLOROETHANE
CAS: 67–72–1; Chemical Formula: CCl_3CCl_3
H.S. No. 1197

OSHA's PEL for hexachloroethane in construction and maritime is a 1-ppm TWA, with a skin notation. The 197–19 ACGIH TLV®–TWA is 1 ppm. The NIOSH REL for this substance is the lowest feasible level, based on hexachloroethane's potential carcinogenicity. OSHA is proposing an 8-hour TWA PEL of 1 ppm for hexachloroethane in agriculture, with a skin notation. Promulgation of this limit will make OSHA's PEL for this substance consistent across all industry sectors.

Hexachloroethane is a nonflammable white solid. This substance finds limited use in veterinary medicine and as a pesticide and is also used to make pyrotechnics and smoke devices (ACGIH 1986, p. 301(87)). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Hexachloroethane is an eye and upper respiratory tract irritant. The oral LD_{50} in rats is 4460 mg/kg (RTECS 1990). A study by Weeks, Angerhofer, Bishop, et al. (1979/Ex. 1–400) reported no adverse effects among several animal species exposed daily to 15- or 48-ppm concentrations of hexachloroethane for a period of 6 weeks. An NCI study (NCI 1978b/Ex. 1–949) reported that "extremely heavy dosages * * * administered continuously for a long period of time" resulted in the development of hepatocellular tumors in mice but not in rats. In 1978, NIOSH

reviewed the results of the NCI (1978b/Ex. 1–949) bioassay discussed above. Both male and female mice in this study exhibited an excess incidence of hepatocellular carcinoma, but rats did not. NCI concluded that early mortality may have obscured detection of a carcinogenic effect in rats (NCI 1978b/Ex. 1–949). Kidney damage was also found in mice and rats treated with hexachloroethane. Based on this evidence, NIOSH (1978r) recommends that exposure to hexachloroethane be maintained at the lowest detectable level. The International Agency for Research on Cancer (IARC) concluded, after reviewing the available literature on this substance, that the evidence for the carcinogenicity of hexachloroethane in animals was limited (IARC 1979, Vol. 20, p. 473).

Little information is available on human responses to hexachloroethane exposure. Workers exposed to the fumes from heated chloroethane developed blepharospasm, photophobia, tearing of the eyes, and hyperemia; these effects cleared completely after the cessation of exposure (Grant 1986, p. 479). Exposure to high but unspecified concentrations of hexachloroethane dust are irritating (Torkelson and Rowe, in Clayton and Clayton 1981).

OSHA preliminarily concludes that exposure to hexachloroethane poses a significant risk of irritation and perhaps of kidney damage and cancer to workers in agriculture. OSHA therefore proposes an 8-hour TWA PEL of 1 ppm, with a skin notation, for this substance in agriculture. The Agency believes that this limit is necessary to substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

sec-HEXYL ACETATE
CAS: 108–84–9
H.S. No. 2090

In general industry, construction, and maritime, OSHA's permissible exposure limit for sec-hexyl acetate is 50 ppm as an 8-hour TWA. There is no limit for this substance in agriculture. The ACGIH has a TLV®–TWA of 50 ppm for sec-hexyl acetate; NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL of 50 ppm for sec-hexyl acetate in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all regulated sectors.

sec-Hexyl acetate is a colorless liquid with a mild, fruity odor. The principal use for this substance is as a solvent in the lacquer industry. It is also used as a fragrance in the cosmetic and perfume industry and as a component in housefly insecticides and beetle attractants (HSDB 19; Clayton and Clayton 1982, p. 2278; Proctor, Hughes, and Fischman 1988, p. 272). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

sec-Hexyl acetate is an irritant of the eyes, mucous membranes, and upper respiratory tract in both animals and humans; at high concentrations, it causes narcosis in animals. The oral LD_{50} in rats is 6160 mg/kg; the dermal LD_{50} for rabbits is 20 gm/kg; and the lowest lethal concentration in rats is 2000 ppm for 4 hours (RTECS 1990). All rats exposed to an 8000-ppm concentration of sec-hexyl acetate died after a 48-hour exposure, while four out of six rats exposed to a 4000-ppm concentration for 4 hours survived (Smyth et al. 1954, pp. 61–68). Application of this substance to the skin of rabbits caused moderate irritation; instillation into the eyes of rabbits caused only minor corneal injury (Smyth et al. 1954, pp. 61–68; Carpenter, et al. 1974, pp. 313–319). Exposure to excessive concentrations of acetates causes eye, nose, and throat irritation and the gradual onset of narcosis in both animals and humans; recovery is slow after exposure is terminated (Clayton and Clayton 1982, p. 2268).

Human subjects experienced eye irritation on exposure to a 100-ppm concentration of sec-hexyl acetate for 15 minutes; concentrations higher than 100 ppm also caused nose and throat irritation (Silverman, et al. 1946, pp. 262–266). No chronic effects have been reported in humans (Proctor, Hughes, and Fischman 1988, p. 272).

Based on this evidence in humans and animals, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing the eye, nose, and throat irritation associated with exposure to sec-hexyl acetate and that they may also be at significant risk of experiencing central nervous system depression. The Agency believes that establishing an 8-hour TWA PEL of 50 ppm is necessary to substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

HEXYLENE GLYCOL
CAS: 107–41–5; Chemical Formula: $(CH_3)_2$-$COHCH_2$-$CHOH$–CH_3
H.S. No. 1204

OSHA has no limit for hexylene glycol in the construction, agriculture, and maritime industries. The ACGIH has a TLV® of 25 ppm as a ceiling limit; there is no NIOSH REL. The Agency is proposing a ceiling PEL of 25 ppm for hexylene glycol in the construction, agriculture, and maritime industries. OSHA recently established this PEL in general industry, and NIOSH (Ex. 8–47) concurred that this limit was appropriate for hexylene glycol.

Hexylene glycol is a colorless liquid that has a mild, sweet odor. It is used as an intermediate, a selective solvent in petroleum refining operations, and as a component of inks, hydraulic fluids, cosmetics, and cements (ACGIH 196, p. 309; Sittig 1985, p. 501).

Hexylene glycol is an irritant of the eyes, skin, and upper respiratory tract; in laboratory animals, it has also been shown to cause central nervous system depression and systemic toxicity. In rats, the oral LD_{50} is 3700 mg/kg; a dermal LD_{50} of 8560 mg/kg has been reported for rabbits (RTECS 1991). Undiluted hexylene glycol instilled into the rabbit eye caused irritation and corneal injury (Smyth and Carpenter 1948/Ex. 1–375). In contact with the skin for 24 hours, 500 mg caused moderate irritation in rabbits (RTECS 1991). Narcosis was produced in mice following a single oral dose of 2.0 ml/kg, and higher doses caused profound central nervous system depression (Smyth and Carpenter 1948/Ex. 1–375). Studies have shown that chronic feeding of this substance causes slight liver and kidney damage in experimental animals (Gosselin, Smith, and Hodge 1984, p. II–178).

At high concentrations, hexylene glycol vapors evoke a strong sensory response in humans: a 5-minute exposure to a 1000-ppm concentration produced eye irritation and throat and upper respiratory tract discomfort. At concentrations of 50 ppm for 15 minutes, most individuals experience slight eye irritation (ACGIH 1986/Ex. 1–3, p. 309). Subjects voluntarily ingesting 5 g hexylene glycol daily for 5 days failed to show adverse effects or urinary abnormalities (Gosselin, Hodge, and Smith 1984, p. II–179).

Based on this evidence in humans and animals, OSHA preliminarily concludes that workers exposed to this substance are at significant risk of experiencing exposure-related effects. The Agency believes that establishing a 25-ppm ceiling limit for hexylene glycol is necessary to substantially reduce these significant risks for workers in construction, maritime, and agriculture. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

HYDROGEN BROMIDE

CAS: 10035–10–6; Chemical Formula: HBr
H.S. No. 1206

The OSHA PEL for hydrogen bromide in the construction and maritime industries is 3 ppm as an 8-hour TWA. The Agency has no PEL for this substance in agriculture. The ACGIH has a TLV® of 3 ppm as a ceiling; there is no NIOSH REL for hydrogen bromide. The Agency is proposing a 3-ppm ceiling limit for hydrogen bromide in the construction, agriculture, and maritime industries. OSHA recently established this PEL in general industry, and NIOSH (Ex. –47) concurred that this was the appropriate limit for hydrogen bromide.

Hydrogen bromide is a corrosive, colorless gas with an acrid odor. It is used in the manufacture of bromides, in organic synthesis, to dissolve certain ores, and as an alkylation catalyst (ACGIH 1986, p. 312). Hydrogen bromide also finds use as a laboratory reagent (HSDB 1985).

Hydrogen bromide is a severe eye, nose, and respiratory tract irritant. In contact with moisture, hydrogen bromide forms hydrobromic acid. Animal studies have demonstrated that hydrogen bromide has a considerably higher acute toxicity than hydrogen chloride (HCl) in mice and a somewhat higher acute toxicity than this chemical in rats (NIOSH 1977i/Ex. 1–1182). In mice, the LC_{50} is 814 ppm for 60 minutes; in rats, the LC_{50} is 2858 ppm for 60 minutes (RTECS 1991). In contact with the skin of rabbits, hydrobromic acid caused corrosive burns (HSDB 1985).

The chief toxic effect of hydrogen bromide in humans is severe primary irritation of the eyes, nose, throat, and lungs. Irritation begins within a few minutes on exposure to concen-trations of hydrogen bromide ranging from 3 to 6 ppm (Connecticut State Department of Health 1955, as cited in ACGIH 1986/Ex. 1–3, p. 312). In contact with the eyes, mucous membranes, or skin, hydrogen bromide solutions cause burns (Parmeggiani 1983, p. 327). Hydrogen bromide's mechanism of action is to form hydrobromic acid on contact with moisture; this acid then neutralizes the alkali of tissues and can cause death as a result of lung edema, laryngeal spasms, or upper respiratory tract inflammation (HSDB 1985). Skin contact in humans causes severe tissue irritation and necrosis (Braker and Mossman 1980, p. 373). Exposure to a 1300 to 2000-ppm concentration of hydrogen bromide is lethal within a few minutes (Braker and Mossman 190, p. 373). A laboratory assistant splashed with a mixture of bromine, phosphorus tribromide, and hydrogen bromide developed cough, lightheadedness, throat congestion, shortness of breath, and bibasilar crackles within 2 weeks of the episode. The patient was hospitalized with bilateral lower-lobe chemical pneumonitis. The authors of this case report believe that this worker subsequently developed bronchiolitis obliterans because her symptoms later recurred (Kraut and Lilis 1988; Chest 94(1):208–210).

Based on this evidence, OSHA preliminarily finds that workers are at significant risk of experiencing severe irritant effects on brief exposure to hydrogen bromide. The Agency considers these irritant effects material impairments of health and believes that the proposed ceiling limit of 3 ppm is necessary to protect workers in the construction, agriculture, and maritime industries from these significant risks. Promulgation of this limit will make OSHA's PEL for hydrogen bromide consistent across all OSHA-regulated sectors.

HYDROGEN CHLORIDE
CAS: 7647–01–0; Chemical Formula: HCl
H.S. No. 2091

In general industry, construction, and maritime, OSHA has a PEL of 5 ppm as a ceiling limit for hydrogen chloride. The Agency currently has no PEL for this substance in agriculture. The ACGIH has a TLV® of 5 ppm (7.5 mg/m³) as a ceiling limit for hydrogen chloride; NIOSH has no REL. OSHA is proposing a PEL of 5 ppm as a ceiling limit for hydrogen chloride in agriculture; promulgation of this limit would mean that the PEL for hydrogen chloride in all OSHA-regulated industry sectors is the same.

Hydrogen chloride is a colorless, corrosive, nonflammable gas with a pungent, suffocating odor. Aqueous solutions of various concentrations are available commercially. Hydrogen chloride is used in the separation of cotton from wool; in the de-linting of cotton; as a catalyst for making oils viscous; in the activation of oil wells; in ore reduction, metal-pickling, and metal cleaning; in food processing; and in many other chemical processes (Braker and Mossman 1980, p. 378; ACGIH 1986, p. 313).

Hydrogen chloride is a strong irritant of the eyes, skin, mucous membranes, and respiratory tract; this substance also causes pulmonary sensitization. The LC_{50} in rats is 3141 ppm for 1 hour (RTECS 1990). The oral LD_{50} in rabbits is 900 mg/kg (RTECS 1990). Animals exposed to a hydrogen chloride concentration of 1350 ppm for 1.5 hours developed corneal clouding, and exposure to 300 ppm for 6 hours caused slight erosion of the corneal epithelium (Patty 1949). Rabbits, guinea pigs, and pigeons exposed to a 100-ppm concentration for 6 hours/day for 50 days exhibited excitation and signs of eye and nose irritation (Jones 1972; Faigel 1964). Rats and mice exposed to hydrogen chloride for 6 hours/day, 5 days/week for 90 days at a 50-ppm concentration showed significant decreases in body weight, and rats exposed to 10, 20, or 50 ppm showed inflammation of the nasal passages (Chemical Industry Institute of Toxicology 1984).

In humans, exposure to high concentrations of hydrogen chloride causes necrosis of the tracheal and bronchial epithelium, pulmonary edema, atelectasis, emphysema, damage to the pulmonary blood vessels, and damage to the liver and other organs (Machle 1944). Male volunteers found concentrations of 50 to 100 ppm barely tolerable for 1 hour; exposure to 35 ppm for brief periods caused irritation of the throat, and 10 ppm was considered the maximal acceptable concentration for prolonged exposures (Henderson 1943). Exposure to concentrations of 5 ppm or above resulted in immediate irritation of the nose and throat, while exposure to concentrations below 5 ppm produced no adverse effects (Elkins 1959). Inhalation of high concentrations of hydrogen chloride for less than 1 hour induced cough and dyspnea among subjects with no past history of asthma or atopy; the same exposure caused severe bronchospasm that required mechanical ventilation in the case of an asthmatic subject. All subjects continued to show hyperresponsiveness more than 1 year later (Boulet 1988). Splashed into the eye and washed out immediately, hydrogen chloride causes white coagulation of the cornea and conjunctival epithelium; however, the damaged epithelium is later replaced by new epithelium, leaving the eye normal (Grant 1986, p. 489). Contact of the skin with the liquid causes skin burns, and repeated skin exposure to dilute solutions may cause dermatitis (M.C.A. 1970). Workers exposed chronically to hydrogen chloride experience gastritis and chronic bronchitis (Bransburg 1946). Chronic exposure to low concentrations has also led to erosion of the enamel of exposed teeth (NAS 1976; Ludewig 1942).

Based on this evidence in humans and animals, OSHA proposes to establish a PEL of 5 ppm as a ceiling for hydrogen chloride in agriculture; adoption of this limit would establish the same PEL for workplaces in all OSHA-regulated industry sectors. The Agency

preliminarily concludes that occupational exposure to hydrogen chloride causes severe irritation of the eyes, skin, and upper respiratory tract. Accordingly, OSHA believes that, in the absence of a permissible exposure limit, workers in agriculture are potentially at significant risk for these exposure-related effects and that the proposed PEL is necessary to substantially reduce these risks.

HYDROGEN FLUORIDE

CAS: 7664–39–3; Chemical Formula: HF

H.S. No. 1208

The OSHA PEL for hydrogen fluoride in the construction and maritime industries is 3 ppm as an 8-hour TWA. The Agency has no limit for this substance in agriculture. The ACGIH has a TLV® of 3 ppm as a ceiling, and the NIOSH RELs for hydrogen fluoride are 3 ppm as a 108-hour TWA and 6 ppm as a 15-minute ceiling. The Agency is proposing a 3-ppm TWA PEL and a 6-ppm 15-minute STEL for hydrogen fluoride in the construction, agriculture, and maritime industries. These are the limits recently established for hydrogen fluoride in general industry, and NIOSH (Ex. 8–47) concurred with these limits in that rule-making.

Hydrogen fluoride is a fuming, colorless liquid; at temperatures above 19°C (66°F), it becomes a colorless gas. It is used as a catalyst in chemical synthesis, as a fluorinating agent, in fluorine and aluminum fluoride production, and in uranium refining operations (ACGIH 1986, p. 315). Hydrogen fluoride also finds use in dye chemistry, metal cleaning, crystal polishing, and iron enameling and galvanizing (HSDB 1985).

Hydrogen fluoride causes severe irritation and burns of the eyes, skin, mucous membranes, and respiratory tract; it can also be absorbed through the skin to cause systemic toxicity. Because fluorides accumulate in the bone, long-term exposure to hydrogen fluoride also can cause osteosclerosis (Rom 1983, p. 276). The LC_{50} in rats is 1276 ppm for 1 hour; in monkeys, the LC_{50} is 1774 ppm for 1 hour (RTECS 1990). Application of a 2-percent solution of hydrogen fluoride to the skin of rabbits for 1 hour caused necrosis (AIHA 1988; Emergency Response Planning Guidelines). Guinea pigs and rabbits survived exposure to hydrogen fluoride concentrations of 40 ppm for a total of 41 hours, but exposure to a concentration of 300 ppm for two hours or more was fatal (Machle, Thamann, Kitzmiller, and Cholak 1934/Ex. 1–519). Animals exposed to a 3-ppm concentration of hydrogen fluoride for 30 days showed no adverse effects (Ronzani 1909, as cited in ACGIH 1986/Ex. 1–3, p. 315). Stokinger (1949a, as cited in ACGIH 1986/Ex. 1–3, p. 315) reported that animals repeatedly exposed to a 7-ppm concentration of hydrogen fluoride on a daily basis exhibited mild respiratory tract irritation. A study by Largent (1961/Ex. 1–1158) demonstrated kidney, liver, and lung damage in laboratory animals repeatedly exposed to a 17-ppm concentration of hydrogen fluoride. At a concentration of 8.6 ppm, however, the pathologic changes seen in exposed animals were minor, except for lung damage in one dog (Largent 1961/Ex. 1–1158). Guinea pigs, rabbits, and monkeys exposed to an 18.5-ppm concentration of hydrogen fluoride for 6 to 8 hours/day, 5 days/week for 2 months showed signs of liver and kidney damage at autopsy; a few animals showed signs of lung damage as well (AIHA 1988).

Hydrogen fluoride is a severe irritant of the eyes, mucous membranes, and lungs in humans. Based on the results of studies with volunteers and findings from accidental overexposures, it is believed that a 5-minute exposure to a 50- to 250-ppm concentration of hydrogen fluoride would be fatal (Proctor, Hughes, and Fischman 1988, p. 278). Largent (1960/Ex. 1–516; 1961/Ex. 1–115) reported that volunteers exposed repeatedly to concentrations of hydrogen fluoride as high as 4.7 ppm for 6 hours/day for 10 to 50 days experienced irritation and burning of the eyes and nose, in addition to reddening of the skin, at all hydrogen fluoride concentrations above 3 ppm. Industrial experience has shown that direct contact of the skin with hydrogen fluoride results in severe burns that may have a delayed onset but later develop into ulcers that eventually scar (Stokinger 1981b/Ex. 1–1127). A report by Eagers (1969, as cited in Stokinger 1981b, above) described several industrial accidents in which workers died in a matter of hours after containers of hydrogen fluoride ruptured and caused accidental splashing of nearby workers (the cause of death was respiratory failure and cardiac arrest). Kleinfeld (1965/Ex. 1–514) reported on a similar case of hydrogen fluoride poisoning that resulted in death from pulmonary edema. When splashed on the skin, hydrogen fluoride can be absorbed in quantities sufficient to cause hypocalcemia and hypomagnesemia, followed by life-threatening cardiac arrhythmia (Proctor, Hughes, and Fischman 1988, p. 279). Chronic exposure to hydrogen fluoride can cause increased density of the bones, which is visible radiographically; this condition may lead to crippling osteosclerosis (Proctor, Hughes, and Fischman 1988, p. 279).

Based on this evidence in humans and animals, OSHA preliminarily finds that workers exposed to hydrogen fluoride are at significant risk of experiencing this substance's severe irritant and corrosive effects. The Agency believes that the effects caused by exposure to hydrogen fluoride constitute material impairments of health and that the proposed limits of 3 ppm as an 8-hour TWA and 6 ppm as a 15-minute STEL are necessary to substantially reduce these risks among workers in construction, maritime, and agriculture. These are the limits recently established for hydrogen fluoride in general industry.

HYDROGEN PEROXIDE

CAS: 7722–84–1; Chemical Formula: H_2O_2

H.S. No. 2092

In general industry, construction, and maritime, OSHA's permissible exposure limit for hydrogen peroxide is 1 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 1 ppm for this substance; NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL of 1 ppm for hydrogen peroxide in agriculture. This is the limit recently established for this substance in general industry.

Pure anhydrous hydrogen peroxide is a colorless, odorless liquid. It is commercially available in 30%, 35%, 50%, 70%, and 90% grades. Hydrogen peroxide is used in water and sewage treatment, as a disinfectant, as an intermediate in chemical synthesis, as a fuel in rockets, and as a bleaching agent and deodorizer for textiles, hair, fur, wood pulp, and foods (AIHA 1978; HSDB 1986; ACGIH 1986, p. 316). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Hydrogen peroxide is an irritant of the eyes, mucous membranes, skin, and respiratory tract in both animals and humans. There is also limited evidence that hydrogen peroxide is carcinogenic in animals (IARC 1985, Vol. 35, p. 303). The oral LD_{50} in mice is 2 g/kg, the dermal LD_{50} in rats is 4060 mg/kg, and the lowest lethal concentration in mice is 227 ppm (RTECS 1991). A 5-minute exposure of mice to approximately 3600 ppm of hydrogen peroxide resulted in pulmonary congestion and minor burns of the nose and jaws; a concentration of approximately 6500 ppm for the same period was lethal to these animals (AIHA 1978; Punte 1956). Sneezing, external body irritation, lacri-mation,

and bleaching of hair occurred in dogs exposed to a 7-ppm concentration of hydrogen peroxide (90-percent solution) for 6 hours/day for 6 months. Thickened skin and lung irritation were seen in these animals at autopsy, but no significant changes were noted in the blood or urine (Oberst et al. 1954). Applied to the eyes of rabbits, hydrogen peroxide causes irritation and corneal damage. The extent of eye damage depends on the concentration of hydrogen peroxide, with stronger concentrations causing greater damage (Grant 1986, p. 493). In oral bioassays, hydrogen peroxide induced adenomas and carcinomas of the duodenum in mice. The International Agency for Research on Cancer has concluded that the evidence for the carcinogenicity of hydrogen peroxide in animals is limited (IARC 1985, Vol. 35, p. 304).

Exposure to high concentrations of hydrogen peroxide vapor or mist causes severe irritation and inflammation of the nose and throat in humans (Proctor, Hughes, and Fischman 1988, p. 282). Dermal application of a 1- to 30-percent solution of hydrogen peroxide results in a characteristic whitening of the skin; if skin contact is prolonged, redness and blistering may occur (Hauschild et al. 1958; Proctor, Hughes, and Fischman 1988, p. 282). Application of a 1- to 3-percent solution of hydrogen peroxide to the eye causes severe pain and reversible injury (Grant 1986, p. 493).

Based on this evidence, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing eye, mucous membrane, skin, and respiratory tract irritation if exposed to hydrogen peroxide at the concentrations permitted by the absence of a limit. The Agency believes that establishing an 8-hour TWA PEL of 1 ppm is necessary to substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

2–HYDROXYPROPYL ACRYLATE

CAS: 999–61–1; Chemical Formula: $CH_2CHCOOCH_2CHOHCH_3$

H.S. No. 1211

OSHA has no limit for 2-hydroxypropyl acrylate in the construction, agriculture, and maritime industries. The ACGIH has a TLV®–TWA of 0.5 ppm, with a skin notation, for 2-hydroxypropyl acrylate; there is no NIOSH REL for this substance. The Agency is proposing a TWA PEL of 0.5 ppm, with a skin notation, for this substance in the construction, agriculture, and maritime industries. This PEL was recently established for 2-hydroxypropyl acrylate in general industry, and NIOSH (Ex. 8–47) concurred with this limit in that rulemaking.

2-Hydroxypropyl acrylate, also called HPA, is a colorless liquid that is used as a monomer for acrylic resins, a binder in nonwoven fabrics, and in the manufacture of thermosetting resins for surface coatings (Sittig 1985, p. 517; ACGIH 1986, p. 320).

2-Hydroxypropyl acrylate causes irritation, severe burns and corrosion of the eyes and skin, and skin sensitization in animals and humans. In experimental animals, 2-hydroxypropyl acrylate has a high acute toxicity. The oral LD_{50} in rats is 250 mg/kg; the dermal LD_{50} in rabbits is approximately 0.25 mg/kg (RTECS 1990; Dow Chemical Company 1977, as cited in ACGIH 1986/Ex. 1–3, p. 320). In guinea pigs, direct contact with HPA caused severe eye burns and skin corrosion and sensitized some of the animals. Rats exposed to an HPA concentration of 650 ppm for 7 hours survived. Longer-term inhalation studies (2 hours/day, 6 days/week for 30 days) in rats, dogs, rabbits, and mice showed that the animals displayed signs of sensory irritation at concentrations of 2-hydroxypropyl acrylate of 5 ppm and above (Dow Chemical Company 1977c, as cited in ACGIH 1986/Ex. 1–3, p. 320).

In humans, 2-hydroxypropyl acrylate has been shown to cause adverse skin effects. A recent study in individuals with allergic contact dermatitis reports that 3 of 24 exposed persons had become sensitized to HPA (Kanerva, Estlander, Jolanski 1988; Contact Dermatitis 18(1):1–5). Another study reports that a worker in an HPA manufacturing facility developed swelling, blisters, and an erythematous papular eruption on his foot, forearms, thighs, and groin after exposure to HPA. These lesions cleared after several days away from work but reappeared after the man returned to his job. This worker showed positive responses to patch testing with HPA and methacrylates; these results demonstrate that he had become sensitized to HPA and had developed a cross-sensitivity to methacrylates (Lovell, Rycroft, Williams, and Hamlin 1985; Contact Dermatitis 12(2):117–18).

Based on this evidence in humans and animals, OSHA preliminarily finds that the proposed limit is necessary to protect exposed workers in construction, maritime, and agriculture from the significant risks of irritation, skin and eye burns, and skin sensitization associated with exposure to 2-hydroxypropyl acrylate. The Agency considers these effects material impairments of health and preliminarily finds that the proposed TWA limit of 0.5 ppm, with a skin notation, is necessary to substantially reduce these risks. Promulgation of this limit in construction, maritime, and agriculture will make OSHA's limit for 2-hydroxypropyl acrylate consistent across all OSHA-regulated sectors.

IODINE

CAS: 7553–56–2; Chemical Formula: I

H.S. No. 2095

In general industry, construction, and maritime, OSHA's current permissible exposure limit for iodine is 0.1 ppm as a ceiling limit. OSHA has no limit in agriculture. The ACGIH TLV® for iodine is 0.1 ppm as a ceiling limit; NIOSH has no REL. OSHA is proposing a ceiling PEL in agriculture of 0.1 ppm for iodine. This is the limit recently established for this substance in general industry.

Iodine is the heaviest of the halogens used in industry. Elemental iodine is a crystalline solid that occurs in blue-black or grayish-black plates or scales. It sublimes when heated to form a violet-colored vapor in air. Iodine is used as a bactericide, fungicide, amebicide, topical antiseptic and disinfectant, and to decontaminate drinking water. It is also used in antihyperthyroid drugs; in biological, biochemical, and chemical research; in dyes and soaps; and in engraving and lithography procedures (HSDB 1984; Clayton and Clayton 1982, p. 2972; ACGIH 1986, p. 323). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Iodine is irritating and corrosive to the skin, eyes, mucous membranes, and upper respiratory tract in both animals and humans. The oral LD_{50} in rats is 14 g/kg; the lowest lethal concentration in rats is 800 mg/m^3 for 1 hour (RTECS 1991). Dogs exposed by intratracheal administration to iodine vapor at a dose of 36 mg/kg for 3 hours died; before death, signs of severe respiratory irritation were present, and autopsy revealed pulmonary edema and subpleural hemorrhage (Lockhardt, Koch, Schroeder, and Weiland 1920). Rats and guinea pigs exposed chronically to iodine vapors (concentration and duration not specified) showed reduced oxygen uptake and disrupted thyroid activity; at autopsy, the lungs of these animals were edematous and hemorrhagic (Begishev et al. 1976).

Industrial exposures to the vapor of iodine have shown this substance to be highly irritating at very low doses.

Exposure of four individuals to a concentration of 0.57 ppm for 5 minutes was without effect, but increasing this concentration to 1.63 ppm was irritating to the eyes within 2 minutes (AIHA 1965). Some workers experience moderate irritation at a concentration of 0.15 to 0.2 ppm and intolerable irritation at a 0.3-ppm concentration (ACGIH 1986, p. 323). The effects of overexposure to iodine include tearing, a feeling of tightness in the chest, sore throat, headache, and, if the overexposure is severe, respiratory symptoms equivalent to those associated with chlorine gassing (Lockhardt, Koch, Schroeder, and Weiland 1920). Chronic exposure to iodine leads to iodism, a condition characterized by tremor, insomnia, weight loss, conjunctivitis, bronchitis, tachycardia, and diarrhea (Seymour 1983; Peterson 1983).

Based on this evidence in humans and animals, OSHA preliminarily concludes that workers in agriculture are potentially exposed to a significant risk of eye, mucous membrane, and upper respiratory tract irritation and may also be at risk of developing iodism, a condition that can be life-threatening if untreated. OSHA believes that establishing a limit in agriculture for iodine at the proposed ceiling level of 0.1 ppm is necessary to protect agricultural workers from these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

IRON SALTS (SOLUBLE)

CAS: Varies with compound; Chemical Formula: Varies with compound

H.S. No. 1217

OSHA has no limit for soluble iron salts in the construction, agriculture, and maritime industries. The ACGIH has a TLV®–TWA of 1 mg/m³ for these substances; there are no NIOSH RELs. The Agency is proposing a TWA PEL of 1 mg/m³ for the soluble iron salts in the construction, agriculture, and maritime industries. This PEL was recently established in general industry, and NIOSH (Ex. 8–47) concurred that this PEL was appropriate for the soluble salts of iron.

The soluble salts of iron include ferric chloride, ferric nitrate, ferric sulfate, ferrous chloride, and ferrous sulfate (ACGIH 1986, p. 328). Ferric salts are used as astringents in treating some skin disorders, and both the ferrous and ferric salts are used in the textile industry and the dye industry (ACGIH 1986, p. 328).

The soluble salts of iron are irritants of the skin and respiratory tract; in large doses, they are corrosive and systemically toxic (ACGIH 1986, p. 328; Gosselin, Smith, and Hodge 1984, p. II–139). Iron salts are highly toxic by intravenous injection and moderately toxic by oral administration; their acute toxicity by inhalation has not been established. The oral LD_{50} in rats for ferric chloride is 172 mg/kg; for ferrous sulfate, the oral LD_{50} in rats is 318 mg/kg (Sax and Lewis 1989, p. 2015). By intravenous administration, the LD_{LO} for ferric chloride in rabbits is 7.2 mg/kg (ACGIH 1986, p. 328). Acutely poisoned animals show alkalosis, hypothermia, anorexia, oligodipsia, oliguria, diarrhea, and alternating irritability and depression before death. At autopsy, congestion of the gastrointestinal tract, liver, kidneys, heart, lungs, brain, spleen, adrenals, and thymus is seen (Clayton and Clayton 1981, p. 1665). Dogs administered 0.2 to 0.8 g of ferric chloride daily on a chronic regimen (duration not specified) failed to develop physiological changes (Clayton and Clayton 1982, p. 1667). When inhaled as dusts and mists, the iron salts are irritants of the respiratory tract (ACGIH 1986, p. 328). Stewart and Faulds (1934/Ex. 1–764) identified the ferric salts as skin irritants.

Although the oral toxicity of the iron salts is considered moderate, marked gastrointestinal irritation results from the ingestion of these salts by humans (U.S. Department of Labor 1941, as cited in ACGIH 1986/Ex. 1–3, p. 328). The estimated fatal dose for humans by ingestion is 30 grams (Smyth 1956/Ex. 1–759). Humans poisoned by iron salts develop the following signs and symptoms: Vomiting, hematemesis, diarrhea, lethargy, coma, irritability, seizures, abdominal pain, and increased cardiac and respiratory rates (Clayton and Clayton 1981, p. 1665). Contact of the eye with ferric or ferrous sulfate or ferric or ferrous chloride causes transient irritation and inflammation, and prolonged contact may lead to brown discoloration of the conjunctiva (Grant 1986, p. 532).

The Agency preliminarily concludes that, in the absence of a limit, employees in construction, maritime, and agriculture are at risk of experiencing the skin and mucous membrane irritation associated with inhalation exposure to these soluble salts. OSHA considers these adverse health effects to be material impairments of health and believes that a 1 mg/m³ 8-hour TWA PEL for the soluble iron salts is necessary to substantially reduce these significant occupational risks. Promulgation of this limit will also make the PEL for these substances consistent across all OSHA-regulated sectors.

ISOAMYL ACETATE

CAS: 123–92–2; Chemical Formula: $CH_3COOCH_2CH(CH_3)C_2H_5$

H.S. No. 2096

In general industry, construction, and maritime, OSHA's permissible exposure limit for isoamyl acetate is 100 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 100 ppm for this substance; NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL in agriculture of 100 ppm for isoamyl acetate. This is the limit recently established for this substance in general industry.

Isoamyl acetate is a colorless liquid with a banana-like odor. It is used widely as a solvent and flavoring agent and as an ingredient in fragrances, polishes, and photographic film. Isoamyl acetate also is used in the extraction of penicillin; as a warning odorant; and in the manufacture of artificial silk, leather, and glass (HSDB 1986; Parmeggiani 1983, p. 783).

In both animals and humans, isoamyl acetate is an irritant of the eyes, mucous membranes, and respiratory tract; at high concentrations, it causes central nervous system depression. Systemic effects also have been shown to occur in laboratory animals exposed to isoamyl acetate. The oral LD_{50} in rats is 16.6 g/kg, and the lowest lethal concentration in cats is 35,000 mg/m³ for an unspecified period (RTECS 1990). A 248-hour exposure to a 7200-ppm concentration of isoamyl acetate was fatal to cats; these animals showed signs of narcosis before death and died of pneumonia (Lehmann and Flury 1943, p. 228; Clayton and Clayton 1982, p. 2274). Changes in the liver, congestion and hypertrophy of the spleen, congestion of the kidney, and degenerative and reparative changes of the tubular epithelium were noted at autopsy in rabbits that had been subacutely poisoned by exposure to isoamyl acetate (Browning 1965, p. 540). In humans, a 30-minute exposure to a 1000-ppm concentration of isoamyl acetate resulted in irritation, difficult breathing, increased pulse rate, and fatigue (ACGIH 1986, p. 329). Severe throat irritation was reported as a result of exposure to 200 ppm, and minor throat discomfort resulted from exposure to a 100-ppm concentration of isoamyl acetate (Amoore 1950, p. 53; Nelson 1943, p. 282). Exposure to a 300-ppm concentration caused noticeable eye irritation, and exposure to higher concentrations caused burning and redness of the eyes (Grant 1986, p. 97). Workers in a hat factory exposed to isoamyl acetate at unspecified concentrations reported experiencing

nervous system disturbances, including headache, drowsiness, palpitation, and excessive fatigue (Browning 1965, p. 541).

Based on this evidence in humans and animals, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing the eye, mucous membrane, and upper respiratory tract irritation associated with exposure to isoamyl acetate and that they may also be at significant risk of experiencing central nervous system depression. The Agency believes that establishing an 8-hour TWA PEL of 100 ppm for this substance is necessary to substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ISOPROPYL ACETATE

CAS: 108–21–4; Chemical Formula: $CH_3COOCH(CH_3)_2$

H.S. No. 1224

OSHA has a 250 ppm 8-hour TWA limit for isopropyl acetate in the construction and maritime industries. The Agency has no PEL for this substance in agriculture. The ACGIH has a TLV®–TWA of 250 ppm and a TLV®–STEL of 310 ppm for isopropyl acetate; there is no NIOSH REL for this substance. The Agency is retaining its 250 ppm 8-hour TWA in construction and maritime, proposing to add a 310 ppm STEL for isopropyl alcohol in these two sectors, and proposing to extend both limits to agriculture. These are the limits recently established for isopropyl acetate in general industry.

Isopropyl acetate is a colorless liquid with a fruity odor. It is used as a solvent for nitrocellulose lacquers and other resins, fats, oils, waxes, and gums. It is also used in perfumes and as a flavoring agent for foods (Sittig 1985, p. 531; ACGIH 1986, p. 336).

Isopropyl acetate causes irritation of the eyes and mucous membranes; at high concentrations, it causes central nervous system depression in both animals and humans (Clayton and Clayton 1981, p. 2268; Gosselin, Smith, and Hodge 1984, p. II–203). The oral LD_{50} in rats is 3000 mg/kg; five of six rats died after a four-hour exposure to a 32,000-ppm concentration of isopropyl acetate, and one of six rats died after a 4-hour exposure to a 16,000-ppm concentration (Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1–440; RTECS 1991). The corneal burns caused by isopropyl acetate generally heal within 3 to 10 days (Clayton and Clayton 1981, p. 2277). Oral administration of isopropyl acetate to rabbits (dose not specified) caused loss of coordination (Clayton and Clayton 1981, pp. 2268–2269).

The primary problems associated with occupational exposure to isopropyl acetate are eye and mucous membrane irritation. In humans, exposure to a 200-ppm concentration of isopropyl acetate for 15 minutes caused mild eye irritation; nose and throat irritation occurred on exposure to higher concentrations (Silverman, Schulte, and First 1946/Ex. 1–142). In two cases involving splashes of isopropyl acetate in the eye, healing was slow but complete (Grant 1986, p. 539). Workers chronically exposed to isopropyl acetate at unspecified concentrations have reported experiencing conjunctival irritation, cough, and a tight feeling in the chest; skin defatting and cracking have also been reported (Parmeggiani 1983, p. 72).

Based on this evidence, OSHA preliminarily concludes that exposure to isopropyl acetate causes irritant effects. The Agency considers these effects material impairments of health and preliminarily finds that the proposed limits are necessary to reduce these significant risks to workers in construction, maritime, and agriculture. Accordingly, OSHA is retaining the 250-ppm 8-hour TWA limit in construction and maritime, proposing a 310-ppm STEL for this substance in these two sectors, and proposing both limits in agriculture. Promulgation of these limits will make OSHA's PELs for isopropyl acetate consistent across all regulated sectors.

ISOPROPYL ALCOHOL

CAS: 67–63–0; Chemical Formula: $CH_3CHOHCH_3$

H.S. No. 1225

The current PEL for isopropyl alcohol in the construction and maritime industries is 400 ppm as an 8-hour TWA. OSHA has no limit for this substance in agriculture. The ACGIH has a TLV®–TWA of 400 ppm and a TLV®–STEL of 500 ppm for isopropyl alcohol. NIOSH has a REL for isopropyl alcohol of 400 ppm as a 10-hour TWA and 800 ppm as a 15-minute ceiling limit but concurs (Ex. 150) with the limits being proposed by OSHA. The Agency is proposing a 400-ppm TWA and a 500-ppm STEL for isopropyl alcohol in the construction, agriculture, and maritime industries. OSHA recently established these PELs in general industry.

Isopropyl alcohol is a colorless, flammable liquid with the odor of rubbing alcohol. It is used as a starting material in the synthesis of acetone, glycerin, and other substances and is widely used throughout industry as a solvent, component of household products, ingredient in cosmetics and medicinals, as rubbing alcohol, and as a flavoring adjuvant (HSDB 1986; ACGIH 1986, p. 337).

Isopropyl alcohol is an irritant of the eyes and mucous membranes; at high concentrations, it causes narcosis in both humans and animals. The oral LD_{50} in rats is 5045 mg/kg, and the dermal LD_{50} in rabbits is 12,800 mg/kg (RTECS 1991). The lowest lethal concentration in rats is 12,000 ppm for 8 hours. Acutely poisoned animals show central nervous system effects before death (RTECS 1991). Mice exposed for 460 minutes to a 3250-ppm concentration of isopropyl alcohol developed ataxia, prostration, and narcosis (Proctor, Hughes, and Fischman 1988, p. 291). Conjunctivitis, iritis, and corneal opacity developed when the eyes of rabbits were instilled with a 70-percent isopropyl alcohol solution (Clayton and Clayton 1981, p. 4563). Applied once to the skin of rabbits, isopropyl alcohol caused a mild degree of irritation (RTECS 1991); however, prolonged contact of isopropyl alcohol with the skin of rabbits produced slight redness, dryness, and superficial desquamation (Clayton and Clayton 1982, p. 4565). Rats given 0.5 to 10 percent isopropyl alcohol in their drinking water for 27 weeks failed to gain weight at the normal rate but showed no gross or microscopic abnormalities at autopsy (Clayton and Clayton 1982, p. 4565). Inhalation of a 394-ppm concentration of isopropyl alcohol for 24 hours caused damage to the nasal mucosa of guinea pigs, although recovery occurred within 2 weeks if exposure was terminated; however, exposure to a concentration of 4550 ppm for the same period caused damage that took longer to recover (Ohashi, Nakai, Koshimo, Esaki, Ikeoka, Hariguchi, Teramoto, and Nakaseko 1988; Env. Res. 46(1):25–38).

Isopropyl alcohol has caused irritation of the eyes, nose, and throat in humans exposed for brief periods to a concentration of 400 ppm; at 800 ppm, these symptoms were more intense (Nelson, Enge, Ross, et al. 1943/Ex. 1–66). In addition, isopropyl alcohol has narcotic effects at higher concentrations. Prolonged or repeated contact of the skin with this solvent causes drying and dermatitis (HSDB 1990). At least one case of dermal sensitivity to isopropyl alcohol has been confirmed by patch test (HSDB 1990). An excess of paranasal sinus cancers, and perhaps of laryngeal cancers, was observed among workers manufacturing isopropyl alcohol (Weil, Smith, and Nale 1952/Ex. 1–453). However, it has been established that the cancers associated with

isopropyl alcohol manufacture were caused by the strong-acid manufacturing process and not by the isopropyl alcohol itself (Proctor, Hughes, and Fischman 1988, pp. 291–292; IARC 1987, Suppl. 7, p. 229).

Based on this evidence, OSHA preliminarily concludes that workers exposed to isopropyl alcohol are at significant risk of experiencing the narcotic and irritant effects associated with exposure to this substance. The Agency believes that the proposed limits, which were recently established for this substance in general industry, are necessary to substantially reduce these significant risks among workers in construction, maritime, and agriculture. OSHA considers the narcosis and eye and mucous membrane irritation associated with chronic and acute exposures to isopropyl alcohol material impairments of health within the meaning of the Act.

n-ISOPROPYLAMINE

CAS: 75–31–0; Chemical Formula: $(CH_3)_2CHNH_2$

H.S. No. 1228

OSHA's limit for n-isopropylamine in the construction and maritime industries is 5 ppm as an 8-hour TWA. The Agency has no PEL for this substance in agriculture. The ACGIH has a TLV®–TWA of 5 ppm and a TLV®–STEL of 10 ppm for n-isopropylamine; there is no NIOSH REL. OSHA is proposing to retain its 8-hour TWA limit of 5 ppm in construction and maritime, proposing to add a STEL of 10 ppm in these sectors, and proposing to extend both limits to agriculture. The Agency recently established these PELs in general industry, and NIOSH (Ex. 8–47) concurred that these limits were appropriate for n-isopropylamine.

n-Isopropylamine is a flammable, volatile, colorless liquid that has an odor similar to that of ammonia. This substance is used as a chemical intermediate in the synthesis of insecticides, pharmaceuticals, bactericides, dyes, rubber accelerators, and textile specialties. It is also used as a dehairing agent and solvent (HSDB 1986).

n-Isopropylamine causes eye, skin, mucous membrane, and respiratory tract irritation; at high concentrations, exposure may cause pulmonary edema. The oral LD_{50} in rats is 820 mg/kg, the LC_{50} in the same species is 4000 ppm for 4 hours, and the dermal LD_{50} in rabbits is 380 mg/kg (RTECS 1990). Applied to the skin of rabbits, isopropylamine caused severe irritation (RTECS 1990). Instilled into the eyes of rabbits, this substance caused injury rated 10 on an ascending severity scale of 1 to 10 (Grant 1986, p. 1041). The most serious effect of exposure to n-isopropylamine in laboratory animals is respiratory tract irritation, which can be severe enough to cause lung edema (Smyth 1956/Ex. 1–759).

Exposure to isopropylamine causes eye, nose, and upper respiratory tract irritation in humans. Volunteers exposed to isopropylamine concentrations of 10 to 20 ppm experienced nose and upper respiratory tract irritation after brief (not further specified) exposures (Clayton and Clayton 1981, pp. 3154–3155). Workers exposed for 8 hours to isopropylamine vapor at an unspecified concentration developed transient corneal edema, manifested as haloes around lights (Proctor, Hughes, and Fischman 1988, p. 292). Splashed into the eye, isopropylamine can cause burns and permanent visual impairment; skin contact may cause irritation, dermatitis, and burns (Proctor, Hughes, and Fischman 1988, p. 292).

Based on this evidence, the Agency preliminarily concludes that both a TWA and STEL are required to protect exposed workers in construction, maritime, and agriculture from the significant risk of eye, nose, and upper respiratory tract irritation that is known to be associated with exposure to isopropylamine. The Agency considers eye, nose, and upper respiratory tract irritation material impairments of health and preliminarily finds that adding a 10-ppm 15-minute STEL to the limit for isopropylamine is necessary to substantially reduce the risk posed to workers by exposure to this substance. Promulgation of these limits will also make OSHA's PELs for isopropylamine consistent across all regulated sectors.

ISOPROPYL ETHER

CAS: 108–20–3; Chemical Formula: $(CH_3)_2CHOCH(CH_3)_2$

H.S. No. 1226

OSHA's limit in construction and maritime for isopropyl ether is 500 ppm as an 8-hour TWA limit. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 250 ppm and a TLV®–STEL of 310 ppm for isopropyl ether. NIOSH has no REL but concurred (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 500 ppm for workplaces in agriculture. This is the limit recently established for this substance in general industry.

Isopropyl ether is a liquid that smells like ether. It is widely used as a solvent in mineral, vegetable, and animal oils and in waxes, paints, varnish removers, and rubber cements (HSDB 1986).

Isopropyl ether is an irritant of the eyes, nose, and upper respiratory tract both in humans and in animals. When applied to the skin of rabbits, a concentration of 20 g/kg caused death in 50 percent of the animals (RTECS 1991). The oral LD_{50} in rats is 8470 mg/kg, and the LC_{50} in the same species is 162 g/m³ (RTECS 1991). Instillation of the liquid into the eyes of rabbits caused minor injury, and repeated skin contact caused dermatitis in rabbits (Proctor, Hughes, and Fischman 1988, p. 293). Experimental animals surviving near-lethal exposures showed severe liver damage at autopsy (HSDB 1990). Animal studies have also shown that exposures to high concentrations of isopropyl ether cause narcosis and death (Machle, Scott, and Treon 1939/ Ex. 1–348). Experimental animals (a monkey, a rabbit, and a guinea pig) survived a one-hour exposure to a 30,000-ppm concentration, although they manifested signs of narcosis during the exposure (Proctor, Hughes, and Fischman 1988, p. 293). Twenty exposures to a 1-percent vapor concentration (i.e., to a 10,000-ppm concentration) produced systemic intoxication and central nervous system depression but no significant blood or organ weight changes (Machle, Scott, and Treon 1939/Ex. 1–348).

Human volunteers exposed for 15 minutes to isopropyl ether concentrations of 300 ppm experienced no overt irritation but complained about the objectionable odor of this substance; however, eye and nose irritation did occur during a 5-minute exposure to an 800-ppm concentration; some volunteers also experienced upper respiratory tract irritation at this concentration.

Based on this evidence, OSHA is proposing a 500 ppm 8-hour TWA limit for isopropyl ether in agricultural workplaces. The Agency preliminary concludes that this limit is necessary to substantially reduce a significant risk among agricultural workers of sensory irritation.

LITHIUM HYDRIDE

CAS: 7580–67–8; Chemical Formula: LiH

H.S. No. 2101

OSHA's PEL for lithium hydride in general industry, construction, and maritime is 0.025 mg/m³ as an 8-hour TWA; there is no PEL in agriculture. The ACGIH TLV®–TWA for lithium hydride is 0.025 mg/m³; there is no NIOSH REL. OSHA is proposing an 8-hour TWA PEL of 0.025 mg/m³ for lithium hydride in agriculture. This is the limit recently established for this substance in general industry.

Pure lithium hydride is an odorless, white, crystalline solid that is pyrophoric and darkens on exposure to light. The commercial product is a

bluish-gray solid. Lithium hydride is used as a reducing and condensing agent, a desiccant, and as an intermediate in chemical synthesis. It is also used in hydrogen generators, in nuclear shielding material, in the manufacture of electronic tubes and ceramics, and in space technology (HSDB 1985; ACGIH 1986; Gosselin, Smith, and Hodge 1984, p. II–105).

Lithium hydride is severely irritating and corrosive to the eyes, mucous membranes, skin, and respiratory tract of both animals and humans. The lowest lethal concentration in rats is 10 mg/m³ for 4 hours; acutely poisoned animals showed pulmonary edema at autopsy (RTECS 1991). Rabbits exposed to a 5-mg/m³ concentration showed signs of eye irritation (RTECS 1991). Single acute exposures of animals to lithium hydride concentrations ranging from 5 to 55 mg/m³ caused respiratory tract irritation in all species and at all concentrations (Browning 1969). Exposure to a 10-mg/m³ concentration caused corrosion of body fur and the skin of the legs and severe inflammation of the eyes and nasal septum (Browning 1969). Animals exposed 4 hours/day to a 5-mg/m³ concentration of lithium hydride for a period of 1 week exhibited coughing, sneezing, dyspnea, and signs of emphysema. However, no exposure-related lesions were noted in the lungs, liver, trachea, lymph nodes, or kidneys of these animals at autopsy (Browning 1969).

A technician working in the vicinity of a cylinder of lithium hydride that exploded had eye contact with some of the material and swallowed a small amount of the dust. The exposure resulted in burns that scarred both corneas and in strictures of the larynx, trachea, bronchi, and esophagus; death occurred 10 months later (Cracovaner 1964). A few milliequivalents of the lithium ion in the plasma can cause anorexia, nausea, tremor, muscle twitches, apathy, confusion, visual disturbances, coma, and death (Goodman and Gilman 1955). A lithium hydride concentration of 0.05 mg/m³ is objectionable to some exposed individuals, and exposure to a 0.1-mg/m³ concentration causes eye and nose irritation (AIHA 1964).

Based on this evidence in humans and animals, OSHA preliminarily concludes that agricultural workers exposed to this substance at the levels permitted by the absence of a permissible exposure limit are at significant risk of experiencing severe irritation of the eyes, skin, mucous membranes, and respiratory tract. The Agency believes that establishing a PEL of 0.025 mg/m³ as an 8-hour TWA will protect workers in agriculture from these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

MALEIC ANHYDRIDE

CAS: 108–31–6; Chemical Formula: $C_4H_2O_3$

H.S. No. 2102

In general industry, construction, and maritime, OSHA's permissible exposure limit for maleic anhydride is 0.25 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV® of 0.25 ppm as an 8-hour TWA for this substance; NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL of 0.25 ppm for maleic anhydride in agriculture. This is the limit recently established for this substance in general industry.

Maleic anhydride is a solid at room temperature and is colorless and crystalline in form. It is commercially available as briquettes, white lumps, tablets, and pellets. Maleic anhydride is used as a chemical intermediate, an ingredient in pesticides, a preservative for fats and oils, in paper sizing, in permanent press fabrics, in the manufacture of resins, and as a raw material for organic synthesis (ACGIH 1986, p. 359; AIHA 1978; HSDB 1987). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Maleic anhydride is both an eye, skin, and upper respiratory tract irritant and a skin and pulmonary sensitizer. The oral LD_{50} in rats is 400 mg/kg, and the dermal LD_{50} in rabbits is 2620 mg/kg (RTECS 1990). In contact with the skin or eyes, maleic anhydride causes severe irritation and caustic burns (Grant 1986, p. 574). Rats injected subcutaneously with 1 mg maleic anhydride twice/week for 61 weeks developed fibrosarcomas (Dickens and Jones 1963).

Because maleic anhydride hydrolyzes in contact with moisture to maleic acid, contact of the eyes, mucous membranes, or skin causes severe irritation and burns. Even at a concentration of 0.25 ppm, humans report experiencing irritation (AIHA 1969), but exposure to 0.22 ppm causes no irritation (Grigoreva 1966). Workers exposed to unspecified concentrations of the dust or vapors have developed punctate epithelial keratitis, but the eyes of these individuals have returned to normal within a few days of exposure (Ghezzi and Scotti 1965; Heersink and Dunne 1976; Kowalski and Slusarczyk-Zalobna et al. 1968). Workers exposed to unspecified concentrations of maleic anhydride under conditions described as "uncontrolled" showed acute systemic effects that were manifested as respiratory, nervous, and cardiovascular system disorders (Titiova and Zakivova 1977). Chronic exposure to maleic anhydride may cause chronic conjunctivitis, keratitis, and bronchitis (AIHA 1969; Tanaka 1976). Repeated exposure to a 1.25 to 2.5 ppm concentration of this substance produced ulceration of the nasal septum and, in some workers, asthma (AIHA 1969). Repeated contact of the skin causes allergic dermatitis (AIHA 1969).

Based on this evidence in humans and animals, OSHA is proposing an 8-hour TWA PEL of 0.25 ppm for maleic anhydride in agriculture; adoption of the proposed level would establish the same PEL for workplaces in all OSHA-regulated industry sectors. The Agency preliminarily concludes that occupational exposure to maleic anhydride causes irritation of the eyes, skin, and upper respiratory tract and pulmonary and skin sensitization. Accordingly, OSHA believes that, in the absence of a permissible exposure limit, workers in agriculture are potentially at significant risk for these exposure-related effects and that the proposed PEL will substantially reduce these risks.

MESITYL OXIDE

CAS: 141–79–7; Chemical Formula: $(CH_3)_2C{=}CHCOCH_3$

H.S. No. 1243

OSHA's limit for mesityl oxide in the construction and maritime industries is 25 ppm as an 8-hour TWA. The Agency has no PEL for this substance in agriculture. The ACGIH has a TLV®–TWA of 15 ppm and a TLV®–STEL of 25 ppm for mesityl oxide; the NIOSH REL is 10 ppm as a 10-hour TWA. OSHA is proposing a 15 ppm 8-hour TWA and a 25 ppm STEL for mesityl oxide in the construction, agriculture, and maritime industries. These are the limits recently established for mesityl oxide in general industry.

Mesityl oxide is an oily, colorless liquid with an odor similar to that of peppermint. It is used as an insect repellant, a paint remover, a solvent, and in ore flotation (ACGIH 1986, p. 361).

Mesityl oxide is an irritant of the eyes and mucous membranes in both animals and humans; at high concentrations, it also causes central nervous system depression. The oral LD_{50} in rats is 1120 mg/kg, the LC_{50} in the same species is 9 g/m³ for 4 hours, and the dermal LD_{50} in rabbits is 5150 mg/kg (RTECS 1990). In contact with the eyes of rabbits, mesityl

oxide caused moderate to severe irritation; in contact with the skin of rabbits, this substance caused mild irritation (RTECS 1990). All guinea pigs exposed to a 2000-ppm concentration of mesityl oxide for up to 422 minutes died. The exposed animals exhibited eye and respiratory tract irritation, gradual loss of corneal and auditory reflexes, and coma before death (Specht, Miller, Valaer et al. 1940). Slight liver, kidney, and lung injury and irritation of the eyes and nose occurred in animals of two species exposed to a 500-ppm concentration for 8 hours a day for 10 days (Specht, Miller, Valaer et al. 1940). Experimental animals exposed to the vapor of mesityl oxide have shown signs of progressive central nervous system depression (Gosselin, Smith, and Hodge 1984, p. II–185). Guinea pigs exposed to mesityl oxide concentrations of 2300, 5000, or 10,000 ppm for as long as 8 hours showed dose-dependent central nervous system effects, as well as signs of respiratory irritation, reduced body temperature, reductions in heart rate and respiratory rate, and loss of reflexes (Clayton and Clayton 1982, p. 4753). Rabbits exposed to a 25-ppm concentration 4 hours/day for 189 days developed anemia and leukopenia (Clayton and Clayton 1982, p. 4753). Smyth, Seaton, and Fischer (1942/Ex. 1–378) reported liver and kidney damage among rats and guinea pigs continuously exposed to a 100-ppm concentration of mesityl oxide for 6 weeks; no adverse effects were reported in animals exposed to 50 ppm.

Silverman, Schulte, and First (1946/Ex. 1–142) found that a majority of test subjects experienced eye irritation on exposure to a 25-ppm concentration of mesityl oxide for 15 minutes and nasal irritation on exposure to 50 ppm. Prolonged or repeated skin contact with this substance causes dermatitis (Proctor, Hughes, and Fischman 1988, p. 312). Exposure to mesityl oxide at high (not further specified) concentrations causes signs of central nervous system depression, such as headache, dizziness, tremors, incoordination, weakness, and decreases in heart and respiratory rate (HSDB 1988).

After reviewing the health evidence for this substance, OSHA preliminarily finds that the proposed 15-ppm TWA and 25-ppm STEL limits are necessary to protect workers in construction, maritime, and agriculture against both the acute and chronic effects associated with exposure to this substance. These effects include possible liver and kidney damage as well as central nervous system depression and eye, mucous membrane, and skin irritation. The Agency considers these exposure-related effects material impairments of health and functional capacity. To reduce these significant occupational risks, OSHA is therefore proposing limits for mesityl oxide of 15 ppm as an 8-hour TWA and 25 ppm as a 15-minute STEL. Promulgation of these limits for workplaces in the construction, maritime, and agriculture sectors will make OSHA's PELs for this substance consistent across all OSHA-regulated sectors.

METHYL ACRYLATE

CAS: 96–33–3; Chemical Formula:

$CH_2=CHCOOCH_3$

H.S. No. 2105

OSHA's PEL for methyl acrylate in general industry, construction, and maritime is 10 ppm as an 8-hour TWA, with a skin notation; there is no PEL in agriculture. The ACGIH has a TLV®–TWA for methyl acrylate of 10 ppm, with a skin notation; there is no NIOSH REL for this substance. OSHA is proposing an 8-hour TWA PEL of 10 ppm, with a skin notation, for methyl acrylate in agriculture. This is the limit recently established for this substance in general industry.

Methyl acrylate is a clear, colorless liquid with an acrid odor (HSDB 1986; ACGIH 1986, p. 369). It is used primarily to produce acrylic and modacrylic fibers. In the dental, medical, and pharmaceutical industries, it is used as a monomer, polymer, and copolymer (IARC 1979, p. 55; Proctor, Hughes, and Fischman 1988, p. 318). Methyl acrylate is also used in paints, coatings, adhesives, plastic panels, and textile finishes (Gosselin, Smith, and Hodge 1984, p. II–409). This substance also finds use as an aid in the timed release and disintegration of pesticides (Clayton and Clayton 1982, p. 2293). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Methyl acrylate causes irritation of the eyes, mucous membranes, respiratory tract, and skin; it has also been shown to cause dermal sensitization and systemic toxicity in both humans and laboratory animals. The oral LD_{50} in rats is 277 mg/kg, and the dermal LD_{50} in rabbits is 1243 mg/kg (RTECS 1991). By inhalation, methyl acrylate causes death in 50 percent of rats exposed to a concentration of 1350 ppm for 4 hours (RTECS 1991). In rabbits, dyspnea, cyanosis, convulsions, and death resulted from a single oral dose of 280 mg/kg (Treon et al. 1949). Rabbits exposed to a 237-ppm concentration of methyl acrylate for 7 hours/day for 11 days exhibited conjunctival and nasal irritation, lacrimation, and labored breathing (Treon et al. 1949). Methyl acrylate causes contact sensitivity in guinea pigs, and contact of this substance with the skin of rabbits causes marked irritation (Treon 1949; Parker and Turk 1983). Methyl acrylate caused moderate irritation when tested on rabbit eyes (RTECS 1991; Grant 1986, p. 1044). In contact with the eye, methyl acrylate causes severe burns of the cornea (HSDB 1986). In laboratory animals, repeated dermal application of small amounts of methyl acrylate caused systemic poisoning in the form of lung, liver, and kidney damage (Gosselin, Smith, and Hodge 1984, p. II–409).

In humans, the lowest concentration of methyl acrylate reported to have irritant effects is 75 ppm (Sandmeyer and Kirwin 1981). Rapid breathing, headache, nausea, lethargy, pulmonary edema, convulsions, and death may result from the inhalation of high (not further specified) concentrations of methyl acrylate (Gosselin, Smith, and Hodge 1984, p. II–409). A fatality resulted from the subcutaneous injection of a 1000 mg/kg dose of methyl acrylate (Clayton and Clayton 1982, p. 2294). An industry study involving 105 workers exposed to methyl acrylate showed positive sensitization reactions in 76.1 percent of the workers; 53.7 percent of these workers showed clinical evidence of having three allergies (dermatitis, eczema, and urticaria) (Khromov 1974). In contact with the skin, 20 parts methyl acrylate per hundred parts of olive oil causes irritation in one-third of individuals and allergic responses in approximately one out of 10 persons (Cavelier et al. 1981).

Based on this evidence in humans and animals, OSHA preliminarily concludes that agricultural workers exposed to this substance at the levels permitted by the absence of a permissible exposure limit are at significant risk of experiencing irritation of the eyes, skin, mucous membranes, and respiratory tract. The Agency believes that establishing a TWA PEL of 10 ppm, with a skin notation, is necessary to protect workers in agriculture from these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

METHYL 2–CYANOACRYLATE

CAS: 137–05–3; Chemical Formula:

$CH_2=C(C=N)COOCH_3$

H.S. No. 1248

OSHA has no limit for methyl 2-cyanoacrylate in the construction,

agriculture, or maritime industries. The ACGIH has a TLV®–TWA of 2 ppm and a TLV®–STEL of 4 ppm for this substance; there is no NIOSH REL. The Agency is proposing a 2-ppm 8-hour TWA and a 4-ppm STEL for methyl 2-cyanoacrylate in the construction, agriculture, and maritime industries. OSHA recently established these PELs in general industry, and NIOSH (Ex. 8–47) concurred that these limits were appropriate for this substance.

Methyl 2-cyanoacrylate is a colorless, viscous liquid that is used to manufacture adhesives, super-glues, and polymers (ACGIH 1986, p. 383; Gosselin, Smith, and Hodge 1984, p. II–409).

Methyl 2-cyanoacrylate is an irritant of the eyes, nose, and upper respiratory tract. The oral LD_{50} in rats is 1.6 to 3.2 g/kg, and the dermal LD_{50} in guinea pigs is greater than 10 ml/kg (ACGIH 1986, p. 383). The LC_{50} in rats is 101 ppm for 6 hours (ACGIH 1986, p. 383). The adverse effects reported in laboratory animals are slight irritation of the skin and corneal damage. Repeated exposures (6 hours/day for 5 days/week) to a concentration of 31.3 ppm for a total of 12 exposures caused only a slight decrease in the rate of weight gain in rats and no nasal or tracheal lesions or systemic toxicity (ACGIH 1986, p. 383). No changes were observed in rats similarly exposed to a concentration of 3.1 ppm (Eastman Kodak 1985, as cited in ACGIH 1986/Ex. 1–3, p. 383). Administered intratesticularly to monkeys at a dose of 100 mg/kg, methyl 2-cyanoacrylate caused reproductive effects (RTECS 1991).

In a simulated workbench exposure, McGee and co-workers reported nasal irritation in humans when the concentration of methyl 2-cyanoacrylate reached 3 ppm and eye irritation at a concentration of 5 ppm (McGee, Oglesby, Raleigh, and Fassett 1968/Ex. 1–424). In contact with the eyes, this substance causes corneal inflammation; however, recovery is usually complete (Grant 1986, p. 291). In contact with the skin of humans, it causes mild irritation (HSDB 1991).

Based on this evidence in humans and animals, OSHA preliminarily concludes that employees in construction, maritime, and agriculture are at risk of experiencing nasal irritation on exposure to methyl 2-cyanoacrylate concentrations of 3 ppm or above and of eye irritation at concentrations of 5 ppm or above. The Agency believes that these adverse effects are material impairments of health and that the proposed PELs are necessary to substantially reduce these significant risks. Accordingly, OSHA is proposing a 2-ppm 8-hour TWA limit and a 4-ppm STEL for methyl 2-cyanoacrylate in the construction, agriculture, and maritime industries. Promulgation of these limits will make the PELs for methyl 2-cyanoacrylate consistent across all OSHA-regulated sectors.

METHYL ISOBUTYL CARBINOL

CAS: 108–11–2; Chemical Formula: $CH_3CHOHCH_2CH(CH_3)_2$

H.S. No. 1261

OSHA has an 8-hour TWA limit of 25 ppm, with a skin notation, for methyl isobutyl carbinol in the construction and maritime industries. The ACGIH has an 8-hour TLV®–TWA of 25 ppm, a TLV®–STEL of 40 ppm, and a skin notation for methyl isobutyl carbinol. NIOSH has no REL for this substance. OSHA is retaining the 8-hour TWA PEL of 25 ppm in construction and maritime, proposing to add a 15-minute STEL of 40 ppm for methyl isobutyl carbinol in these two sections, and additionally proposing both limits for workplaces in agriculture. NIOSH (Ex. 8–47) concurred that these limits, which were recently established for methyl isobutyl carbinol in general industry, are appropriate for this substance.

Methyl isobutyl carbinol is a colorless, stable liquid. It is used as a solvent for dyestuffs, oil, gums, resins, waxes, and cellulose esters. Methyl isobutyl carbinol is also used in flotation processes and brake fluids and as an intermediate in chemical synthesis (ACGIH 1986, p. 401; Clayton and Clayton 1982, p. 4613).

In addition to sensory irritation, methyl isobutyl carbinol causes central nervous system depression in humans and experimental animals. The oral LD_{50} in rats is 2590 mg/kg, the lowest lethal concentration in the same species is 2000 ppm for 4 hours, and the dermal LD_{50} in rabbits is 3560 mg/kg (RTECS 1990). Anesthesia was observed in two of five mice given 1.0 ml/kg of methyl isobutyl carbinol orally; all exposed mice became anesthetized when the dose was increased to 1.5 or 2.0 ml/kg (McOmie and Anderson 1949). Gastrointestinal irritation and mesenteric congestion were also observed in these animals at autopsy (McOmie and Anderson 1949). Applied to the skin of rabbits, a dose of 10 mg methyl isobutyl carbinol produced mild irritation; instilled into rabbit eyes, methyl isobutyl carbinol caused severe irritation (RTECS 1990).

At a concentration of 50 ppm for 15 minutes, human volunteers experienced eye irritation (Silverman et al. 1946). At concentrations above 50 ppm, nose and upper respiratory tract irritation occurred (Silverman et al. 1946). It is expected that narcosis would also be experienced by humans exposed to high concentrations of methyl isobutyl carbinol.

In view of the finding that exposure to a 50-ppm concentration of methyl isobutyl carbinol can result in eye irritation in humans in as little as 15 minutes, OSHA has preliminarily concluded that a limit on both full-shift and short-term exposure is necessary. The Agency considers the irritant and narcotic effects associated with exposure to this substance to be material impairments of health. To reduce these significant risks, OSHA is proposing a 25-ppm 8-hour TWA, a 15-minute STEL of 40 ppm, and a skin notation for this substance in the construction, maritime, and agricultural industries. Promulgation of these limits will make the PELs for methyl isobutyl carbinol consistent across all OSHA-regulated sectors.

METHYL ISOCYANATE

CAS: 624–83–9; Chemical Formula: CH_3NCO

H.S. No. 2106

OSHA's PEL for methyl isocyanate in general industry, construction, and maritime is 0.02 ppm as an 8-hour TWA, with a skin notation; there is no PEL in agriculture. The ACGIH TLV®–TWA for methyl isocyanate is 0.02 ppm as an 8-hour TWA, with a skin notation; there is no NIOSH REL for this substance. OSHA is proposing to extend the 8-hour TWA PEL of 0.02 ppm, and the skin notation, to agriculture. Promulgation of this PEL will make OSHA's limit for this substance consistent across all regulated sectors.

Methyl isocyanate is the chemical responsible for the disaster in Bhopal, India, in 1984 that killed 2,000 people and affected as many as 100,000 individuals. This substance is used in the manufacture of carbaryl insecticides and in the production of polyurethane plastics and foams (ACGIH 1986, p. 403).

Methyl isocyanate is a severe irritant of the eyes, mucous membranes, and skin and a skin and pulmonary sensitizer in both humans and animals. The oral LD_{50} in rats is 69 mg/kg, and the LC_{50} in the same species is 6.1 ppm for 6 hours (RTECS 1990). The dermal LD_{50} in rabbits is 213 mg/kg (RTECS 1990). Acutely poisoned animals exhibit signs of severe eye and pulmonary irritation before death (ACGIH 1986, p. 403). Application of this substance to the skin of rabbits, followed by occlusion, resulted in bleeding and marked edema of the skin (ACGIH 1986, p. 403). All 16 guinea pigs that were challenged with 0.01 ml of a 0.01 percent solution of methyl isocyanate developed skin

sensitization (ACGIH 1986, p. 403). On a 1 to 10 scale of increasing severity, methyl isocyanate caused corneal damage to rabbit eyes that was rated 10 (Smyth and Carpenter 1969). Rats exposed to a 0.21 ppm concentration of methyl isocyanate for 30 minutes on each of 6 days were followed for 90 days after exposure; although all of the rats survived, their lungs showed damage at autopsy (Sethi, Dayal, and Singh 1989). When administered to rats by inhalation or by intravenous or subcutaneous administration, methyl isocyanate caused a dose-related depression in cardiovascular activity (Kumar, Sachan, Pant, et al. 1989). After a single 2-hour exposure to methyl isocyanate at a concentration of 1, 3, or 10 ppm, male and female rats in the 10-ppm group had a 42 and 36 percent incidence, respectively, of fibrosis of the secondary bronchi (Bucher and Uraih 1989). In all dose groups, male rats (but not females or mice of either sex) showed slight increases in the incidence of pheochromocytomas of the adrenal medulla and adenomas of the pancreas (Bucher and Uraih 1989).

Human volunteers exposed to methyl isocyanate at a concentration of 0.04 ppm for 1 to 5 minutes reported experiencing no effects, but exposure to a 2-ppm concentration caused tearing, and increasing the concentration to 21 ppm caused "unbearable" irritation (Kimmerle and Eben 1964). In another experiment, however, a 10-minute exposure to a 0.05-ppm concentration caused eye irritation in all exposed volunteers (ACGIH 1986, p. 403). Some individuals exposed to methyl isocyanate develop pulmonary sensitization (Proctor, Hughes, and Fischman 1988, p. 337). Exposure causes the following signs and symptoms of severe respiratory irritation: difficult breathing, chest pain, coughing, and increased bronchial secretion (Rye 1973). The victims of the Bhopal disaster died of pulmonary edema, which was often accompanied by destruction of the alveolar walls (Marwick 1985). The victims also experienced severe visual impairment and showed clinical signs of damage to the liver and kidneys (Marwick 1985).

Based on this evidence in humans and animals, OSHA preliminarily concludes that agricultural workers exposed to methyl isocyanate at the concentrations permitted in the absence of a limit are at significant risk of experiencing severe eye and lung irritation as well as pulmonary sensitization. The Agency believes that establishing a PEL of 0.02 ppm as an 8-hour TWA will protect workers in agriculture from these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

METHYL MERCAPTAN

CAS: 74–93–1; Chemical Formula: CH_3SH

H.S. No. 1263

OSHA has a ceiling limit of 0.5 ppm for methyl mercaptan in the construction and maritime industries. There is no PEL for agriculture. The ACGIH has a TLV®–TWA of 0.5 ppm for methyl mercaptan. NIOSH has a 15-minute ceiling limit of 0.5 ppm for this substance. OSHA is proposing an 8-hour TWA of 0.5 ppm for methyl mercaptan in the construction, maritime, and agriculture industries. This is the limit recently established for this substance in general industry.

Methyl mercaptan is a flammable, water-soluble gas with a disagreeable odor like that of rotten cabbage. Methyl mercaptan is used in the production of pharmaceuticals, pesticides, fumigants, fungicides, jet fuel, dyes, and plastics. It is also used to give odor to natural gas and to synthesize methionine (ACGIH 1986, p. 405; Clayton and Clayton 1981, p. 2066).

In addition to sensory irritation, methyl mercaptan causes narcosis and pulmonary edema in humans and experimental animals. In rats, the LC_{50} is 675 ppm for 4 hours (RTECS 1990). In one study, animals exposed to methyl mercaptan experienced restlessness, muscular weakness, paralysis, convulsions, respiratory depression, and cyanosis (Shults, Fountain, and Lynch 1970). Male rats exposed to concentrations of methyl mercaptan ranging from 1600 to 2200 ppm for an unspecified period lost their righting reflexes, but female rats exposed to 500 ppm for 30 to 35 minutes showed no adverse effects (Clayton and Clayton 1981, p. 2068). Rats exposed continuously for 90 days to a 50-ppm concentration of methyl mercaptan showed hematologic and lung effects, and mice exposed on the same regimen showed both hematologic and hepatic effects (Clayton and Clayton 1981, p. 2068).

A worker found comatose after emptying containers of methyl mercaptan developed elevated blood pressure, wheezing, tachycardia, rigidity of the extremities, and deep coma; death was caused by a pulmonary embolus that developed 28 days after exposure. Before death, this worker, who had a glucose-6-phosphate dehydrogenase deficiency, developed methemoglobinemia and severe hemolytic anemia (Proctor, Hughes, and Fischman 1988, p. 338). In a nonfatal incident involving methyl mercaptan exposure, the exposed worker became cyanotic, experienced convulsions, and subsequently developed a lung abscess (Shults, Fountain, and Lynch 1970). A 1918 report by Pickler (Clayton and Clayton 1981, p. 2068) describes the accidental exposure (for several hours) of 28 students to a concentration of methyl mercaptan estimated to be 4 ppm. These individuals experienced headaches and nausea that lasted for 24 hours, and one student showed signs of liver involvement, demonstrated by the appearance of epithelial cells, protein, and red blood cells in the urine; these effects subsided in 6 weeks (Clayton and Clayton 1981, p. 2068). In another incident involving accidental overexposure to an unknown concentration of methyl mercaptan, the worker reported signs of mucosal and respiratory tract irritation and developed bronchopneumonia 20 hours later; this patient later recovered fully (Clayton and Clayton 1981, p. 2068). Long-term exposure to low concentrations (not further specified) of methyl mercaptan caused dermatitis in exposed individuals (Clayton and Clayton 1981, p. 2068).

Based on this evidence in humans and animals, OSHA preliminarily concludes that methyl mercaptan poses significant risks of mucous membrane and respiratory tract irritation, dermatitis, central nervous system effects, and possible liver damage to workers in the construction, maritime, and agriculture industries. The Agency believes that the proposed 8-hour TWA PEL of 0.5 ppm is necessary to substantially reduce the risk of these exposure-related effects, which constitute material impairments of health. Promulgation of this limit will also make OSHA's PEL for methyl mercaptan consistent across all OSHA-regulated sectors.

METHYL METHACRYLATE

CAS: 80–62–6; Chemical Formula: $CH_2=C(CH_3)COOCH_3$

H.S. No. 2107

OSHA's PEL for methyl methacrylate in general industry, construction, and maritime is 700 ppm as an 8-hour TWA; there is no PEL in agriculture. The ACGIH TLV®–TWA for this substance is 100 ppm as an 8-hour TWA; there is no NIOSH REL. OSHA is proposing an 8-hour TWA of 100 ppm for methyl methacrylate in agriculture. This is the limit recently established for this substance in general industry.

Methyl methacrylate is a colorless liquid that is used captively in the production of acrylic polymers. This substance also finds use in the

manufacture of surface coating resins (latexes, lacquers, and enamels). Latex house paints, interior gloss and semi-gloss paints, and interior flat wall and ceiling paints account for the major portion of methyl methacrylate used in this country (IARC 1979, p. 189). This substance is also an ingredient in many consumer goods, including floor polishes, adhesives, binders, paper coatings, sealants, leather base coatings and finishes, and many other applications (IARC 1979, p. 189).

Methyl methacrylate is an eye, skin, and upper respiratory tract irritant, a central nervous system depressant and neurotoxin, and a skin sensitizer. In animals, it is also embryotoxic and produces developmental effects. The oral LD_{50} in rats is 7872 mg/kg; in mice, the LC_{50} is 18,500 mg/m³ (4500 ppm) for 2 hours (RTECS 1990). Dogs exposed to lethal concentrations (11,000 ppm) of methyl methacrylate showed signs of central nervous system depression and a drop in blood pressure before dying of respiratory arrest; autopsy revealed damage to the liver and kidneys (Speakman et al. 1945). Male rats exposed over a 5-month period to a methyl methacrylate concentration of 116 ppm for 7 hours/day, 5 days/week showed, at autopsy, tracheal mucosa that were denuded of cilia (Tansy et al. 1980). Pregnant rats exposed on gestation days 6 through 15 to a 27,500-ppm concentration of methyl methacrylate for less than 1 hour displayed signs of maternal toxicity, and their offspring had an increased incidence of fetal death, hematomas, and skeletal deformities (Nicholas et al. 1979). In another teratogenicity study, methyl methacrylate caused embryotoxic and developmental effects in the offspring of rats given intraperitoneal injections during pregnancy (RTECS 1990). A 2-year cancer bioassay conducted by the National Toxicology Program reported no evidence of carcinogenicity in male and female rats and mice exposed to methyl methacrylate concentrations ranging from 250 to 1000 ppm (NTP 1986).

Workers exposed to airborne concentrations of methyl methacrylate ranging from 170 to 250 ppm complained of irritation, and some reports place the human irritation level at 125 ppm (ACGIH 1986, p. 406; Seravotsnoe 1976; RTECS 1990). Dental technicians who regularly handle methyl methacrylate cement have developed paresthesias of the fingers, indicating axonal degeneration in those areas of the fingers in contact with the cement (Seppalainen and Rajaniemi 1984). In one group of 200 workers exposed to the vapor at a concentration of 2 to 200 mg/m³ (0.5 to 50 ppm), 119 individuals reported headaches and several others complained of excessive fatigue, sleep disturbances, loss of memory, and irritability (IARC 1979, p. 194). There are reports of skin sensitization in humans 10 days after skin application, and several volunteers reacted when challenged with liquid methyl methacrylate (Lefaux 1968).

Based on this evidence in humans and animals, OSHA preliminarily concludes that agricultural workers exposed to this substance at the levels permitted by the absence of a limit are at significant risk of experiencing sensory irritation, axonal neuropathies, and skin sensitization. OSHA considers these effects material impairments of health. The Agency believes that establishing a PEL of 100 ppm as an 8-hour TWA will substantially reduce these risks for workers in agriculture. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

METHYL n-AMYL KETONE

CAS: 110-43-0; Chemical Formula: $CH_3COC_5H_{11}$

H.S. No. 1264

The OSHA limit for methyl n-amyl ketone in the construction and maritime industries is 100 ppm as an 8-hour TWA. There is no PEL for this substance in agriculture. The ACGIH has a TLV®-TWA of 50 ppm, and NIOSH has a REL for this substance of 100 ppm as a 10-hour TWA. OSHA is retaining its current limit of 100 ppm as a TWA for methyl n-amyl ketone in the construction and maritime sectors and is proposing the same limit for agriculture. NIOSH (Ex. 150) agreed that a 100-ppm PEL for methyl n-amyl ketone was appropriate when the Agency recently established this limit in general industry. Extending this PEL to agriculture will thus make OSHA's limit for this substance consistent across all OSHA-regulated sectors.

Methyl n-amyl ketone, also called 2-heptanone, is a water-white liquid with a fruity odor. It is used as a solvent for synthetic resin finishes and nitrocellulose lacquers, as a flavoring agent, and in perfumes (ACGIH 1986, p. 374; Hawley's 1987, p. 758).

In addition to sensory irritation, methyl n-amyl ketone causes narcosis in experimental animals. The oral LD_{50}s in rats and mice are 1670 mg/kg and 730 mg/kg, respectively (RTECS 1990). The lowest lethal concentration in rats is 4000 ppm for 4 hours (RTECS 1990). The dermal LD_{50} in rabbits is 12,600 mg/kg (RTECS 1990). Exposing guinea pigs to 4800 ppm of methyl n-amyl ketone caused narcosis and death in 4 to 8 hours; exposure to a concentration of 2000 was strongly narcotic (Specht et al. 1940). In contact with the skin of rabbits, methyl n-amyl ketone caused mild irritation (RTECS 1990). Instilled into rabbit eyes, this substance caused injury rated 2 on an ascending severity scale of 1 to 10 (HSDB 1985). Johnson et al. (1978/Ex. 1-335) found no neurologic impairment in rats and monkeys exposed to 131 ppm or 1025 ppm methyl n-amyl ketone 6 hours/day, 5 days/week for 9 months, and no gross or histopathologic changes were found in these animals at autopsy (Johnson, Setzer, Lewis, and Hornung 1978/Ex. 1-335).

There are no case reports of industrial poisonings in exposed workers, and the concentration at which irritation or narcosis would begin in humans is not known. Methyl n-amyl ketone has been tested in volunteers for its potential to cause skin sensitization, and the results of these tests have been negative (Clayton and Clayton 1981, p. 4758).

Based on this evidence, OSHA is retaining its 8-hour TWA PEL of 100 ppm for methyl n-amyl ketone in the construction and maritime industries and is proposing to extend this limit to workplaces in agriculture. The Agency preliminarily finds that, based on effects seen in animals, exposure to this substance is likely to be associated with both irritant and narcotic effects and that these effects constitute material health impairments. To reduce these significant occupational risks, OSHA is therefore proposing an 8-hour TWA PEL of 100 ppm in agriculture for methyl n-amyl ketone. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

alpha-METHYL STYRENE

CAS: 98-83-9; Chemical Formula: $C_6H_5C(CH_3)=CH_2$

H.S. No. 1267

OSHA has a ceiling limit of 100 ppm for alpha-methyl styrene in the construction and maritime industries. There is no limit in agriculture. The ACGIH has a 50 ppm TLV®-TWA and a 100 ppm TLV®-STEL for alpha-methyl styrene. NIOSH has no REL for this substance. The Agency is proposing a 50-ppm 8-hour TWA and a 10-ppm 15-minute STEL for alpha-methyl styrene in the agriculture, construction, and maritime industries. NIOSH (Ex. 8-47) concurred with these limits when the Agency recently established them for alpha-methyl styrene in general industry.

alpha-Methyl styrene is a combustible, colorless liquid that has a sharp, aromatic odor and is used as a polymerization monomer. This substance also finds use as a chemical intermediate (Clayton and Clayton 1981, p. 3324).

In addition to sensory irritation, alpha-methyl styrene causes central nervous system depression in humans and experimental animals. The oral LD_{50} in rats is 4900 mg/kg, and the lowest lethal concentration in the same species is 3000 ppm for an unspecified period of time (RTECS 1990). Applied to the eyes of rabbits, a dose of 91 mg caused a mild degree of irritation; in contact with the skin, alpha-methyl styrene caused moderate irritation (RTECS 1990). Twenty applications of this substance to rabbit skin, however, caused inflammation, hyperemia, edema, and hyperkeratosis (Clayton and Clayton 1981, p. 3324). Guinea pigs and rats exposed to 3000 ppm 7 hours/day for 3 to 4 days died. At autopsy, slight liver and kidney changes were observed in guinea pigs after exposure to 800 ppm for 27 days (Wolf et al. 1956/Ex. 1–404). Rats, guinea pigs, rabbits, mice, and monkeys exposed 7 hours/day, 5 days/week for 6 months to a 200-ppm concentration of alpha-methyl styrene showed no adverse effects either on gross or microscopic examination (Wolf, Rowe, McCollister et al. 1956/Ex. 1–404). A 1981 NIOSH teratogenicity study in pregnant rats injected intraperitoneally with alpha-methyl styrene on days 1 through 15 of gestation showed that, at a dose of 250 mg/kg, alpha-methyl styrene caused a significantly increased number of resorptions (Hardin, Bond, Sikov, Andres, Beliles, and Niemier 1981; Scand. J. Work Environ. Health 7(4):66–75).

In humans, a 2-minute exposure to a 200-ppm concentration of alpha-methyl styrene caused eye irritation and complaints about this substance's unpleasant odor (Wolf et al. 1956/Ex. 1–404). A Russian study in workers exposed for 4 to 5 years to alpha-methyl styrene at concentrations of 3 to 7 mg/m³ (0.6 to 1.5 ppm) reports that these workers showed signs of occupational poisoning, including asthenia, vasovegatative disorders, reduced arterial pressure, and hematopoietic changes (temporary leukemia, thrombocytopenia, and reticulocytopenia) (Kapkaev and Suhanova 1986). Another Russian study reports a high incidence of sensitization dermatitis in workers in a synthetic rubber plant who were dermally exposed to alpha-methyl styrene and other substances during rubber manufacture (Mirzoyan 1972).

To ensure that workers in construction, maritime, and agriculture are protected against the irritant, sensitization, and narcotic effects of exposure to this substance, OSHA is proposing a 50-ppm 8-hour TWA limit and a 100-ppm 15-minute STEL for alpha-methyl styrene in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that these combined limits are necessary to substantially reduce the risk that workers in these sectors will experience these exposure-related effects, which constitute material impairments of health. Promulgation of these limits will also make OSHA's PELs for this substance consistent across all OSHA-regulated industries.

o-METHYLCYCLOHEXANONE

CAS: 583–60–8; Chemical Formula: $CH_3C_6H_9CO$

H.S. No. 1270

OSHA's limit for o-methylcyclohexanone in construction and maritime is 100 ppm as an 8-hour TWA, with a skin notation. There is no limit in agriculture. The ACGIH has a 50-ppm 8-hour TLV®–TWA, a 75-ppm 15-minute TLV®–STEL, and a skin notation for o-methylcyclohexanone. NIOSH has no REL for this substance. The Agency is proposing PELs of 50 ppm as an 8-hour TWA and 75 ppm as a 15-minute STEL, and a skin notation, for o-methylcyclohexanone in construction, maritime, and agriculture. NIOSH (Ex. 8–47) concurred that these limits were appropriate for o-methylcyclohexanone when the Agency recently established them in general industry.

ortho-Methylcyclohexanone is a somewhat viscous liquid with an acetone-like odor. It is a solvent that is used in the manufacture of lacquers, varnishes, and plastics. ortho-Methylcyclohexanone is also used in the leather industry and as a rust remover (ACGIH 1986, p. 386). The commercial product contains a mixture of isomers; however, toxicity data describe the effects of the ortho isomer only.

In addition to sensory irritation, o-methylcyclohexanone causes narcosis in experimental animals and may also do so in humans. The oral LD_{50} in rats is 2140 mg/kg, and the dermal LD_{50} in rabbits is 1635 mg/kg (RTECS 1991). The LC_{50} in rats is 2890 ppm for 4 hours (Smyth et al. AIHAJ 30:470, 1969). In contact with the eyes of rabbits, o-methylcyclohexanone caused injury graded 5 on an ascending severity scale of 1 to 10 (Grant 1986, p. 1045). At an exposure concentration of 2500 ppm, rabbits and cats experienced drowsiness and difficult and irregular respiration and had a staggering gait (Lehman and Flury 1943a/Ex. 1–962). After 15 minutes of exposure to 3500 ppm, several species of animals lost coordination; after 30 minutes, the animals became prostrate. Rabbits exposed to 1822 ppm for 6 hours/day for 3 weeks showed signs of conjunctival irritation, became lethargic, and salivated excessively (Lehman and Flury 1943a/Ex. 1–962). Repeated application of large doses of o-methylcyclohexanone to the skin of rabbits produced irritation, tremor, narcosis, and death; the minimal lethal dose was reported to be between 4.9 and 7.2 g/kg (Treon, Crutchfield, and Kitzmiller 1943. J. Ind. Hyg. Toxicol. 25:199–214).

There are no reports of chronic or systemic effects in humans exposed to this substance; however, it is expected that severe exposure to o-methylcyclohexanone would produce the same irritant and narcotic effects in humans as in experimental animals (Proctor, Hughes, and Fischman 1988, p. 328). The American Conference of Governmental Industrial Hygienists (ACGIH) reports that exposure to a 100-ppm concentration will not cause narcotic effects but may cause eye and respiratory tract irritation (ACGIH 1986, p. 386).

Based on this evidence, OSHA preliminarily concludes that workers in construction, maritime, and agriculture who are exposed to o-methylcyclohexanone are at significant risk of these exposure-related effects. The Agency considers the irritation potentially caused by exposure to this substance a material impairment of health and believes it necessary to establish PELs for o-methylcyclohexanone of 50 ppm as an 8-hour TWA and 75 ppm as a 15-minute STEL. These limits, and the skin notation, will substantially reduce this significant risk in construction, maritime, and agricultural workplaces. Promulgation of these PELs for o-methylcyclohexanone will also make OSHA's limits for this substance consistent across all OSHA-regulated sectors.

OSMIUM TETROXIDE

CAS: 20816–12–0; Chemical Formula: OsO_4

H.S. No. 1298

In construction and maritime operations, OSHA has an 8-hour TWA limit of 0.002 mg/m³ for osmium tetroxide. The ACGIH has a TLV®–TWA of 0.002 mg/m³ and a TLV®–STEL of 0.006 mg/m³; NIOSH has no REL for this

substance. OSHA is proposing to add a 15-minute STEL of 0.006 mg/m³ to the 8-hour TWA PEL of 0.002 mg/m³ for osmium tetroxide in the construction and maritime industries and to extend these limits to agriculture. NIOSH (Ex. 8–47) concurred that these limits are appropriate for this substance when the Agency recently established them in general industry.

Osmium tetroxide is a noncombustible, colorless to pale yellow solid with a disagreeable, chlorine-like odor. Osmium tetroxide is used as a fat stain in pathological laboratories. It is also used as an oxidizing agent for converting olefins to glycols, in photography, and as a catalyst in organic synthesis. Osmium tetroxide was formerly used in fingerprinting (ACGIH 1986, p. 456; Merck 1983, p. 989; AIHA 1988).

Osmium tetroxide causes severe eye, nose, and respiratory tract irritation in humans and experimental animals. The oral LD_{50}s in rats and mice are 14 mg/kg and 162 mg/kg, respectively (RTECS 1990). The lowest lethal concentration in rats inhaling osmium tetroxide for 4 hours is 40 ppm (approximately 400 mg/m³) (RTECS 1990). In contact with the eyes of rabbits, a 1 percent solution of osmium tetroxide caused severe corneal damage and permanent opacity (Grant 1986, p. 682). Exposure for 30 minutes to osmium tetroxide at a concentration of 130 mg/m³ caused irritation of the mucous membranes, labored breathing, and death in rabbits; at autopsy, pulmonary edema was evident (Brunot 1933/Ex. 1–776). Toxic bone marrow effects have been reported in guinea pigs exposed 8 hours/day for 60 days to an unspecified concentration of osmium tetroxide; these animals developed normochromic anemia, as evidenced by a decrease in the number of erythrocytes, leukocytes, and reticulocytes as well as a decrease in hemoglobin levels (Hamilton and Hardy 1974a/Ex. 1–957; HSDB 1989).

Industrial experience indicates that osmium tetroxide fume concentrations in a precious metal refining plant ranged from 0.1 to 0.8 mg/m³; intermittent exposure to these concentrations produced symptoms (sometimes delayed) of lacrimation, visual disturbance, frontal headache, conjunctivitis, and cough in exposed workers (McLaughlin, Milton, and Perry 1946/Ex. 1–749). Complaints of persistent and severe nose and throat irritation were also reported (Hamilton and Hardy 1974a/Ex. 1–957). One investigator (Brunot 1933/Ex. 1–776) developed lacrimation and corneal edema (manifested as haloes around lights) after a brief exposure to osmium tetroxide at an unspecified concentration. Fairhall (1949d, as cited in ACGIH 1986/Ex. 1–3, p. 459) reported a human fatality resulting from inhalation exposure to osmium tetroxide (the concentration and duration of the exposure are not known). A laboratory worker heating osmium in the laboratory produced sufficient osmium tetroxide to cause blepharospasm, ocular pain, photophobia, and redness of the eyes (Grant 1986, p. 682). In contact with the skin, osmium compounds (including osmium tetroxide) cause eczema and dermatitis (AIHA 1988).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to osmium tetroxide, even for short periods, poses a significant risk of ocular and respiratory effects to exposed workers. The Agency considers these effects material impairments of health and preliminarily finds that the proposed limits are necessary to substantially reduce these significant risks. Accordingly, OSHA is proposing an 8-hour TWA PEL of 0.002 mg/m³ and a 15-minute STEL of 0.006 mg/m³ for osmium tetroxide in the construction, maritime, and agriculture sectors. Promulgation of these limits will make OSHA's PELs for this substance consistent across all OSHA-regulated sectors.

PARAFFIN WAX FUME

CAS: 8002–74–2; Chemical Formula: C_nH_{2n+2}

H.S. No. 1302

OSHA has no limit for paraffin wax fume in the construction, maritime, or agriculture industries. The ACGIH has a TLV®–TWA of 2 mg/m³; NIOSH has no REL for this substance. OSHA is proposing an 8-hour TWA limit of 2 mg/m³ for paraffin wax fume in construction, maritime, and agriculture. NIOSH (Ex. 8–47) concurred that this limit is appropriate for paraffin wax fume when the Agency recently established it in general industry.

Paraffin is a white or slightly yellow, odorless solid that is derived from petroleum. Paraffin is used for making candles, as a sealant or coating for paper and food products, for extracting perfumes from flowers, and as a chewing gum base. It has also been used in crayons, in stoppers for acid bottles, in cosmetics, in photography, as a lubricant, in the packaging of tobacco products, and as an anti-frothing agent in sugar refining (ACGIH 1986, p. 455; Hawley's 1987, p. 873).

In addition to mild eye, nose, and throat irritation (Genium MSDS 1990, No. 712), exposure to paraffin wax fume can cause nausea in humans. Instilled into rabbit eyes, 100 mg of paraffin caused mild irritation; applied to the skin of rabbits, this substance caused a mild degree of irritation (RTECS 1989). Although paraffin is relatively nontoxic in its solid state, the fume generated when it is in the molten state causes discomfort and nausea (Queries and Minor Notes, *JAMA* 1938/Ex. 1–308). In the most recent report of industrial exposure effects, paraffin fume is reported to cause no discomfort in most workers when the concentration is maintained at or below 2 mg/m³, although one instance of mild discomfort was reported at concentrations between 0.6 and 1 mg/m³ (Massachusetts Division of Occupational Hygiene 1970, as cited in ACGIH 1986/Ex. 1–3, p. 455). Chronic exposure of the skin to paraffin wax has caused dermatitis (Genium MSDS 1990, No. 712). There are reports that workers exposed to this substance for 10 years or longer have a higher than normal risk of developing scrotal cancer (Genium MSDS 1990, No. 712).

OSHA preliminarily finds that the adverse effects associated with excessive exposure to paraffin wax fume—sensory irritation and nausea—constitute material impairments of health. OSHA believes that the proposed PEL of 2 mg/m³ will substantially reduce these significant risks for workers in construction, maritime, and agriculture. In addition, promulgation of this limit will make the PEL for paraffin wax fume consistent across all OSHA-regulated sectors.

PHENYL ETHER

CAS: 101–84–8; Chemical Formula: $(C_6H_5)_2O$

H.S. No. 1314

OSHA's permissible exposure limit for phenyl ether in general industry, construction, and maritime is an 8-hour TWA limit of 1 ppm. There is no PEL in agriculture. The ACGIH recommends a TLV®–TWA of 1 ppm and a TLV®–STEL of 2 ppm for phenyl ether. NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL of 1 ppm for phenyl ether in agriculture. This is the limit recently established for this substance in general industry.

Phenyl ether is either a colorless liquid or a solid, depending on temperature; its vapor has a characteristically disagreeable odor. This substance finds use as a chemical intermediate, a heat-transfer medium, and an ingredient in perfumes and soaps (Hawley's 1987, p. 429). It is also used in the manufacture of high-temperature lubricants and surfactants (HSDB 1990).

Phenyl ether is an irritant of the eyes and skin (RTECS 1990; HSDB 1990). The

oral LD_{50} in rats is 3370 mg/kg (RTECS 1990); single oral doses of between 1 and 2 g/kg administered to experimental animals of various species have caused liver, spleen, kidney, thyroid, or gastrointestinal injury in those surviving the exposure (Vogel, Snyder, and Schulman 1984/Ex. 1–681). Repeated inhalation studies in rats, rabbits, and dogs have shown that a total of 20 exposures to a 4.9-ppm concentration of phenyl ether for 5 days/week, 7 hours/day produced no adverse effects. Eye and nasal irritation were observed in rats and rabbits exposed to a 10-ppm concentration (Hefner, Leong, Kociba, and Gehring 1975/Ex. 1–329). Skin and eye irritation have been reported to result only from prolonged or repeated contact with the undiluted liquid.

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 1 ppm for the agriculture sector. The Agency preliminarily concludes that this limit will protect workers in agriculture from the odor and irritant effects associated with exposure to phenyl ether. OSHA believes that these effects are material impairments of health within the meaning of the Act. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PHENYL ETHER-BIPHENYL MIXTURE

CAS: 8004–13–5; Chemical Formulas: $(C_6H_5)_2O$ and $C_6H_5C_6H_5$

H.S. No. 2127

In general industry, construction, and maritime, OSHA's permissible exposure limit for phenyl ether-biphenyl mixture is 1 ppm as an 8-hour TWA. There is no limit for this substance in agriculture. The ACGIH has no current TLV® for this substance; there is no NIOSH REL. OSHA is proposing to establish a PEL in agriculture of 1 ppm as an 8-hour TWA for phenyl ether-biphenyl mixture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Phenyl ether-biphenyl mixture, also called Dowtherm A, is a straw-colored liquid that has a disagreeable aromatic odor and that darkens on use. This substance is used as a heat transfer agent, as a dye carrier for printing and textiles, and in the production of acetyl chloride and polyester fibers (NIOSH/OSHA Occupational Health Guideline 1981, p. 3).

Phenyl ether-biphenyl mixture causes irritation of the eyes, mucous membranes, and skin, and liver and kidney damage in humans and animals. The oral LD_{50}s in rats, mice, rabbits, and guinea pigs are 2460 mg/kg, 3210 mg/kg, 4200 mg/kg, and 3000 mg/kg, respectively (RTECS 1991). Rabbits exhibited mild eye and skin irritation when 500 mg was applied to their eyes and skin, respectively, for 24 hours (RTECS 1991). Rabbits developed the following signs of mild skin irritation when undiluted Dowtherm A was applied daily to their ears: Hyperemia, edema, exfoliation, hair loss, and enlargement of the hair follicles (Hake and Rowe 1963, in Clayton and Clayton 1981, p. 2544). Small groups of rats given repeated doses of Dowtherm A by stomach tube (doses of 0.5 to 1.0 g/kg) 5 days/week for a total of 132 doses exhibited slight growth retardation and increased liver and kidney weights (rats in the 0.5 g/kg dose group); those given 1.0 g/kg had moderate growth depression and, at autopsy, showed histopathological changes in the kidneys, liver, and spleen (Hake and Rowe 1963, in Clayton and Clayton 1981, p. 2544). At autopsy, rats fed phenyl ether-biphenyl mixture at doses of 0.25 and 0.5 g/kg/day showed signs of moderate liver and kidney degeneration after 2 months of exposure (Hake and Rowe 1963, in Clayton and Clayton 1981, p. 2544). When subjected to 37 8-hour exposures to a 14-ppm concentration of this substance, rats, guinea pigs, and a monkey showed signs of emaciation, starvation, and a slight increase in liver weight; no significant histopathological changes were observed at autopsy (Hake and Rowe 1963, in Clayton and Clayton 1981, p. 2544). Rats, guinea pigs, rabbits, and monkeys exposed to the vapors of this substance at a concentration of 7 to 10 ppm for 7 hours/day, 5 days/week for 6 months showed no adverse effects (HSDB 1991).

The lowest toxic concentration of phenyl ether-biphenyl mixture reported in humans is 3 ppm; at this concentration, irritation of the eyes, nose, and pulmonary tract were observed (RTECS 1991). Exposure to vapor concentrations of 7 to 10 ppm causes extreme nausea and is painful to the eyes and upper respiratory tract (Proctor and Hughes 1978, p. 412). In one case, a man was sprayed in the eyes with this substance; although there was no immediate discomfort, he felt stinging and burning of the eyes and the surrounding skin within 5 minutes (Grant 1986, p. 373). In four other cases, corneal burns resulted from exposure to diphenyl oxide; recovery was complete within 48 hours (Grant 1986, p. 373). Severe degenerative lesions of the liver and kidneys can result from accidental ingestion (Parmeggiani 1983, p. 643).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to phenyl ether-biphenyl mixture causes eye, mucous membrane, and skin irritation. The Agency believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these exposure-related effects and that the proposed PEL of 1 ppm as an 8-hour TWA will substantially reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PHOSGENE

CAS: 75–44–5; Chemical Formula: $Cl_2C=O$

H.S. No. 2129

In general industry, construction, and maritime, OSHA has an 8-hour TWA limit of 0.1 ppm for phosgene. There is no PEL for this substance in agriculture. The ACGIH TLV®–TWA for phosgene is 0.1 ppm (0.40 mg/m³) as an 8-hour TWA. The NIOSH REL for phosgene is 0.1 ppm (0.4 mg/m³) as a 10-hour TWA, with a 15-minute ceiling of 0.2 ppm (0.8 mg/m³). OSHA is proposing an 8-hour TWA PEL of 0.1 ppm for phosgene in agriculture. This is the limit recently established for this substance in general industry.

Phosgene is a poisonous, colorless, nonflammable gas or volatile liquid with a sweet, pungent, or musty odor (AIHA 1990, p. 1). Phosgene is used as an intermediate in the production of polyurethane, isocyanates, polycarbonates, pesticides, herbicides, pharmaceuticals, and dyes and as a chlorinating agent for the separation of uranium ores. This substance was used as a war gas in World War I. Phosgene gas is formed during arc or gas welding in the presence of chlorinated hydrocarbons, such as in locations with degreasing operations, and during the welding of aluminum (Grayson 1985, pp. 868–869; Hawley's 1987, p. 908; Braker and Mossman 1980, p. 596; AIHA 1990, p. 2). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Phosgene is a severe respiratory irritant; exposure to high concentrations causes pulmonary edema, and long-term exposure to low concentrations causes chronic lung disease. The LC_{50} in monkeys is 600 mg/m³ (150 ppm) for 1 minute, and the LC_{50} in rats is 1400 mg/m³ (350 ppm) for 30 minutes (RTECS 1991). Exposure of experimental animals to phosgene has caused extreme congestion, pulmonary edema, lobular and pseudolobular pneumonia, atelectasis, emphysema, and chronic pneumonitis (Chemical Manufacturer's Phosgene Panel 1988). Rats exposed to a 0.5-ppm concentration for 120 minutes developed chronic pneumonitis, with

thickening of the respiratory bronchioles and alveolar damage (Rinehart 1964; Gross 1965). Forty-one percent of all goats, cats, rabbits, guinea pigs, rats, and mice exposed to a 0.8-mg/m³ (0.2-ppm) concentration of phosgene for 5 hours/day for 5 consecutive days developed pulmonary edema and 4 percent of these animals developed extensive lung lesions (Cameron 1942). Cats exposed to high phosgene concentrations (not further specified) developed corneal opacification (Laquer 1921).

The acute hazard posed by exposure of humans to phosgene is pulmonary edema; the edema may not develop for 1 to 24 hours after exposure (Diller 1985). Based on observations made when this substance was used in warfare, the estimated LC_{50} in humans is 3200 mg/m³ (800 ppm) for 1 minute or 400 ppm for 2 minutes (Chemical Warfare Service 1944; Chasis 1944). In humans, the irritation caused by phosgene does not give sufficient warning of hazardous concentrations (Stokinger 1957). Exposure of humans to a 3-ppm concentration can cause immediate irritation of the throat, exposure to 4 ppm causes immediate irritation of the eyes, and exposure to 4.8 ppm causes coughing; brief exposure to 50 ppm can be rapidly fatal (Cucinell 1974). Liquid phosgene splashed into the eyes caused total opacification and perforation of both corneas in one individual (D'Osvaldo 1928). In contact with the skin, liquid phosgene causes skin burns (AIHA 1978). A group of 326 workers exposed to phosgene concentrations ranging from nondetectable to greater than 0.13 ppm showed no signs of chronic lung disease (NIOSH 1976). Six workers exposed chronically to low concentrations of phosgene developed chronic lung disease (Galdston, Luetscher, and Longcope et al. 1947).

The evidence described above shows that phosgene exposure causes severe irritation of the eyes and nose and damage to the lungs; exposure to high concentrations may cause pulmonary edema and death. Accordingly, OSHA preliminarily concludes that, in the absence of a limit, workers in agriculture are potentially at significant risk of experiencing these exposure-related effects. The Agency believes that the proposed limit of 0.1 ppm for phosgene in agriculture will substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PHOSPHORIC ACID

CAS: 7664–38–2; Chemical Formula: H_3PO_4

H.S. No. 1322

OSHA has a limit for phosphoric acid in the construction and maritime sectors of 1 mg/m³ as an 8-hour TWA. The ACGIH has a TLV®–TWA limit of 1 mg/m³ and a TLV®–STEL of 3 mg/m³ for phosphoric acid; NIOSH has no REL. The Agency is retaining an 8-hour TWA of 1 mg/m³ in construction and maritime, proposing a 15-minute STEL of 3-mg/m³ in these two sectors, and proposing both limits in agriculture. NIOSH (Ex. 8–47) concurred that these limits are appropriate for phosphoric acid when the Agency recently established them in general industry.

Phosphoric acid is a colorless, odorless solid at temperatures below 21°C but becomes a viscous, clear liquid at higher temperatures. It is used in fertilizers, pharmaceuticals, animal feed, detergents, foods, and beverages and in water treatment, pickling and rustproofing metals, sugar refining, and electropolishing. Phosphoric acid is also used as a catalyst in ethanol manufacture, as a binder for ceramics, and as a laboratory reagent (ACGIH 1986, p. 483; Hawley's 1987, p. 910).

Exposure to phosphoric acid causes sensory irritation; contact of the tissues with phosphoric acid also causes corrosive burns. The oral LD_{50} in rats is 1530 mg/kg, and the dermal LD_{50} in rabbits is 2740 mg/kg (RTECS 1990). In contact with the skin of rabbits for 24 hours, phosphoric acid caused severe skin irritation; instilled into the eyes of rabbits, this substance caused severe eye irritation (RTECS 1990).

In humans, phosphoric acid mist causes mild irritation of eyes, throat, and skin, and the dust is reported to be especially irritating to the skin when moisture is present. The AIHA Hygienic Guide for phosphoric acid reports that this substance is less hazardous than either nitric or sulfuric acid (AIHA 1957/Ex. 1–709). The lowest lethal dose for humans (route not specified) is estimated to be 220 mg/kg (RTECS 1990). At concentrations ranging from 0.8 to 5.4 mg/m³, fumes of phosphorus pentoxide (the anhydride of phosphorus acid) caused no discomfort, but exposure to concentrations ranging from 3.8 to 11.3 mg/m³ caused coughing in unacclimated workers (Rushing 1957, in ACGIH 1986, p. 483). A dilute solution buffered to a pH of 2.5 caused stinging but no permanent eye injury when dropped into the human eye. A 75-percent solution of this substance, however, caused severe burns when placed on the skin (AIHA Hygienic Guide Series 1957).

To protect workers in construction, maritime, and agriculture from the risk of eye, skin, and respiratory tract irritation associated with exposure to this substance, OSHA is retaining the TWA limit of 1 mg/m³ in construction and maritime, proposing a STEL of 3 mg/m³ in these sectors, and proposing both limits in agriculture. The Agency preliminarily concludes that both 8-hour TWA and STEL limits are necessary to substantially reduce the risk that workers in these sectors will experience these material health impairments. In addition, promulgation of these limits will make OSHA's PELs for phosphoric acid consistent across all OSHA-regulated sectors.

PHOSPHORUS PENTACHLORIDE

CAS: 10026–13–8; Chemical Formula: PCl_5

H.S. No. 2131

OSHA's PEL for phosphorus pentachloride in general industry, construction, and maritime is 1 mg/m³ as an 8-hour TWA; there is no PEL in agriculture. The ACGIH has a TLV®–TWA of 0.1 ppm (1 mg/m³) for phosphorus pentachloride; there is no NIOSH REL. OSHA is proposing an 8-hour TWA PEL of 1 mg/m³ for phosphorus pentachloride in agriculture. This is the limit recently established for this substance in general industry.

Phosphorus pentachloride is a white to pale-yellow crystalline solid that fumes in moist air. It has a pungent, unpleasant odor (ACGIH 1986, p. 485; Proctor, Hughes, and Fischman 1988, p. 413). Phosphorus pentachloride is used in the manufacture of agricultural chemicals, plasticizers, chlorinated compounds, gasoline additives, surfactants, and pharmaceuticals. It is also used as a dehydrating agent and in the manufacture of acetylcellulose (Proctor, Hughes, and Fischman 1988, p. 413; Sittig 1985, p. 722). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In contact with moisture or moist tissues, phosphorus pentachloride decomposes to phosphorus oxychloride, phosphoric acid, and hydrochloric acid (Clayton and Clayton 1982, p. 2126). Phosphorus pentachloride is a severe irritant of the eyes, mucous membranes, and upper respiratory tract. The oral LD_{50} in rats is 660 mg/kg; the LC_{50} for the same species is 205 mg/m³ for 4 hours (RTECS 1990). A concentration of 120 ppm phosphorus pentachloride was fatal to mice in 10 minutes (Proctor, Hughes, and Fischman 1988, p. 413; ACGIH 1986, p. 485).

In humans, exposure to phosphorus pentachloride (at an unspecified concentration) has caused irritation of the eyes and respiratory passages and the development of bronchitis (von Oettingen 1952). Workers exposed to high concentrations of this substance may experience erosion of dental enamel, and chronic exposure may lead to chronic bronchitis (Rom 1983, p. 498). Severe overexposure to phosphorus pentachloride vapor may cause delayed-onset pulmonary edema. Repeated skin contact with this material may cause dermatitis (Proctor, Hughes, and Fischman 1988, p. 413).

Based on this evidence in humans and animals, OSHA preliminarily concludes that agricultural workers exposed to this substance at the levels permitted by the absence of a limit are at significant risk of experiencing eye, mucous membrane, and upper respiratory tract irritation and may also be at risk of developing chronic bronchitis. The Agency believes that establishing a PEL of 1 mg/m^3 as an 8-hour TWA will protect workers in agriculture from these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PHOSPHORUS TRICHLORIDE
CAS: 7719–12–2; Chemical Formula: PCl_3
H.S. No. 1325

In the construction and maritime industries, OSHA's limit for phosphorus trichloride is 0.5 ppm as an 8-hour TWA. The ACGIH has a TLV®–TWA of 0.2 ppm and a TLV®–STEL of 0.5 ppm. NIOSH has no REL for phosphorus trichloride but concurs (Ex. 8–47) that the proposed limits are appropriate. OSHA is proposing an 8-hour TWA PEL of 0.2 ppm and a 15-minute STEL of 0.5 ppm for workplaces in construction, maritime, and agriculture.

Phosphorus trichloride is a noncombustible, colorless, fuming liquid. Phosphorus trichloride is used as an intermediate in the preparation of pesticides, surfactants, gasoline additives, plasticizers, and dyestuffs. It is also used as a chlorinating agent and catalyst and as a ingredient of textile finishing agents (ACGIH 1986, p. 486).

In addition to severe sensory irritation, phosphorus trichloride causes corrosive burns in contact with the eyes or skin. The oral LD_{50} in rats is 550 mg/kg, and the lowest lethal concentration in the same species is 104 ppm for 4 hours (RTECS 1990). Acutely poisoned animals showed nephrosis at autopsy (Weeks et al. 1964). Cats and guinea pigs showed signs of mild systemic poisoning after exposure to 0.7 ppm for 6 hours (Butjagin 1904, as cited in ACGIH 1986/Ex. 1–3, p. 486). At a concentration of 2 to 4 ppm, cats developed breathing difficulty and conjunctivitis after 6 hours; when the concentration was increased to between 23 and 90 ppm, the corneas of these animals became cloudy (Grant 1986, p. 736). The primary occupational hazards associated with exposure to phosphorus trichloride are respiratory irritation and poisoning involving cough, bronchitis, pneumonia, and conjunctivitis (Henderson and Haggard 1943e/Ex. 1–1086; International Labour Office 1934b, as cited in ACGIH 1986/Ex. 1–3, p. 486; Sassi 1954/Ex. 1–931). The onset of pulmonary edema, which follows severe overexposures, may be delayed for several hours (Clayton and Clayton 1981, p. 2128). Long-term exposure leads to chronic cough and wheezing, although these effects are nonprogressive and are not associated with fibrotic lung disease (Proctor, Hughes, and Fischman 1988, p. 414).

Because of the acutely irritating effects of this substance, OSHA preliminarily concludes that both a TWA and a STEL are necessary to substantially reduce the significant risk of respiratory and eye irritation potentially posed to workers exposed to phosphorus trichloride in construction, maritime, and agriculture. The Agency considers these adverse effects to be material impairments of health. Therefore, OSHA is proposing to revise its limit for phosphorus trichloride to 0.2 ppm as an 8-hour TWA and to add a 0.5 ppm 15-minute STEL for this substance in the construction, maritime, and agriculture industries. Promulgation of these limits will make OSHA's PELs for phosphorus trichloride consistent across all regulated sectors.

POTASSIUM HYDROXIDE
CAS: 1310–58–3; Chemical Formula: KOH
H.S. No. 1334

OSHA has no limit for potassium hydroxide in the construction, maritime, and agriculture industries. The ACGIH has a TLV® of 2 mg/m^3 as a ceiling. NIOSH has no REL for this substance but concurs with the limit being proposed (Ex. 8–47). OSHA is proposing a ceiling limit of 2 mg/m^3 for potassium hydroxide in the construction, maritime, and agriculture industries; this is the limit recently established for this substance in general industry.

Potassium hydroxide is a white, deliquescent solid that is available in the form of pellets, sticks, lumps, or flakes. It is used in the manufacture of liquid fertilizers, herbicides, liquid soap, potassium carbonate, and tetra potassium pyrophosphate and as an absorbent for carbon dioxide and hydrogen sulfide. It is also used in dyestuffs, paint removers, food additives, storage batteries, and fuel cells and in electroplating operations (Hawley's 1987, p. 956; Merck 1983, p. 1102).

In addition to sensory irritation, potassium hydroxide causes tissue destruction of the eyes and skin and, if ingested, rapid corrosion of the esophagus and stomach. The oral LD_{50} in rats is 273 mg/kg (RTECS 1990). In rabbits and guinea pigs, contact of the skin with 50 mg potassium hydroxide for 24 hours caused severe skin irritation; instilled into rabbit eyes, 1 mg of this substance caused a moderate degree of eye irritation (RTECS 1990). In a skin painting study in mice, tumors identical to those resulting from repeated coal tar applications developed on the skin after 46 weeks of painting with potassium hydroxide (Clayton and Clayton 1981, p. 3056).

Inhalation of potassium hydroxide causes severe irritation of the respiratory tract and leads to sneezing, coughing, and pain (Clayton and Clayton 1981, p. 3056). Repeated exposure may cause perforation of the nasal septum (ACGIH 1986, p. 495). Accidental ingestion of a solution of potassium hydroxide may produce corrosion and perforation of the esophagus and stomach (Clayton and Clayton 1981, p. 3056). Miners exposed to potassium hydroxide concentrations above 2 mg/m^3 had the following respiratory symptoms: phlegm production, mild shortness of breath, and chronic cough (Markham JW et al. 1981, AIHAJ 42(9):671). Contact of the eye with potassium hydroxide causes disintegration and sloughing of the conjunctival and corneal epithelium, corneal opacification, edema, and ulceration of the eyes (Grant 1986, p. 756).

Based on this evidence, OSHA preliminarily concludes that exposure to potassium hydroxide causes irritation ranging in degree from mild to chemical pneumonitis (Proctor, Hughes, and Fischman 1988, p. 420); in addition, this substance is corrosive, destroying any tissue it comes into contact with. OSHA believes that these exposure-related effects constitute material health impairments. To substantially reduce these significant risks, OSHA is proposing a ceiling limit of 2 mg/m^3 for potassium hydroxide in the construction, maritime, and agriculture industries. Promulgation of this limit will make OSHA's PEL for potassium hydroxide consistent across all OSHA-regulated sectors.

PROPYLENE GLYCOL MONOMETHYL ETHER

CAS: 107–98–2; Chemical Formula: $CH_3OCH_2CHOHCH_3$

H.S. No. 1343

In the construction, maritime, and agriculture industries, OSHA has no limit for propylene glycol monomethyl ether. The ACGIH has a TLV®–TWA of 100 ppm and a TLV®–STEL of 150 ppm for this substance. NIOSH has no REL. OSHA is proposing an 8-hour TWA of 100 ppm and a 15-minute STEL of 150 ppm for propylene glycol monomethyl ether in the construction, maritime, and agriculture sectors. NIOSH (Ex. 8–47) concurred that these limits were appropriate when the Agency recently established them in general industry.

Propylene glycol monomethyl ether is a flammable, colorless liquid with a characteristic ether-like odor. It is used as a solvent for celluloses, acrylics, dyes, inks, and stains. Propylene glycol monomethyl ether also is used in the solvent-sealing of cellophane.

In addition to sensory irritation, exposure to this substance causes central nervous system depression and liver and kidney effects in experimental animals. The oral LD_{50} in rats is 5660 mg/kg, and the lowest lethal concentration in the same species is 7000 ppm for 6 hours (RTECS 1990). The dermal LD_{50} in rabbits lies between 13 and 14 g/kg (Rowe, McCollister, Spencer et al. 1954). Acutely poisoned animals showed central nervous system depression before death (HSDB 1990). Applied to the skin of rabbits, propylene glycol monomethyl ether caused mild irritation, and instillation into rabbit eyes produced the same effect (RTECS 1990). Repeated application of 7 to 10 ml/kg/day propylene glycol monomethyl ether to the skin of rats over a 90-day period caused narcosis and death, while doses of 2 to 4 ml/kg/day caused only mild narcosis (Rowe, McCollister, Spencer et al. 1954, Arch. Ind. Hyg. Occup. Med. 9:509). Rats and monkeys exposed on 132 of 186 days to a propylene glycol mono-methyl ether concentration of 800 ppm showed no adverse effects, and rats and guinea pigs exposed to a 1500-ppm concentration on 130 of 184 days also showed no adverse effects (Rowe, McCollister, Spencer et al. 1954). Rats given twenty-six 3 g/kg doses of this substance over a 35-day period showed minor liver and kidney effects at autopsy; 1 g/kg doses given on the same regimen produced no gross or histopathological changes (Rowe, McCollister, Spencer et al. 1954).

In humans, exposure to propylene glycol monomethyl ether causes tearing of the eyes at concentrations above 100 ppm (specific concentration not specified) (Stewart, Baretta, Dodd, and Torkelson 1970; Arch. Env. Hlth. 20:218). However, narcotic effects are not seen in exposed individuals until the concentration approaches 100 ppm (Stewart et al. 1970).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to this substance in the workplace is potentially associated with irritant and narcotic effects. The Agency considers effects of this nature material impairments of health and believes that the proposed PELs of 100 ppm as an 8-hour TWA and 150 ppm as a 15-minute STEL are necessary to reduce these significant occupational risks. Promulgation of these limits will also make OSHA's PELs for propylene glycol monomethyl ether consistent across all OSHA-regulated sectors.

ROSIN CORE SOLDER PYROLYSIS PRODUCTS

CAS: None; Chemical Formula: None

H.S. No. 1350

OSHA has no limit for rosin core solder pyrolysis products, measured as formaldehyde, in the construction, maritime, and agriculture industries. The ACGIH has a TLV®–TWA of 0.1 mg/m³; NIOSH has no REL for this substance. The Agency is proposing an 8-hour TWA of 0.1 mg/m³ for rosin core pyrolysis products in the construction, maritime, and agriculture industries. This is the limit recently promulgated for this substance in general industry.

Rosin is a pale yellow to amber, translucent solid that consists of about 90 percent resin acids and 10 percent neutral material. Gum rosin is used as a soldering flux, and soldering with this substance or a solder that contains it causes the release of thermal decomposition products (ACGIH 1986, p. 514). The pyrolysis products of rosin core solder include acetone, methyl alcohol, aldehydes, carbon monoxide and dioxide, methane, ethane, abietic acid, and related diterpene acids (ACGIH 1986, p. 514).

Exposure to these products causes marked irritation of the eyes, nose, and throat (Cralley and Cralley 1985, p. 180). A 2-week exposure of guinea pigs and rats to these products at average concentrations of 0.96 mg/m³ caused a reduction in the rate of weight gain in male guinea pigs, abnormal liver-to-body-weight ratios in guinea pigs of both sexes, and abnormal heart-to-body-weight ratios in male rats; at autopsy, the lungs of these animals were hyperemic (Industrial Bio-test Laboratories, Inc. 1967, as cited in ACGIH 1986/Ex. 1–3, p. 514).

In humans, slight bronchial irritation has been reported on exposure to a rosin core pyrolysis product concentration of 1 mg/m³ or below (Industrial Bio-test Laboratories, Inc. 1967, as cited in ACGIH 1986/Ex. 1–3, p. 514). Several workers who were chronically exposed to pyrolysis product levels as high as 0.15 mg/m³ had to be removed from exposure because of intractable upper respiratory tract irritation; when concentrations were kept below 0.10 mg/m³, such irritation was not reported (Christy 1965, as cited in ACGIH 1986/Ex. 1–3, p. 514). In a study designed to quantify dose-response levels for irritation in human volunteers, subjects were exposed for 15 minutes to these products at aldehyde concentrations (measured as formaldehyde, which is the best indirect measure of rosin pyrolysis products) of 0.04 to 0.2 mg/m³ (U.S. Public Health Service 1965, as cited in ACGIH 1986/Ex. 1–3, p. 514). Subjects detected the odor at 0.07 mg/m³, and 80 percent of subjects reported moderate to severe irritation of the eyes, nose, and throat at concentrations of 0.12 mg/m³ or above. At levels below 0.05 mg/m³, fewer than 10 percent of subjects experienced irritation. Mucous membrane irritation occurred in 30 percent of subjects exposed to a concentration of 0.07 mg/m³ (U.S. Public Health Service 1965, as cited in ACGIH 1986/Ex. 1–3, p. 514).

Based on this evidence, OSHA is proposing an 8-hour TWA limit of 0.1 mg/m³, measured as formaldehyde, for rosin core solder pyrolysis products in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that this limit is necessary to protect employees in these sectors from the significant risk of eye, nose, and throat irritation that is associated with exposure to these pyrolysis products. Promulgation of this limit will also make OSHA's PELs for this substance consistent across all OSHA-regulated sectors.

SODIUM BISULFITE

CAS: 7631–90–5; Chemical Formula: $NaHSO_3$

H.S. No. 1365

OSHA has no limit for sodium bisulfite in construction, maritime, and agricultural workplaces. The ACGIH has a TLV®–TWA of 5 mg/m³; NIOSH has no REL for this substance. The Agency is proposing a limit of 5 mg/m³ as an 8-hour TWA in the construction, maritime, and agriculture industries. NIOSH (Ex. 8–47) agreed with the selection of this limit when OSHA recently established it in general industry.

Sodium bisulfite is a white crystalline powder that has an odor like that of sulfur dioxide. It is used as a food preservative (particularly as a lettuce freshener in salad bars) and as a reducing agent, analytical reagent, dietary supplement, and color preservative for pale crepe paper. It is also used in cask sterilization, fermentation, textiles, and copper and brass plating and to bleach groundwood (Hawley's 1987, p. 1054).

In addition to sensory irritation, sodium bisulfite causes central nervous system effects in animals and humans. The oral LD_{50} in rats is 2 g/kg (Dow Chemical Company 1977d, as cited in ACGIH 1986/Ex. 1–3, p. 534), and the intraperitoneal LD_{50} in rats is 475 mg/kg (RTECS 1990). Animals acutely poisoned by intraperitoneal administration convulsed and had altered sleep times (RTECS 1990). In other acute poisonings, death is preceded by irritability, restlessness, convulsions, periods of apnea, terminal respiration, and cardiovascular collapse (Gosselin, Smith, and Hodge 1984, p. II–123).

In humans, sodium bisulfite causes eye and mucous membrane irritation, dermatitis, and sensitization reactions. Industrial exposure to unspecified concentrations of sodium bisulfite has caused irritation of the eyes and respiratory tract that was described as mild (ACGIH 1986, p. 534). A recent study (Howland WC, Simon RA, J. Allergy, Clin. Immunol. 83(6):1079–1082, 1989) has shown that sodium bisulfite-treated lettuce is capable of provoking bronchospasm in sulfite-sensitive individuals who have asthma. Another study has shown that a worker exposed to sodium bisulfite during the preparation of animal feed developed acute dermatitis on the exposed areas of his body (face, neck, hands, and forearms); positive patch tests confirmed that sodium bisulfite was the allergen (Dinis, Brandao, and Faria 1988; Contact Dermatitis 18(3):170–171).

Based on this evidence in humans and animals, OSHA preliminarily finds that exposure to sodium bisulfite presents a significant occupational risk of irritation, dermatitis, and sensitization, and that these effects constitute material impairments of health. To substantially reduce these risks, OSHA believes it necessary to establish a 5-mg/m^3 8-hour TWA for sodium bisulfite in construction, maritime, and agriculture workplaces, and this is the limit being proposed. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

SODIUM HYDROXIDE

CAS: 1310–73–2; Chemical Formula: NaOH

H.S. No. 1367

OSHA has an 8-hour TWA of 2 mg/m^3 for sodium hydroxide in the construction and maritime industries. The ACGIH TLV® is a ceiling limit of 2 mg/m^3, and NIOSH currently has a REL for this substance of 2 mg/m^3 as a 15-minute ceiling short-term limit. OSHA is proposing a 2-mg/m^3 ceiling limit for sodium hydroxide in the construction, maritime, and agriculture industries. NIOSH (Ex. 8–47) concurred with this limit when OSHA recently established it in general industry.

Sodium hydroxide is a white, deliquescent solid used in the manufacture of chemicals, as a neutralizing agent in petroleum refining, and as an ingredient in detergents and soaps. This substance is also widely used in textile processing, vegetable oil refining, and the reclamation of rubber. In addition, sodium hydroxide is used for regenerating ion exchange resins, in organic fusions, as a lab reagent, in etching and electroplating, and as a food additive (ACGIH 1986, p. 535; Hawley's 1987, p. 1062).

Sodium hydroxide is a severe irritant of the eyes, mucous membranes, and skin. In addition to sensory irritation, sodium hydroxide destroys tissue on contact. The lowest lethal oral dose in rabbits is 500 mg/kg (RTECS 1990). A 1-percent solution of sodium hydroxide instilled into the eyes of monkeys caused severe eye irritation, and 500 mg placed on the skin of rabbits caused severe skin irritation (RTECS 1990). Two of 10 rats exposed for 30 minutes twice a week to an aerosol of 40 percent aqueous sodium hydroxide whose particles were less than 1 um in diameter died after 3 weeks. Histopathological examination showed mostly normal lung tissue with foci of enlarged alveolar septae, emphysema, bronchial ulceration, and enlarged lymph adenoidal tissues (Wands 1981b, in Clayton and Clayton 1982, p. 3062).

Although the inhalation of sodium hydroxide is usually of secondary importance in industrial exposures, the effects of inhaling the dust or mist vary from mild irritation of the nose, which occurs on brief exposure to 2 mg/m^3, to severe pneumonitis and ulceration of the nasal septum, which may occur at very high (but not further specified) exposures (Proctor, Hughes, and Fischman 1988, p. 444; ACGIH 1986, p. 535). The greatest industrial hazard is rapid tissue destruction of the eyes or skin upon contact either with the solid or with concentrated solutions (Proctor, Hughes, and Fischman 1988, p. 444). Contact of sodium hydroxide with the eyes causes disintegration and sloughing of the conjunctival and corneal epithelium, corneal opacification, marked edema, and ulceration; after 7 to 13 days, gradual recovery may begin or the lesion may progress to ulceration and corneal opacification. In severe eye burns, symblepharon, with overgrowth of the cornea by a vascularized membrane, progressive or recurrent corneal ulceration, and permanent corneal opacification may occur (Proctor, Hughes, and Fischman 1988, p. 444). Grant (1986/Ex. 1–975) states that sodium hydroxide causes "some of the most severe, blinding injuries of the eye." On the skin, solutions of 25 to 50 percent sodium hydroxide cause a sensation of irritation within about 3 minutes; with solutions of 4 percent, the sensation of burning does not occur until several hours later. If not removed from the skin, sodium hydroxide causes severe burns and deep ulcerations. Exposure to the dust or mist of sodium hydroxide may cause multiple small burns, with temporary loss of hair (Proctor, Hughes, and Fischman 1988, p. 445). Nagao and co-workers (1972) examined skin biopsies from volunteers who had a 1 N solution (equal to a 4-percent solution) of sodium hydroxide applied to their arms for 15 to 180 minutes. Progressive changes, beginning with dissolution of the cells in the horny layer and progressing through edema to total destruction of the epidermis, occurred in these individuals within 60 minutes (Nagao, Stroud, Hamada et al. 1972).

The irritant effect of sodium hydroxide and its markedly corrosive action on all body tissue can result even from brief (one minute or more) exposures to airborne concentrations above the 2-mg/m^3 level; the acute nature of these effects is evident in the studies described above. Based on this evidence, OSHA preliminarily concludes that establishing a ceiling of 2 mg/m^3 in the construction, maritime, and agriculture industries is necessary to reduce the significant risks of eye and skin burns and respiratory irritation that occur even on brief exposure to sodium hydroxide. OSHA considers the irritant effects resulting from exposure to sodium hydroxide material impairments of health and is accordingly proposing a ceiling limit of 2 mg/m^3 for sodium hydroxide in the construction, maritime, and agriculture industries. Promulgation of this PEL will make the limit for sodium hydroxide consistent across all OSHA-regulated sectors.

SODIUM METABISULFITE

CAS: 7681–57–4; Chemical Formula: $Na_2S_2O_5$
H.S. No. 1368

OSHA has no exposure limit for sodium metabisulfite in the construction, maritime, and agriculture industries. The ACGIH has an 8-hour TLV®–TWA of 5 mg/m³; NIOSH has no REL for this substance. The Agency is proposing a 5-mg/m³ TWA for sodium metabisulfite in the construction, maritime, and agriculture industries. NIOSH (Ex. 8–47) concurred with the selection of this limit when OSHA established it in general industry.

Sodium metabisulfite can occur either in the form of a solid block or as white crystals; this substance smells like sulfur dioxide. Sodium metabisulfite is used as a preservative, in the manufacture of pharmaceuticals and foods, and as a lab reagent (HSDB 1989).

A 2-year study conducted by the Dow Chemical Company (1977e, as cited in ACGIH 1986/Ex. 1–3, p. 535), in which rats ingested 0.215 percent sodium metabisulfite, demonstrated that oral administration of sodium metasulfite caused no adverse effects in the rats. The LD_{50} in rats is 115 mg/kg by intravenous administration (RTECS 1990). Oral administration of sodium metabisulfite to rats at doses of 20 mg/kg or 40 mg/kg produced stillbirths or other adverse effects on the offspring (RTECS 1990).

In humans, inhalation of the dust of sodium metasulfite causes mucous membrane and sensitization effects similar to those that caused by sodium bisulfite (ACGIH 1986, p. 535; Nichol, Nix, Chung, and Barnes 1989, in Thorax 44(12):1009–1014). Fifteen of 18 asthmatic and atopic volunteers who were challenged with sodium metabisulfite developed bronchoconstriction of sufficiently severe degree to cause a 20-percent reduction from baseline levels in forced expiratory volume in one second (FEV_1) (Nichol, Nix, Chung, and Barnes 1989). In addition, prolonged or repeated exposure to the dust of this substance causes dermatitis (Hazardous Substance Fact Sheet 1985, p. 2).

Based on this evidence in humans and animals, OSHA is proposing an 8-hour TWA of 5 mg/m³ for sodium metabisulfite in the construction, maritime, and agriculture industries. The Agency preliminarily finds that establishing this limit is necessary to reduce the risk of eye and skin irritation and pulmonary sensitization associated with exposure to high concentrations of sodium metabisulfite dust. OSHA has preliminarily determined that these effects constitute material impairments of health. Accordingly, OSHA is proposing a 5-mg/m³ limit as an 8-hour TWA for this substance; promulgation of this limit will make the PELs for this substance consistent across all OSHA-regulated sectors.

SULFUR MONOCHLORIDE
CAS: 10025–67–9; Chemical Formula: S_2Cl_2
H.S. No. 1376

OSHA's PEL for sulfur monochloride in the construction and maritime industries is 1 ppm as an 8-hour TWA. There is no PEL in agriculture. The ACGIH has a TLV® of 1 ppm as a ceiling limit, and NIOSH has no REL for this substance. The Agency is proposing to revise its limit for sulfur monochloride in the construction and maritime industries to a 1 ppm ceiling and to extend this limit to agriculture. NIOSH (Ex. 8–47) concurred with this limit when OSHA recently established it for general industry.

Sulfur monochloride is an amber, oily, nonflammable, fuming liquid that has a penetrating odor. It is used in the manufacture of insecticides, to vulcanize rubber, in chemical synthesis, to harden wood, in textile finishing, and in gold extraction. Sulfur monochloride is also used as a polymerization catalyst for vegetable oils (ACGIH 1986, p. 545; Merck 1983, p. 1288).

In addition to sensory irritation, exposure to sulfur monochloride can lead to burns of the eyes and skin. Sulfur monochloride is a primary irritant that affects the upper respiratory tract by releasing hydrochloric acid (HCl) on contact with moisture (Henderson and Haggard 1943g, as cited in ACGIH 1986/Ex. 1–3, p. 545). Animal toxicity studies revealed that mice exposed to a 150-ppm concentration of sulfur monochloride died after 1 minute (Flury and Zernik 1931k/Ex. 1–979). Cats exposed for 15 minutes to 60 ppm sulfur monochloride all died within a few days, but exposure to a concentration of 12 ppm for 15 minutes was tolerated by these animals (Henderson and Haggard 1943g, as cited in ACGIH 1986/Ex. 1–3, p. 545).

In humans, sulfur monochloride causes tearing of the eyes and coughing; if the exposure is severe, pulmonary edema may develop (Proctor, Hughes, and Fischman 1988, p. 454). Splashed into the eyes, this substance causes severe damage and may cause permanent scarring; in contact with the skin, it causes burns (ACGIH 1986, p. 545). A study by Elkins (1959g, as cited in ACGIH 1986/Ex. 1–3, p. 545) of workers in the rubber industry found that exposure to sodium monochloride concentrations of 2 to 9 ppm was mildly irritating; however, because of moisture in the air, the concentrations to which these workers were exposed may have included a high proportion of hydrochloric acid.

Based on this evidence, the Agency preliminarily concludes that workers in the construction, maritime, and agriculture industries are at significant risk from the primary irritation that could occur even on short-term exposure to elevated concentrations of sulfur monochloride. Since 2 ppm is reported to be an effect level for mild irritation, OSHA preliminarily finds that revising its 8-hour TWA limit in construction and maritime to 1 ppm as a ceiling and extending this ceiling limit to agriculture is a reasonable and necessary action to protect workers in these sectors from the significant risk of sensory irritation, which constitutes a material impairment of health. Therefore, OSHA is proposing a ceiling limit for sulfur monochloride of 1 ppm in the construction, maritime, and agricultural sectors. Promulgation of this limit will also make the PEL for this substance consistent across all OSHA-regulated sectors.

SULFUR PENTAFLUORIDE
CAS: 5714–22–7; Chemical Formula: S_2F_{10}
H.S. No. 1377

OSHA's limit for sulfur pentafluoride in construction and maritime workplaces is 0.025 ppm as an 8-hour TWA. There is no PEL in agriculture. The ACGIH has a ceiling limit for sulfur pentafluoride of 0.01 ppm, and NIOSH has no REL for this substance. The Agency is proposing a 0.01 ppm ceiling limit for sulfur pentafluoride in construction, maritime, and agriculture. NIOSH concurred (Ex. 8–47) with this limit when OSHA recently established it for general industry.

Sulfur pentafluoride is a colorless gas or liquid with a sulfur dioxide-like odor. Sulfur pentafluoride is used as a reactant in research applications (Genium MSDS 1989, No. 262).

Sulfur pentafluoride causes severe respiratory tract irritation in animals. The LC_{50} in rats is 0.2 ppm for 10 minutes; in monkeys, the LC_{50} is 0.9 ppm for 10 minutes (RTECS 1990). Saunders, Shoshkes, DeCarlo, and Brown (1953/Ex. 1–610) established that the intravenous LD_{50} for sulfur pentafluoride in rabbits is 5.8 mg/kg, and that death was due to fulminant pulmonary edema. In a study in which rats were exposed to sulfur pentafluoride for 16 to 18 hours, concentrations of 0.1 ppm caused lung irritation, 0.5 ppm resulted in severe pulmonary lesions, and 1 ppm proved fatal (Greenberg and Lester 1950/Ex. 1–590). One-hour exposures to 10 ppm

sulfur pentafluoride caused diffuse hemorrhagic lesions in the lungs of rats, and 1-hour exposures to 1 ppm caused severe congestion of the lungs. Rats exposed for one hour to 0.1 ppm showed no effects. Subsequent examination of rats surviving the 10- and 1-ppm exposures revealed that the lungs had returned to normal after 24 hours (Greenberg and Lester 1950/Ex. 1–590). Dogs exposed intravenously to a 1-mg/kg dose of sulfur pentafluoride experienced vascular shock and died from acute pulmonary edema (RTECS 1989). There are no reports of human exposure to sulfur pentafluoride.

Based on this evidence, OSHA is proposing to revise the sulfur pentafluoride limit in construction and maritime to 0.01 ppm as a ceiling and to extend this limit to agriculture. The 0.01-ppm ceiling is appropriate because of evidence showing that even brief exposures to 1 ppm caused pulmonary effects and prolonged exposures to 0.1 ppm caused lung irritation in animals. OSHA preliminarily concludes that this limit is necessary to substantially reduce the risks of irritation and other pulmonary effects potentially associated with exposure to this substance. The Agency considers these effects material impairments of health. Promulgation of a 0.01 ppm ceiling for sulfur pentafluoride will make OSHA's PEL for this substance consistent across all regulated sectors.

SULFURIC ACID

CAS: 7664–93–9; Chemical Formula: H_2SO_4

H.S. No. 2147

In general industry, construction, and maritime, OSHA's permissible exposure limit for sulfuric acid is 1 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 1 mg/m³ and a TLV®–STEL of 3 mg/m³. NIOSH has a REL of 1 mg/m³ and concurs (Ex. 8–47) with the limit being proposed. OSHA is proposing to establish an 8-hour TWA limit of 1 mg/m³ for sulfuric acid in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Sulfuric acid is a dense, oily, colorless, odorless liquid. This substance is the most widely used of all industrial chemicals, finding use in the manufacture of fertilizers, chemicals, plastics, and explosives, in metal cleaning, petroleum refining, in the pickling of metal, and in electroplating (ACGIH 1986, p. 544(87); Proctor, Hughes, and Fischman 1988, p. 451).

Sulfuric acid is a severe irritant of the eyes, skin, mucous membranes, and respiratory tract in humans and animals; this substance also causes dental erosion. The oral LD_{50} in rats is 2140 mg/kg (RTECS 1990). In rats and mice, the 2-hour LC_{50}s are 510 mg/m³ and 320 mg/m³, respectively (RTECS 1990). Guinea pigs are more sensitive to sulfuric acid effects than rats, mice, or rabbits: exposure to a sulfuric acid concentration of 87 mg/m³ for 2.75 hours killed guinea pigs, but rats, mice, and rabbits survived exposure to 203 mg/m³ for 7 hours (Treon, Dutra, Cappel et al. 1950, in ACGIH 1986, p. 544(87)). Rabbits showed severe eye irritation after 5 mg of sulfuric acid was instilled into their eyes, followed after 30 seconds by rinsing (RTECS 1990). Guinea pigs exposed continuously for 24 hours/day tolerated sulfuric acid concentrations as high as 4 mg/m³ for as long as 140 days; at autopsy, signs of pulmonary pathology were seen (Thomas et al. 1958, in ACGIH 1986, p. 544(87)). Monkeys exposed continuously for 2 years to concentrations of sulfuric acid between 0.1 and 1 mg/m³ developed increasingly severe pulmonary lesions; exposure to a concentration of 2.5 mg/m³ caused impairment of pulmonary ventilation (Alarie et al. 1975, in Proctor, Hughes, and Fischman 1988, p. 453).

The lowest lethal oral dose in humans is estimated to be 135 mg/kg; the lowest reported toxic concentration is 3 mg/m³ for 24 weeks (RTECS 1989). In an experiment designed to determine thresholds for irritant effects, subjects reported throat tickling and scratching at a mean minimum concentration of 0.72 mg/m³, considerable irritation at the base of the esophagus and eye irritation at 1.1 to 2.1 mg/m³, and acute irritation of the eyes and mucous membranes and reflex coughing at concentrations between 2.4 and 6.0 mg/m³. Exposure to a sulfuric acid concentration of 1.0 to 1.1 mg/m³ caused slight changes in respiration, and increasing the concentration to 1.8 to 2.0 mg/m³ produced changes in respiratory amplitude and rhythm in all subjects (NIOSH Criteria Document 1974). A worker sprayed in the face with fuming sulfuric acid suffered skin burns and pulmonary edema (NIOSH Criteria 1974). When splashed in the eye, concentrated sulfuric acid causes severe damage and may cause blindness; a splash of dilute sulfuric acid produces transient effects (Grant 1986, pp. 866–868). Corrosion of the dental enamel has been seen in some sulfuric acid plant workers and in battery plant workers (Premysl 1952; Malcolm and Paul 1961, in ACGIH 1986, p. 544(87)). Exposure to an average concentration of 0.23 mg/m³ sulfuric acid caused a high incidence of teeth etching and erosion; these effects occurred after 4 months of exposure (Gamble et al. 1984, in HSDB 1990). Workers chronically exposed to sulfuric acid may develop signs and symptoms such as skin lesions, tracheobronchitis, stomatitis, conjunctivitis, and gastritis (Raule 1955, in ACGIH 1986, p. 544(87)). A total of 225 lead acid battery plant workers were studied using industrial hygiene measurements (personal samples), a symptom questionnaire, and spirometry. In acclimated workers, there was no evidence of acute symptoms or of declines in pulmonary function when the concentration of sulfuric acid was kept below 1 mg/m³ (Gamble et al. 1984, in Environ. Res. 35(1):11–29). A recent epidemiology study suggests that exposure to higher (not further specified) concentrations of sulfuric acid is associated with an excess risk of upper respiratory tract cancer, and specifically with laryngeal cancer (Soskoline et al. 1984, in Am. J. Epidemiol. 120(3):358–369).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to sulfuric acid causes severe eye, skin, and respiratory irritation, and corrosion of the dental enamel of the teeth. OSHA preliminary concludes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse exposure-related effects. The Agency believes that the proposed 8-hour TWA limit of 1 mg/m³ for sulfuric acid is necessary to substantially reduce the risks of these material health impairments. Extension of this limit to the agricultural sector will make OSHA's PEL for sulfuric acid consistent across all regulated sectors.

TETRAHYDROFURAN

CAS: 109–99–9; Chemical Formula: $(C_2H_4)_2O$

H.S. No. 1387

OSHA's PEL for tetrahydrofuran in construction and maritime workplaces is 200 ppm as an 8-hour TWA. There is no PEL in agriculture. The ACGIH has a 200 ppm TLV®–TWA and a 250 ppm TLV®–STEL for tetrahydrofuran. The Agency is retaining the 8-hour TWA PEL of 200 ppm in the construction and maritime industries and is proposing to extend it to agricultural workplaces; in addition, OSHA is proposing to add a 250-ppm STEL for tetrahydrofuran in the construction, maritime, and agriculture industries. NIOSH (Ex. 8–47) concurred that these limits were appropriate when OSHA recently established them in general industry.

Tetrahydrofuran is a colorless liquid with an odor like that of ether. This substance is used as a solvent for

natural and synthetic resins and in lithium aluminum hydride reduction and polymerization. It is also a chemical intermediate (ACGIH 1986, p. 564).

In addition to sensory irritation, tetrahydrofuran causes central nervous system depression in animals and humans. The oral LD_{50} in rats is 2816 mg/kg, and the LC_{50} in the same species is 21,000 ppm for 3 hours (RTECS 1990). Lehmann and Flury (1943c/Ex. 1–679) reported that animals exposed to concentrations of tetrahydrofuran exceeding 3000 ppm for 8 hours/day for 20 days showed signs of irritation of the upper respiratory tract as well as kidney and liver injury. Applied to the skin of rabbits, aqueous solutions of tetrahydrofuran that exceeded 20 percent caused irritation. One study (Stoughton and Robbins 1936/Ex. 1–597) found that tetrahydrofuran concentrations in excess of 25,000 ppm were needed to anesthetize dogs. The anesthesia process in these animals showed a delayed induction period and poor recovery. In other studies with dogs (Zapp 1971, as cited in ACGIH 1986/Ex. 1–3, p. 564), exposure to a 200-ppm concentration of tetrahydrofuran for 6 hours produced an observable effect on the pulse pressure of these animals within three to 4 weeks; however, despite exposure to this concentration for 9 weeks, followed by exposure for 3 weeks to nearly twice this concentration, no histopathologic changes were observed in the critical organs at autopsy. The work of Jochmann (1961/Ex. 1–1021), in which tetrahydrofuran was given orally and peritoneally to a variety of laboratory animals, showed both liver and kidney damage in these animals at autopsy; however, some of the effects observed by this author may have been caused by peroxide contamination of the tetrahydrofuran. Rats and mice were exposed for 13 weeks to concentrations of 66, 200, 600, 1800, or 5000 ppm. Rats exposed to 5000 ppm became ataxic, and mice exposed to 1800 or 5000 ppm became narcotic. At autopsy, minimal exposure-related effects were seen in the livers of both mice and rats in the high-dose group; however, morphologic changes were seen only in the high-dose mice (Chabra, Elwell, Chou, Miller, and Renne 1990, in Funda. Appl. Toxicol. 14(2):338–345).

In humans, exposure to tetrahydrofuran at a concentration of 25,000 ppm causes anesthesia (RTECS 1990). The symptoms of overexposure that have been reported in humans include headache, nausea, and dizziness, all indicative of central nervous system depression (AIHA 1959). The technicians involved in the experiment described above involving the anesthetization of dogs (Stoughton and Robbins 1936/Ex. 1–597) developed severe occipital headaches as a result of their exposure. Two plumbers exposed to unspecified concentrations of tetrahydrofuran when they were repairing pipes with glue containing this substance developed mucous membrane irritation, mild central nervous system effects, and toxic hepatitis, as evidenced by elevated liver enzymes. The enzymes returned to normal values within 14 days of the exposure (Garnier, Rosenberg, Puissant, Charvet, and Efthymion 1989).

Based on this evidence in humans and animals, OSHA preliminarily concludes that the proposed PELs of 200 ppm as an 8-hour TWA and 250 ppm as a 15-minute STEL are necessary to substantially reduce the significant risk of irritation and central nervous system depression associated with exposure to tetrahydrofuran. The Agency believes that these adverse health effects constitute material impairments of health. Promulgation of these limits for the construction, maritime, and agriculture industries will make OSHA's PELs for tetrahydrofuran consistent across all OSHA-regulated sectors.

TETRANITROMETHANE
CAS: 509–14–8
H.S. No. 2157

OSHA's PEL for tetranitromethane in general industry, construction, and maritime is 1 ppm as an 8-hour TWA; there is no limit in agriculture. The ACGIH TLV®–TWA for tetranitromethane is 1 ppm; there is no NIOSH REL for this substance. OSHA is proposing a TWA PEL of 1 ppm for tetranitromethane in agriculture. This is the limit recently established for this substance in general industry.

Tetranitromethane is a colorless to pale-yellow liquid with an acrid, biting odor. It is used as an oxidizing agent in rocket propellants, in explosives, as a diesel fuel additive, and as a laboratory reagent. It has been proposed for use as an irritant war gas (HSDB 1989; Clayton and Clayton 1981, p. 4157; ACGIH 1986, p. 567).

Tetranitromethane is a severe irritant of the eyes and respiratory tract in both animals and humans; at high concentrations, it causes methemoglobinemia and central nervous system damage. The oral LD_{50} in rats is 130 mg/kg; the 4-hour LC_{50} in the same species is 18 ppm (RTECS 1989). Rats exposed to 1230 ppm for 36 minutes exhibited lacrimation, rhinorrhea, gasping, and cyanosis before death; autopsy revealed pulmonary edema (Horn 1954). Cats exposed to a 0.1 to 0.4 ppm concentration of tetranitromethane showed signs of mild eye irritation, and cats exposed to a 10-ppm concentration of tetranitromethane for 20 minutes exhibited illness and died 10 days after exposure (AIHA 1964; Flury and Zernik 1931). A concentration of 33 ppm killed 65 percent of rats exposed for 6.5 hours, and 58 percent of rats exposed to a 6.35-ppm concentration for 6 hours/day, 5 days/week for 6 months died. Autopsy of the chronically exposed animals revealed lung damage (Horn 1954). Intravenous injection of tetranitromethane resulted in methemoglobinemia, anemia, central nervous system damage, and pulmonary edema in three species of experimental animals (Horn 1954).

In humans, the lowest lethal dose of tetranitromethane by inhalation is estimated to be approximately 500 mg/kg (or about 35 grams); the few deaths and poisonings that have occurred in workers handling heated trinitrotoluene (TNT) have been attributed to tetranitromethane contamination of the TNT (Clayton and Clayton 1982, p. 4157). Laboratory workers manufacturing tetranitromethane have reported experiencing irritation of the eyes, nose, and respiratory passages; dizziness; headache; chest pain; dyspnea; and, rarely, skin irritation (Horn 1954). In contact with the skin, tetranitromethane causes mild burns (AIHA 1964).

Based on this evidence in humans and animals, OSHA preliminarily concludes that agricultural workers exposed to this substance at the levels permitted by the absence of a permissible exposure limit are at significant risk of experiencing irritation of the eyes and respiratory tract. At high concentrations, they may also be at risk of death. The Agency believes that establishing a PEL of 1 ppm as an 8-hour TWA will protect workers in agriculture from these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

TETRASODIUM PYROPHOSPHATE
CAS: 7722–88–5; Chemical Formula: $Na_4P_2O_7$
H.S. No. 1389

OSHA has no limit for tetrasodium pyrophosphate in the construction, maritime, or agriculture industries. The ACGIH has an 8-hour TLV®–TWA of 5 mg/m^3; NIOSH has no REL for this substance but concurs that the proposed limit is appropriate (Ex. 8–47). OSHA is proposing an 8-hour TWA limit of 5 mg/m^3 for this substance in the

construction, maritime, and agriculture industries; this is the limit recently established for tetrasodium pyrophosphate in general industry.

Tetrasodium pyrophosphate is a white powder or a crystalline solid. This substance is used as a water softener, in soaps and detergents, as a dispersing and emulsifying agent, as a metal cleaner, and as a nutrition supplement. Tetrasodium pyrophosphate is also used in boiler water treatment, in drilling muds, dyeing, wood scouring, and as a sequestrant (ACGIH 1986, p. 567).

In addition to sensory irritation, tetrasodium pyrophosphate causes kidney damage in experimental animals exposed subchronically by oral administration. The oral LD_{50} in rats is 4000 mg/kg (RTECS 1990). Administered to rats in the diet for 4 to 6 months, all dietary levels exceeding 1 percent tetrasodium pyrophosphate caused kidney damage (HSDB 1989).

In humans, exposure to the alkaline dust of tetrasodium pyrophosphate is associated with irritation of the eyes and respiratory passages (Dow Chemical Co. 1977, in ACGIH 1986, p. 567). Contact of this substance with the skin of humans causes irritation (Hazardous Substance Fact Sheet 1986, p. 2). Ingestion of this substance can cause nausea, vomiting, and diarrhea (Gosselin, Smith, and Hodge 1984, p. II–121).

Based on this evidence, OSHA preliminarily concludes that exposure to this substance poses a significant risk of eye, skin, and respiratory tract irritation to workers in the construction, maritime, and agriculture industries. The Agency believes that these effects represent material impairments of health. Accordingly, OSHA is proposing a 5-mg/m^3 8-hour TWA limit for tetrasodium pyrophosphate in the construction, maritime, and agriculture industries; promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

THIOGLYCOLIC ACID

CAS: 68–11–1; Chemical Formula: $C_2H_4O_2S$

H.S. No. 1392

OSHA has no PEL for thioglycolic acid in the construction, maritime, or agriculture industries. The ACGIH has a TLV®–TWA of 1 ppm, with a skin notation. NIOSH has no REL for this substance. The Agency is proposing a 1 ppm TWA, and a skin notation, for thioglycolic acid in the construction, maritime, and agricultural industries. NIOSH [Ex. 8–47] concurred with this limit when OSHA recently established it in general industry.

Thioglycolic acid is a colorless, combustible liquid with the unpleasant odor characteristic of chemicals in the sulfhydryl group. Thioglycolic acid is used in the manufacture of pharmaceuticals, thioglycolates, permanent wave solutions, and depilatories and as a vinyl stabilizer (ACGIH 1986, p. 571).

In addition to sensory irritation, thioglycolic acid causes central nervous system effects in experimental animals. The oral LD_{50} in rats is 114 mg/kg. The lowest lethal concentration in mice is 7 mg/m^3 (1.9 ppm) for 2 hours (RTECS 1990). The lowest lethal concentration in rabbits by skin absorption is 300 mg/kg (RTECS 1990). The dermal LD_{50} for a 10 percent solution of thioglycolic acid is 848 mg/kg (ACGIH, 1986, p. 571/Ex. 1–3). A 5-ml/kg dermal dose of a 10-percent solution of thioglycolic acid caused weakness, gasping, and convulsions in guinea pigs. After receiving an oral dose of 125 mg/kg, female rats died; autopsy revealed liver effects and signs of gastrointestinal irritation (Dow Chemical Co. 1973b, as cited in ACGIH 1986, p. 571). Rabbits experienced conjunctival inflammation, dense corneal opacity, and iritis after thioglycolic acid was instilled into their eyes. The substance is also a severe skin irritant in animals (HSDB 1989).

In humans, contact of the skin or eyes with thioglycolic acid causes severe burns and blistering (Merck 1983, p. 1337). Hairdressers exposed to thioglycolates in permanent wave solutions have reported developing eczematous rashes on the face, scalp, and hands after contact with these materials (HSDB 1989). One accident has been reported in which a person was splashed in the eyes and on the face, legs, and arms with concentrated thioglycolic acid and subsequently experienced second-degree burns of the skin. The less injured eye returned to normal quickly, but the conjunctiva in the other eye became necrotic and the corneal became clouded. Most of this damage eventually cleared, and the victim was left with only a slight opacity in one eye (Grant 1986, p. 905).

Based on this evidence in humans and animals, OSHA preliminarily concludes that thioglycolic acid poses a significant risk of eye and skin burns and irritation, effects that constitute material impairments of health. OSHA believes that the proposed 1-ppm 8-hour TWA limit for this substance is necessary to reduce these risks for workers in the construction, maritime, and agricultural industries. In addition, because evidence in animals shows that thioglycolic acid solutions readily penetrate the skin, OSHA preliminarily finds that a skin notation is necessary to limit dermal contact. Promulgation of the proposed limit and the skin notation will make OSHA's PEL for this substance consistent across all OSHA-regulated sectors.

1,2,4–TRICHLOROBENZENE

CAS: 120–82–1; Chemical Formula: $C_6H_3Cl_3$

H.S. No. 1405

OSHA has no limit for 1,2,4-trichlorobenzene in the construction, maritime, or agriculture industry. The ACGIH has a TLV® ceiling limit of 5 ppm for 1,2,4-trichlorobenzene; NIOSH has no REL for this substance. OSHA is proposing a 5-ppm ceiling limit for this substance in the construction, maritime, and agricultural industries. NIOSH (Ex. 8–47, Table N1) concurred that the proposed limit was appropriate when the Agency recently established it in general industry.

1,2,4-Trichlorobenzene is a colorless, stable liquid at room temperature; it has an aromatic odor. 1,2,4-Trichlorobenzene is used as an insecticide, as a dielectric fluid, and as a heat transfer medium. It is also used in lubricants and in organic synthesis (ACGIH 1986, p. 593). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In addition to sensory irritation, 1,2,4-trichlorobenzene causes central nervous system effects and liver and kidney damage in experimental animals. The oral LD_{50} in rats is 756 mg/kg (RTECS 1990), and the acute percutaneous LD_{50} in rats is 6139 mg/kg (Brown, Muir, and Thorpe 1969/Ex. 1–537). Sublethal doses administered repeatedly to guinea pigs caused liver damage; acute and short-term exposure (fifteen 6-hour exposures to concentrations between 70 and 200 ppm), however, failed to kill these animals (Gage 1970/Ex. 1–318). The inhalation toxicity of 1,2,4-trichlorobenzene was studied by Treon (1950, as cited in ACGIH 1986/Ex. 1–3, p. 593), who determined that the target organs of exposure in cats, dogs, rats, rabbits, and guinea pigs included the liver, kidneys, ganglion cells at all brain levels, and mucous membranes. Irritation of the lungs and changes in respiration were seen in those animals that later died as a result of exposure. In a separate study conducted by Rowe (1975, as cited in ACGIH 1986/Ex. 1–3, p. 593), 20 male rats, 4 rabbits, and 2 dogs were exposed to 1,2,4-trichlorobenzene at concentrations of 30 or 100 ppm for 7 hours/day, 5 days/week for a total of 30

exposures in 44 days. No adverse exposure-related effects were detected in exposed animals in the 30-ppm group, with the exception of an elevation of urinary porphyrins in the rats on days 15 and 30 of exposure. A second inhalation study involved exposure to 1,2,4-trichlorobenzene 7 hours/day, 5 days/week for 26 consecutive weeks (Coate, Schoenfisch, Busey, and Lewis 1977, as cited in ACGIH 1986/Ex. 1–3, p. 593). Thirty rats, 16 rabbits, and 9 monkeys, all males, were exposed at concentrations of 25, 50, or 100 ppm. Microscopic changes were seen in the parenchymal cells of the livers and kidneys of all rats after weeks 4 and 13 of exposure, but no adverse effects were seen in animals of any other species. In a 90-day gavage study in rats given 1, 10, 100, or 1000 ppm 1,2,4-trichlorobenzene, males in the high-dose groups (100 or 1000 ppm) showed increased liver-to-body weight ratios; at autopsy, significant histopathological changes in the liver, kidney, and thyroid were seen in animals in the highest-dose group (Cote, Chu, Villeneuve, Secous, and Valli 1988, in Drug Chem. Toxicol. 11(1):11–28). 1,2,4-Trichlorobenzene has been shown to have embryotoxic effects in rats by oral administration and reproductive effects in female rats by intraperitoneal administration (RTECS 1990). Another recent study failed to show teratogenicity in the offspring of rats exposed to 150 or 300 ppm 1,2,4-trichlorobenzene on days 6 and 15 of gestation (Black, Valli, Ruddick, and Villeneuve 1988, in Bull. Environ. Contam. Toxicol. 41(5):719–726).

In workers, prolonged or repeated skin contact with 1,2,4-trichlorobenzene causes dermal irritation as a result of the defatting action of this chemical (Powers, Coate, and Lewis 1975/Ex. 1–658). In some cases, exposure to concentrations of 3 to 5 ppm caused eye and throat irritation (Rowe 1975, as cited in ACGIH 1986/Ex. 1–3, p. 593). This substance also has been reported to cause liver injury in humans (Gosselin, Smith, and Hodge 1984, p. II–173). One worker who was massively overexposed to trichlorobenzene experienced hemorrhages of the lungs (NRC 1977, p. 774). Exposure to trichlorobenzene has been reported to cause aplastic anemia, leukopenia, and thrombocytopenia in one non-occupational case, and a man whose job required him to fill drums with mono-, orthodi-, and trichlorobenzene developed anemia after 3 years of such exposure (Girard et al. 1969, in J. Med. Lyon 50:771–773).

Based on this evidence, the Agency preliminarily concludes that the PEL being proposed is necessary to protect workers in the construction, maritime, and agriculture sectors from the risk of eye, throat, and dermal irritation and possible liver damage associated with exposure to this substance. OSHA considers these exposure-related effects material impairments of health. To afford workers in these sectors protection from these effects, OSHA is proposing a ceiling limit of 5 ppm for 1,2,4-trichlorobenzene in the construction, maritime, and agriculture industries. Promulgation of this limit will make the PEL for 1,2,4-trichlorobenzene consistent across all OSHA-regulated sectors.

TRIETHYLAMINE

CAS: 121–44–8; Chemical Formula: $(C_2H_5)_3N$

H.S. No. 1408

OSHA has an 8-hour TWA limit of 25 ppm for triethylamine in the construction and maritime industries. There is no PEL in agriculture. The ACGIH has a 10 ppm TLV®–TWA and a 15 ppm TLV®–STEL for triethylamine. NIOSH has no REL for this substance. OSHA is proposing an 8-hour TWA limit of 10 ppm and a 15-minute STEL of 15 ppm for this substance in the construction, maritime, and agriculture industries. NIOSH (Ex. 8–47) concurred that these limits were appropriate when OSHA recently established them in general industry.

Triethylamine is a flammable, colorless liquid with a strong ammonia-like odor. Triethylamine is used in the preparation of quaternary ammonium compounds. It is also used as a catalytic solvent in chemical synthesis, as an accelerator activator for rubber, in the curing and hardening of polymers, and as a corrosion inhibitor and propellant (Hawley's 1987, p. 1180).

In addition to sensory irritation, triethylamine causes ocular effects in exposed workers. The oral LD_{50} in rats is 460 mg/kg, the LC_{50} in mice is 6 g/m^3 for 2 hours, and the dermal LD_{50} in rabbits is 750 mg/kg (RTECS 1990). Guinea pigs exposed for 30 minutes to a triethylamine concentration of 2000 ppm survived, but four of six animals died when exposed to this level for 2 hours; two of six guinea pigs died during a 4-hour exposure to a concentration of 1000 ppm, but all survived similar exposures at the 250- and 500-ppm levels (Carpenter, Smyth, and Shaffer 1948/Ex. 1–892). Applied to the skin of rabbits, triethylamine caused mild irritation; instilled into the eyes of rabbits, it caused severe irritation (RTECS 1990). Rabbits exposed repeatedly to a concentration of 50 ppm exhibited marked irritation of the cornea and of pulmonary tissue (Brieger and Hodes 1951/Ex. 1–408; Carpenter and Smyth 1946/Ex. 1–859).

In humans, the effects of repeated triethylamine exposure correspond to those of ethylamine and diethylamine and include irritation of the eyes and mucous membranes and corneal edema and other ocular effects (Brieger and Hodes 1951/Ex. 1–408). Humans exposed to a 12-mg/m^3 concentration of triethylamine experienced changes in their visual fields (RTECS 1989). Triethylamine was also found to inhibit monoamine oxidase activity, resulting in central nervous system stimulation (De Bruin 1976/Ex. 1–895).

Based on this evidence in humans and animals, OSHA preliminarily concludes that workers exposed to triethylamine are at significant risk of experiencing sensory irritation and ocular effects, both material impairments of health. Therefore, OSHA is proposing limits for triethylamine of 10 ppm as an 8-hour TWA and 15 ppm as a 15-minute STEL for workplaces in the construction, maritime, and agriculture industries. Promulgation of these PELs will make OSHA's limits for triethylamine consistent across all OSHA-regulated sectors.

TURPENTINE

CAS: 8006–64–2; Chemical Formula: $C_{10}H_8$

H.S. No. 2166

In general industry, construction, and maritime, OSHA's permissible exposure limit for turpentine is 100 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 100 ppm for this substance; NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL in agriculture of 100 ppm for turpentine. This is the limit recently established for this substance in general industry.

Turpentine is a volatile mixture of hydrocarbon isomers obtained either from pine gum or pine wood. Gum turpentine is a yellowish, sticky, opaque, combustible material, and the wood distillate (oil of turpentine) is a flammable, colorless liquid with a characteristic odor (Sittig 1985, p. 906; Genium MSDS 1984, No. 375; Hawley's 1987, p. 1200). Turpentine is used as an insecticide, a chemical intermediate, and a flavoring agent. It is also used in the manufacture of cleaning materials, putty, cutting and grinding fluids, and mastics, and as an ingredient in some perfumes, deodorizers, pharmaceuticals, polishes, oils, inks, and stimulating ointments (HSDB 1989). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal

Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Turpentine is an irritant of the eyes, skin, and respiratory tract and causes central nervous system depression in both animals and humans; in humans, exposure also may cause dermal sensitization and gastrointestinal and urinary tract effects. Injection of turpentine into rabbits' eyes produced corneal opacification (Grant 1986, p. 961). The oral LD_{50} in rats is 5760 mg/kg, and the inhalation LC_{50} for the same species is 12 g/m³ for 6 hours (RTECS 1989). Cats exposed to a concentration of turpentine of between 540 and 720 ppm exhibited signs of eye and respiratory tract irritation and had mild convulsions; at 1440 ppm, they developed paralysis within 150 to 180 minutes (HSDB 1989). Dogs exposed to an 818-ppm concentration of turpentine for 3.5 to 4.5 hours developed nausea, incoordination, mild paralysis, and weakness (Grant 1986, p. 961; Clayton and Clayton 1981, p. 3245); however, no effects were seen in dogs exposed to a 180-ppm concentration for 3.5 hours/day for 8 days (Lehmann and Flury 1943). Turpentine has also caused skin tumors in mice and rats (RTECS 1989; Clayton and Clayton 1981, p. 3245).

The lowest reported oral dose of turpentine that is lethal in humans is 441 mg/kg (RTECS 1989). Exposure to a 75-ppm concentration for 3 to 5 minutes irritates the nose and throat, and exposure to 175 ppm irritates the eyes and is considered intolerable (Grant 1986, p. 961; Nelson 1943). Exposure to a turpentine concentration of between 720 and 1100 ppm causes headache, dizziness, nausea, chest pain, and visual disturbances in exposed individuals (Clayton and Clayton 1981, p. 3235). Ingestion of turpentine causes burning pain in the mouth and throat, nausea, vomiting, diarrhea, abdominal pain, excitement, ataxia, confusion, stupor, seizures, fever, and tachycardia and may cause respiratory failure and death (Gosselin, Smith, and Hodge 1984, p. III–394). Repeated or prolonged skin contact causes skin sensitization, with bullous dermatitis and eczema (Clayton and Clayton 1981, p. 3244; Sittig 1985, p. 907). A case-control study of workers demonstrated a statistically significant association between chronic exposure (greater than 5 years) to terpenes (a principal component of turpentine) and the development of respiratory tract cancers (HSDB 1989).

Based on this evidence, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing the eye, skin, mucous membrane, and respiratory tract irritation and central nervous system depression associated with exposure to turpentine. The Agency believes these effects are material impairments of health and that establishing an 8-hour TWA PEL of 100 ppm in agriculture is necessary to substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

VANADIUM PENTOXIDE DUST, RESPIRABLE

CAS: 1314–62–1; Chemical Formula: V_2O_5

H.S. No. 1421

The OSHA PEL for vanadium pentoxide (V_2O_5) dust in the construction and maritime industries is a ceiling of 0.5 mg/m³. There is no PEL in agriculture. The ACGIH has an 8-hour TLV®–TWA of 0.05 mg/m³ for vanadium pentoxide dust, and NIOSH has a 15-minute ceiling of 0.05 mg/m³ for this substance. The Agency is proposing a 0.05 mg/m³ 8-hour TWA limit for vanadium pentoxide dust in the construction, maritime, and agriculture industries. This is the limit recently established for this dust in general industry.

Vanadium pentoxide is a yellow to rust-brown crystalline compound; the powdered material is used as a catalyst in the preparation of vanadium alloys and compounds, as an oxidation catalyst, as a component of ferrovanadium steels, and in the manufacture of pigments and glazes. Vanadium pentoxide dust is also used as a catalyst in the textile industry, in the manufacture of UV filter glass, in photographic developers, and in the cleaning and maintenance of furnaces (NIOSH/OSHA Occupational Health Guideline 1981, p. 3).

In addition to sensory irritation, exposure to vanadium dust causes central nervous system effects in experimental animals. Because the literature often fails to distinguish between the dust and the fume, much of the following material may apply to one or both forms. Exposure to a concentration of 205 mg/m³ for 7 hours was lethal to rabbits; animals exhibited signs of pulmonary edema before death (HSDB 1985). The oral LD_{50} in rats is 10 mg/kg, and the lowest lethal concentration in the same species is 70 mg/m³ for 2 hours (RTECS 1990). Animals acutely poisoned by oral administration developed marked diarrhea, an exudate from the nose, hind limb paralysis, difficult breathing, and convulsions (Gosselin, Smith, and Hodge 1984, p. II–148). Dogs, rats, guinea pigs, and rabbits exposed to V_2O_5 (at a V/m³ concentration of 0.5 mg/m³) 6 hours/day for 6 months showed no exposure-related histopathological effects at autopsy (Clayton and Clayton 1981, p. 2021). Another study that exposed rabbits to unspecified concentrations of vanadium pentoxide dust for 8 months showed no fibrotic changes in the lungs of these animals.

Several studies indicate that OSHA's current exposure limit is not sufficient to protect workers in construction and maritime against vanadium pentoxide dust's respiratory effects, which include bronchitis, emphysema, tracheitis, pulmonary edema, and bronchial pneumonia. Seven cases of upper respiratory tract irritation were reported in boiler cleaners exposed to vanadium pentoxide concentrations ranging from 2 to 85 mg/m³ (Sjöberg 1951/Ex. 1–437). Williams (1952/Ex. 1–456) reported eight cases of vanadium poisoning in workers cleaning boilers in an atmosphere ranging from 30 to 104 mg/m³. Gul'ko (1956, as cited by Hudson 1964/Ex. 1–880) observed eye and bronchial irritation in workers exposed to concentrations between 0.5 and 2.2 mg/m³. A study by Lewis (1959/Ex. 1–345) indicated that workers exposed to vanadium pentoxide levels of 0.2 to 0.5 mg/m³ experienced a higher incidence of respiratory symptoms than did controls. Tebrock and Machle (1968/Ex. 1–446) reported that workers exposed to average concentrations of 1.5 mg/m³ vanadium pentoxide in a mixed dust developed conjunctivitis, tracheobronchitis, and dermatitis. A single 8-hour exposure to an average concentration of 0.2 mg/m³ (respirable vanadium pentoxide dust) caused severe upper respiratory tract irritation in five human volunteers, and two other subjects exposed to a 0.1-mg/m³ concentration also developed delayed cough and an increase in mucus production (Zenz and Berg 1967/Ex. 1–405). Frequent cases of asthma have been reported among workers exposed to vanadium pentoxide dust, which suggests that asthma may be a result of chronic exposure (NIOSH Criteria 1977). A recent study (Huang 1989) reports that 76 Chinese workers exposed to vanadium pentoxide fume at a concentration of 1.2 to 18 mg/m³ and to the dust at concentrations ranging from 1.27 to 27 mg/m³ had lung markings on their chest roentgenograms and reported the following signs and symptoms: Cough (63 workers), cough and expectoration (53 workers), difficult breathing (27 cases), wheezing and rales (31 cases). Fifteen of these workers

showed evidence of pulmonary fibrosis on their roentgenograms.

In the construction, maritime, and agriculture industries, OSHA is proposing a limit of 0.05 mg/m³ as an 8-hour TWA for respirable vanadium dust, measured as vanadium pentoxide. The Agency preliminarily concludes that this limit will prevent or substantially reduce the significant risks of eye and bronchial irritation, respiratory symptoms, conjunctivitis, and coughing seen in workers exposed to this substance at concentrations ranging from 0.1 to 2.2 mg/m³. OSHA considers these exposure-related effects material impairments of health. Promulgation of the new PEL will also make OSHA's limit for this substance consistent across all OSHA-regulated sectors.

VANADIUM PENTOXIDE FUME

CAS: 1314–62–1; Chemical Formula: V_2O_5

H.S. No. 1422

OSHA's PEL for vanadium pentoxide (V_2O_5) fume in the construction and maritime industries is 0.1 mg/m³ as a ceiling limit. There is no PEL in agriculture. The ACGIH has an 8-hour TLV®–TWA of 0.05 mg/m³ for vanadium pentoxide fume, and NIOSH has a 0.05 mg/m³ 15-minute ceiling for this substance. OSHA is proposing an 8-hour TWA of 0.05 mg/m³ for vanadium pentoxide fume in the construction, maritime, and agriculture industries. This is the limit recently established for the fume in general industry.

Vanadium pentoxide is a yellow to rust-brown noncombustible crystalline compound; its fume appears as finely divided particulate dispersed in air. Vanadium pentoxide fume may be generated when vanadium pentoxide is used in the production of pellets from electric furnaces, in metallurgical processes in furnaces, in the manufacture of semiconductors fused with sodium oxide, in welding, and in the fabrication of alloys for use as an additive in special steels (NIOSH/OSHA Occupational Health Guideline 1981, p. 3).

In addition to sensory irritation, exposure to vanadium fume causes central nervous system effects in humans and experimental animals. In humans, overexposure to the fume causes metal fume fever. Because the toxicology literature often fails to distinguish between the dust and the fume, much of the following material may apply to one or both forms. There are no acute toxicity data specifically for the fume.

Vanadium pentoxide fume's chief toxic effects in humans are manifested in the respiratory passages: bronchitis, emphysema, tracheitis, pulmonary edema, and bronchial pneumonia can result from exposure. Seven cases of upper respiratory tract irritation were reported in boiler cleaners exposed to vanadium pentoxide concentrations ranging from 2 to 85 mg/m³ (Sjôberg 1951/Ex. 1–437). Williams (1952/Ex. 1–456) reported eight cases of vanadium poisoning in workers cleaning boilers in an atmosphere ranging from 30 to 104 mg/m³. Gul'ko (1956, as cited by Hudson 1964/Ex. 1–880) observed eye and bronchial irritation in workers exposed to concentrations of 0.5 to 2.2 mg/m³. A study by Lewis (1959/Ex. 1–345) indicated that workers exposed to concentrations of 0.2 to 0.5 mg/m³ experienced a higher incidence of respiratory symptoms than did controls. Tebrock and Machle (1968/Ex. 1–446) reported that workers exposed to average concentrations of 1.5 mg/m³ vanadium pentoxide in a mixed dust/fume atmosphere developed conjunctivitis, tracheobronchitis, and dermatitis. A single 8-hour exposure to an average concentration of 0.2 mg/m³ (respirable vanadium dust) caused severe upper respiratory tract irritation in five human volunteers, and two other subjects exposed to a 0.1-mg/m³ concentration also developed delayed cough and an increase in mucus production (Zenz and Berg 1967/Ex. 1–405). Frequent cases of asthma have been reported among workers exposed to vanadium pentoxide, which suggests that asthma may be a result of chronic exposure (NIOSH Criteria 1977). A recent study (Huang 1989) reports that 76 Chinese workers exposed to vanadium pentoxide fume at a concentration of 1.2 to 18 mg/m³ and to the dust at concentrations ranging from 1.27 to 27 mg/m³ had lung markings on their chest roentgenograms and reported the following signs and symptoms: cough (63 workers), cough and expectoration (53 workers), difficult breathing (27 cases), wheezing and rales (31 cases). Fifteen of these workers showed evidence of pulmonary fibrosis on their roentgenograms.

Based on this evidence in humans and animals, the Agency preliminarily concludes that the proposed TWA limit is necessary to protect workers in construction, maritime, and agriculture from the significant risks of eye, skin, and upper respiratory tract irritation; conjunctivitis; pulmonary damage; and systemic poisoning associated with exposure to vanadium pentoxide fume. The Agency considers these exposure-related effects to be material impairments of health. Accordingly, OSHA is proposing a PEL of 0.05 mg/m³ as an 8-hour TWA for this substance in these sectors. Promulgation of this limit will also make the PEL for the fume consistent across all OSHA-regulated sectors.

VINYL ACETATE

CAS: 108–05–4; Chemical Formula: $CH_3COOCH = CH_2$

H.S. No. 1424

There is no OSHA limit for vinyl acetate in the construction, maritime, or agriculture industries. The ACGIH has an 8-hour TLV®–TWA limit of 10 ppm and a 20 ppm TLV®–STEL for vinyl acetate. NIOSH has a 15-minute ceiling limit of 4 ppm for this substance. OSHA is proposing to establish a 10-ppm 8-hour TWA and a 20-ppm 15-minute STEL for this substance in construction, maritime, and agriculture. These are the limits recently established for this substance in general industry.

Vinyl acetate is a volatile liquid that polymerizes in light to a colorless, transparent mass; in industry, this substance usually contains an inhibitor, such as hydroquinone. Vinyl acetate is used for the manufacture of polyvinyl resins and in latex paints, paper coatings, adhesives, textile finishes, and safety glass interlayers (Hawley's 1987, p. 1222).

Vinyl acetate causes irritation of the eyes, nose, and throat. The oral LD_{50} in rats is 2920 mg/kg, the LC_{50} in rabbits is 2500 ppm for 4 hours, and the dermal LD_{50} in rabbits is 2335 mg/kg (RTECS 1990). Instilled into rabbit eyes, vinyl acetate caused mild irritation; in contact with the skin, this substance also caused irritation (RTECS 1990). Some beagle dogs exposed to a 240-ppm concentration of vinyl acetate for 4 hours showed signs of eye irritation (Mellon Institute 1968). Rats exposed repeatedly to a vinyl acetate concentration of 100 ppm showed no adverse effects (Proctor, Hughes, and Fischman 1988, p. 504). Rats and mice inhaling 13.2 or 68 mg/m³ vinyl acetate continuously for 4 months showed a dose-related pulmonary response; findings included emphysema and lung atelectases, as well as effects on the central nervous system, adrenal glands, and hypophysis (HSDB 1989). In mice, vinyl acetate caused a dose-dependent decrease in sperm production and a reduction in testicular weight when the animals were exposed to this substance at a dose of 125 mg/kg/day (Lahdetie 1988). In one oral carcinogenicity bioassay, rats given 100 g/kg vinyl acetate for 2 years developed uterine and thyroid tumors (RTECS 1990); however, another cancer bioassay (Maltoni 1976) was negative (cited in

Proctor, Hughes, and Fischman 1988, p. 505).

The basis for the proposed limits is an epidemiologic report by Deese and Joyner (1969/Ex. 1–412) describing 15 years of industrial experience with vinyl acetate production. These authors reported that vinyl acetate is not a significant irritant at exposure concentrations of 5 to 10 ppm but that it causes cough and hoarseness around 22 ppm. They also found no evidence of adverse chronic effects resulting from exposure to concentrations of 5 to 10 ppm, as determined from medical records and examinations of workers exposed to these levels. While conducting air sampling for the study, the primary author (Deese) experienced hoarseness at vinyl chloride concentrations of 4.2 to 5.7 ppm, and eye irritation at 5.7 to 6.8 ppm. Three chemical operators and one technician involved in this study did not report any subjective responses at these levels. Severe skin irritation or blistering may occur in workers whose skin is in contact with vinyl acetate (NIOSH Criteria Document 1978). One worker whose eye was burned by a splash of vinyl acetate recovered fully within 48 hours (Grant 1986, p. 978). Changes in pulmonary function were observed in persons employed in the production of vinyl acetate (HSDB 1987).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to this substance causes eye, throat, and skin irritation and possible changes in pulmonary function. The Agency considers these effects material impairments of health and believes that the proposed PELs will substantially reduce the risk that workers in construction, maritime, and agriculture will experience these effects. Promulgation of these limits will also make the PELs for vinyl acetate consistent across all OSHA-regulated sectors.

VINYL TOLUENE

CAS: 25013–15–4; Chemical Formula: $CH_3C_6H_4CH=CH_2$

H.S. No. 1427

OSHA's PEL for vinyl toluene in construction and maritime is an 8-hour TWA of 100 ppm. There is no PEL in agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. The ACGIH has a TLV®–TWA of 50 ppm and a 100-ppm TLV®–STEL for vinyl toluene. OSHA is proposing an 8-hour TWA PEL for this substance of 100 ppm in agriculture. This is the limit recently established for this substance in general industry.

Vinyl toluene is a colorless liquid with a strong, disagreeable odor that finds use as a solvent, in resin production, in the plastics industry, and as a component of insecticides (ACGIH 1986, p. 630; Clayton and Clayton 1981, p. 3319). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Wolf, Rowe, McCollister et al. (1956/Ex. 1–404) noted fatty degeneration of the liver and an increase in kidney and liver weights in rats, guinea pigs, rabbits, and monkeys subjected to approximately 100 7- to 8-hour exposures to vinyl toluene at a concentration of 1250 ppm. Some deaths occurred among the rats in this group. Animals exposed to vinyl toluene at a 600-ppm concentration appeared normal and showed no blood or urine abnormalities, no gross or microscopic tissue changes, and no changes in growth rate or organ weight (Wolf, Rowe, McCollister et al. 1956/Ex. 1–404). When vinyl toluene was dropped into the eyes of rabbits, it caused conjunctival irritation; in contact with rabbit skin, it caused reversible necrosis and edema (Proctor, Hughes, and Fischman 1988, p. 507). By intraperitoneal administration, vinyl toluene is fetotoxic in rats (RTECS 1989).

Human volunteers reported experiencing eye and nose irritation at a 400-ppm concentration and objectionable odor at a concentration of 300 ppm. At 50 ppm, the odor of vinyl toluene was detectable by volunteers, but these individuals experienced no irritation and did not find the odor intolerable (ACGIH 1986/Ex. 1–3, p. 630).

OSHA is proposing an 8-hour TWA limit of 100 ppm in agriculture. The Agency preliminarily concludes that the proposed limit is necessary to protect workers in this sector against the significant risk of intolerable odor and irritation caused by vinyl toluene exposures in the workplace. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

VM & P NAPHTHA

CAS No. 8032–32–4; Chemical Formula: none

H.S. No. 1429

OSHA has no PEL for VM & P (Varnish Makers' and Printers') naphtha in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 300 ppm for VM & P naphtha, and NIOSH has no REL for this substance. OSHA is proposing a 300-ppm 8-hour TWA and a 400-ppm STEL for VM & P naphtha in the construction, maritime, and agriculture sectors. NIOSH concurred (Ex. 8–47) that these limits were appropriate when the Agency recently established them in general industry.

VM & P naphtha, also known as ligroin, is a colorless, flammable liquid that has an aromatic odor. VM & P naphtha is used as a diluent for paints, coatings, resins, printing inks, and rubber cement and as a solvent (ACGIH 1986, p. 631).

In addition to sensory irritation, VM & P naphtha causes central nervous system depression in humans and animals. The LC_{50} in rats is 3400 ppm for 4 hours (RTECS 1990). Rats lost coordination and convulsed after being exposed for 15 minutes at room temperature to a saturation concentration of this substance (Carpenter 1975). Temporary hematologic effects were observed in rats and dogs exposed to a concentration of 1200 ppm for 40 days (Clayton and Clayton 1981, p. 3389). A study in which rats and beagles received inhalation doses of 500 ppm VM & P naphtha for 30 hours per week for 13 weeks resulted in no chronic or latent effects (Carpenter, Kinkead, Geary et al. 1975a/Ex. 1–302). These authors also noted that the acute toxicity of VM & P naphtha for rats and other species was four times greater than that of rubber solvent naphtha, which has a limit of 400 ppm.

Seven human volunteers exposed to a VM & P naphtha concentration of 880 ppm for 15 minutes reported upper respiratory tract, eye, and nose irritation, in addition to olfactory fatigue (ACGIH 1986/Ex. 1–3, p. 631). Elkins (1959d, as cited in ACGIH 1986/Ex. 1–3, p. 631) noted one case of a worker, exposed to levels of VM & P naphtha averaging 800 ppm, who developed unspecified chronic effects. Elkins also reported that the VM & P naphtha level producing significant irritation in human volunteers was about half as great for this form of naphtha as for rubber solvent naphtha. An accidental exposure of 19 workers to an unspecified concentration of this substance caused dyspnea; two of these workers developed cyanosis and tremor (Wilson 1976).

The Agency preliminarily concludes that a 300-ppm 8-hour TWA PEL and a 400-ppm 15-minute STEL are necessary to protect workers in construction, maritime, and agriculture from irritation and central nervous system effects associated with naphtha exposure. OSHA considers these effects material impairments of health and is therefore proposing both a 300-ppm 8-hour TWA and a 400-ppm 15-minute STEL for VM &

P naphtha in the construction, maritime, and agriculture sectors. Promulgation of these limits will make the PELs for VM & P naphtha consistent across all OSHA-regulated sectors.

XYLENES, (o-, m-, and p-ISOMERS)
CAS: 1330–20–7; Chemical Formula: $C_6H_4(CH_3)_2$
H.S. No. 1431

The OSHA limit for the xylenes is 100 ppm as an 8-hour TWA in the construction and maritime industries. There is no PEL in agriculture. The ACGIH has an 8-hour TLV®–TWA of 100 ppm and a TLV®–STEL of 150 ppm for xylene. NIOSH has a recommended exposure limit of 100 ppm as a TWA and a 10-minute ceiling limit of 200 ppm for this substance. OSHA is proposing a 100 ppm TWA and 150 ppm STEL for xylene in the construction, maritime, and agriculture industries. NIOSH (Ex. 8–47) concurred with these limits when they were established recently in general industry.

The xylene isomers are clear, flammable liquids that have an aromatic hydrocarbon odor. Xylene is present in gasoline and in many petroleum solvents. Xylene is used to make insecticides, drugs, and dyes, as a solvent in paints and other coatings (especially alkyl resins), and in rubber cements (ACGIH 1986, p. 637).

In addition to sensory irritation, xylene causes narcosis in humans and animals at high concentrations. The 4-hour LC_{50} in rats is 5000 ppm and the oral LD_{50} in the same species is 4300 mg/kg (RTECS 1990). In contact with the skin of rabbits, 500 mg caused moderate skin irritation; instilled into rabbit eyes, this substance caused mild eye irritation. Rats exposed to a concentration of 1600 ppm for 2 or 4 days showed signs of mucous membrane irritation, became incoordinated, lost weight, and had an increased erythrocyte count (NIOSH Criteria Document 1975). Rats were exposed orally to p-xylene at 125, 250, 500, 1000, or 2000 mg/kg or to 800 or 1600 ppm xylene for 4 hours. Rats exposed to doses above 250 mg/kg or to 1600 ppm showed a significant depression in flash evoked potentials (FEPs) (FEP is an index of the functional integrity of the visual system) (Dyer, Bercegeay, and Mayo 1988, Neurotoxicol. Teratol. 10(2):147–153). Leukopenia, kidney congestion, and hyperplasia of the bone and spleen were seen in rats exposed to 980 ppm for 7 days. Some mice died after being exposed to 2010 ppm m-xylene or o-xylene for 24 hours; however, those exposed to 4912 ppm p-xylene survived (NIOSH Criteria Document 1975). Rats and rabbits exposed to a mixture of xylene isomers at a concentration of 690 ppm for eight hours daily, six days per week showed no blood abnormalities, but rabbits exposed on the same regimen at 1150 ppm for 55 days showed a decrease in red and white blood cell counts and an increase in platelet count (Fabre and Truhaut 1954, as cited in ACGIH 1986/Ex. 1–3, p. 637). Sprague-Dawley rats were given 250, 1000, or 2000 mg/kg m-xylene, o-xylene, or p-xylene by gavage for 10 days, or 150, 750, or 1000 mg/kg mixed xylene isomers by gavage for 90 days. Males in the highest dose group of the 10-day study had significantly reduced body weights and significantly increased relative liver weights (all isomers). In the 90-day study, rats of both sexes had dose-related increases in relative liver and kidney weights (Condie, Hill, and Borzelleca 1988, Drug Chem. Toxicol. 11(4):329–354). Teratogenicity studies in rats, hamsters, rabbits, and mice administered xylene by two routes of exposure (inhalation and oral) have shown that this substance causes developmental abnormalities and is fetotoxic (RTECS 1990; HSDB 1989).

Studies of workers exposed to xylene revealed headache, fatigue, lassitude, irritability, and gastrointestinal disturbances as the most common symptoms (Gerarde 1960d/Ex. 1–738a). At unspecified exposure levels, Browning (1965b/Ex. 1–1016) also noted gastrointestinal disturbances, in addition to kidney, heart, liver, and neurological damage; blood dyscrasias, some of which resulted in death, were also reported in these workers. A study by Nelson, Enge, Ross, et al. (1943/Ex. 1–66), in which human volunteers were exposed to 200 ppm xylene, found eye, nose, and throat irritation in the subjects at this level of exposure. Volunteers exposed for 3 hours 40 minutes to m-xylene either at 200 ppm or at 135 ppm combined with two 20-minute 400 ppm peak exposures per day showed changes in their visual evoked potentials during exercise; these changes suggest that exposure to m-xylene activated these subjects' arousal levels (Sappalainen, Laine, Salmi, Riihimaki, and Verkkala 1989, in Int. Arch. Occup. Environ. Health 61(7):443–449). Morley, Eccleston, Douglas, and colleagues (1970/Ex. 1–794) observed liver dysfunction and renal impairment in three workers overexposed to xylene (estimated concentration of 10,000 ppm). One of these workers died, but the others recovered slowly. Furniture polishers were reported by Matthaus (1964/Ex. 1–830) to have suffered corneal damage as a result of exposure to xylene at unknown concentrations. A workman was exposed to nearly pure xylene for 3 years during boat hull repair (estimated exposure = several hundred ppm). He developed the following signs and symptoms of acute organic brain syndrome: dysphasia, fine tremor, agitation, breathlessness, fatigue, impaired concentration and short-term memory, and confusion, hyperreflexia, and unstable gait. The worker recovered over the next 2 years after exposure was terminated (Roberts, Lucas, Marsden, and Traner 1988, in Lancet II (8605):273).

Based on this evidence, OSHA preliminarily concludes that both a TWA and a STEL are necessary to prevent the risks of narcosis, blood effects, and irritant effects among workers exposed to xylene in the construction, maritime, and agriculture industries. The Agency considers these exposure-related effects to be material impairments of health. Therefore, to reduce these occupational risks among workers exposed to xylene, OSHA is proposing a 100-ppm TWA and a 150-ppm STEL for this substance in the construction, maritime, and agricultural industries. Promulgation of these limits will make OSHA's PELs for xylene consistent across all OSHA-regulated sectors.

ZINC CHLORIDE (FUME)
CAS: 7646–85–7; Chemical Formula: $ZnCl_2$
H.S. No. 1435

OSHA's limit for zinc chloride fume in the construction and maritime industries is 1 mg/m³ as an 8-hour TWA. There is no PEL in agriculture. The ACGIH has an 8-hour TLV®–TWA of 1 mg/m³ and a TLV®–STEL of 2 mg/m³ for zinc chloride fume. NIOSH has no REL for this substance. OSHA is proposing an 8-hour TWA of 1 mg/m³ and a STEL of 2 mg/m³ for zinc chloride fume. NIOSH (Ex. 8–47) concurred that these limits were appropriate when OSHA recently established them in general industry.

Zinc chloride fume is white and has an acrid odor. Zinc chloride is used in soldering fluxes, to galvanize iron, as a wood preservative, in the textile industry, as an ingredient in adhesives, dentifrices, deodorants, and embalming fluids, and in organic synthesis and petroleum refining. It is also the main ingredient of some screening smokes (ACGIH 1986, p. 643).

Zinc chloride fume causes sensory irritation in humans and animals at high concentrations. The oral LD_{50} in rats is 350 mg/kg, and the lowest lethal concentration in the same species is 1960 mg/m³ for 10 minutes (RTECS 1990).

In humans, zinc chloride fume is highly caustic and damages the mucous membranes of the nasopharynx and respiratory tract. Exposure to the fumes of zinc chloride may result in a severe pneumonitis that is caused by irritation of the respiratory tract (Gafafer 1964/Ex. 1–1149). There are two reports of solderers who developed occupational asthma (confirmed by positive bronchial challenge) after soldering with soft corrosive soldering fluxes that contained both zinc chloride and ammonium chloride (Weir, Robertson, Jones, and Burge 1989, in Thorax 44(3):220–223). One instance in which a worker inhaled zinc chloride fumes resulted in advanced pulmonary fibrosis that ended in death (Milliken, Waugh, and Kadish 1963/Ex. 1–751), and 10 deaths and 25 nonfatal cases of pneumonitis occurred in workers caught in a tunnel when 79 smoke generators caught fire and generated zinc chloride fumes (Hunter 1955/Ex. 1–853). A firefighter exposed to a high concentration of zinc chloride fume experienced nausea, sore throat, and chest tightness. After some improvement in health, this worker developed tachypnea, substernal soreness, fever, cyanosis, and coma prior to death 18 days after initial exposure (Melliken, Waugh, and Kadish 1963). A recent NIOSH study (Zey and Richardson 1985) reports that the zinc chloride fumes generated by smoke-generating devices reach concentrations of between 11 to 498 mg/m^3 and that firefighters have experienced severe adverse effects, including death, from such exposures (in NIOSH HETA 85–274–1979, 1985). Five Danish military workers were acutely overexposed to zinc chloride smoke during military training; two of these men breathed zinc chloride at an unspecified concentration for 1 to 2 minutes and subsequently developed severe adult respiratory distress syndrome (ARDS). Both patients died; autopsy revealed microvascular obliteration, widespread occlusion of the pulmonary arteries, and extensive interstitial and intra-alveolar fibrosis (Hjortso, Qvist, Bud, Thomsen, Andersen, et al. 1988, in Inten. Care Med. 14(1):17–24). Other studies have shown that long-term zinc chloride exposures cause skin ulceration (Sax 1957/Ex. 1–1095). It has also been suggested that zinc chloride exposure may have chronic effects (Hamilton and Hardy 1974b/Ex. 1–958). In an investigation of the adverse effects of zinc chloride fume exposures, Ferry (1966, as cited in ACGIH 1986/Ex. 1–3, p. 643) reported that no sensory effects occurred when 30-minute exposures were limited to 0.07 and 0.4 mg/m^3; however, this researcher noted that these levels did corrode metal.

Based on this evidence, OSHA preliminarily concludes that exposure to this substance is associated with a significant risk of damage to the eyes, skin, and respiratory tract and of pneumonitis, all of which are considered by OSHA to be material impairments of health. The Agency believes that the proposed PELs will substantially reduce these risks. Accordingly, OSHA is proposing a 1 mg/m^3 TWA limit and 2 mg/m^3 STEL for this substance in the construction, maritime, and agriculture industries. Promulgation of these limits will make the PELs for zinc chloride fume consistent across all OSHA-regulated sectors.

Preliminary Conclusions for the Group of Sensory Irritants

OSHA preliminarily finds that sensory irritation poses an occupational health risk to workers in the construction, maritime, and agricultural sectors who are exposed to these substances in their places of work. Among the adverse health consequences of exposure to sensory irritants are acute breathing difficulty, lacrimation, conjunctivitis, sensitization, persistent coughing, and upper respiratory tract irritation. OSHA has preliminarily determined that these effects constitute material impairments of health and functional capacity within the meaning of the Act. In addition to the pain and suffering associated with these signs and symptoms, workers experiencing irritant effects find it difficult if not impossible to concentrate on the job at hand; they therefore work less safely and less productively than nonexposed employees. Reducing exposures from levels that have been associated with these effects to levels where such consequences are substantially less likely to occur will substantially reduce the significant risk posed to workers at current levels. Furthermore, many of the substances in this group have been demonstrated to have adverse effects on other organ systems, including the cornea, lungs, kidney, liver, central nervous system, and gastrointestinal tract. In addition, promulgation of the proposed limits in construction, maritime, and agriculture will make the Agency's limits for these contaminants consistent across all OSHA-regulated industries.

4. Substances for Which Proposed Limits Are Based on the Avoidance of Liver or Kidney Effects

Introduction

Two of the target organs most often affected by toxic exposure to industrial chemicals are the liver and the kidney. In recognition of this target organ toxicity, OSHA is establishing new or revised limits for 34 hepato- or nephrotoxic compounds. For these substances, the liver or kidney appears to be the organ most sensitive to the effects of exposure. Thus, establishing permissible exposure limits that are low enough to prevent toxicity to these target organs often also protects other organ systems.

Table C4–1 lists these hepatotoxic substances, along with their CAS numbers, H.S. numbers, 1987–1988 ACGIH TLV®s, and NIOSH RELs. In addition, Table C4–1 shows OSHA's current PELs for these substances in construction and maritime. Table C4–2 provides the same information for the nephrotoxins addressed in this section of the proposal. OSHA has no PELs for these substances in agriculture. Both on Tables C4–1 and C4–2, the right-hand column shows the limits OSHA is proposing today for construction, maritime, and agricultural workplaces. These limits are identical to those recently promulgated in general industry.

TABLE C4–1.—LIST OF SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED PRIMARILY ON AVOIDANCE OF LIVER TOXICITY

H.S. No./chemical name	CAS No.	Current OSHA PEL in construction and maritime *	1987–1988 ACGIH TLV®**	NIOSH REL †	Proposed OSHA PEL in construction, maritime, and agriculture *
2001 Acetylene tetrabromide	79–27–6	1 ppm TWA	1 ppm TWA		1 ppm TWA.
2003 Aldrin	309–00–2	0.25 mg/m^3 TWA, Skin.	0.25 mg/m^3 TWA, Skin	Lowest reliably detectable level ††	0.25 mg/m^3 TWA, Skin.

TABLE C4–1.—LIST OF SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED PRIMARILY ON AVOIDANCE OF LIVER TOXICITY—Continued

H.S. No./chemical name	CAS No.	Current OSHA PEL in construction and maritime *	1987–1988 ACGIH TLV***	NIOSH REL†	Proposed OSHA PEL in construction, maritime, and agriculture *
1011 Allyl chloride	107–05–1	1 ppm TWA	1 ppm TWA, 2 ppm STEL	1 ppm TWA, 3 ppm Ceiling (15-min).	1 ppm TWA, 2 ppm STEL.
1072 Carbon tetrabromide	558–13–4		0.1 ppm TWA, 0.3 ppm STEL		0.1 ppm TWA, 0.3 ppm STEL.
2028 Chlordane	57–74–9	0.5 mg/m³ TWA, Skin.	0.5 mg/m³ TWA, 2.0 mg/m³ STEL, Skin.		0.5 mg/m³ TWA, Skin.
2029 Chlorinated diphenyl oxide	55720–99–5	0.5 mg/m³ TWA	0.5 mg/m³ TWA, 2.0 mg/m³ STEL.		0.5 mg/m³ TWA.
2033 Chlorobenzene	108–90–7	75 ppm TWA	75 ppm TWA		75 ppm TWA.
2035 Chlorodiphenyl (42% Chlorine) (PCB).	53469–21–9	1 mg/m³ TWA, Skin.	1 mg/m³ TWA, 2 mg/m³ STEL, Skin.	0.001 mg/m³ TWA ††.	1 mg/m³ TWA, Skin.
2036 Chlorodiphenyl (54% Chlorine) (PCB).	11097–69–1	0.5 mg/m³ TWA, Skin.	0.5 mg/m³ TWA, 1 mg/m3 STEL, Skin.	0.001 mg/m³ TWA ††.	0.5 mg/m³ TWA, Skin.
1089 o-Chlorostyrene	2039–87–4		50 ppm TWA, 75 ppm STEL		50 ppm TWA, 75 ppm STEL.
1108 Cyclohexanone	108–94–1	50 ppm TWA	25 ppm TWA, Skin	25 ppm TWA	25 ppm TWA, Skin.
1126 1,1–Dichloroethane	75–34–3	100 ppm TWA	200 ppm TWA, 250 ppm STEL		100 ppm TWA.
2065 Dimethyl acetamide	127–19–5	10 ppm TWA, Skin.	10 ppm TWA, Skin		10 ppm TWA, Skin.
1145 Dioxane	123–91–1	100 ppm TWA, Skin.	25 ppm TWA, Skin	1 ppm Ceiling (30-min) ††.	25 ppm TWA, Skin.
1168 Ethylene dichloride	107–06–2	50 ppm TWA	10 ppm TWA	1 ppm TWA, 2 ppm Ceiling (15-min) ††.	1 ppm TWA, 2 ppm STEL.
2087 Hafnium	7440–58–6	0.5 mg/m³ TWA	0.5 mg/m³ TWA		0.5 mg/m³ TWA.
2089 Hexachloronaphthalene	1335–87–1	0.2 mg/m³ TWA, Skin.	0.2 mg/m³ TWA, Skin		0.2 mg/m³ TWA, Skin.
1205 Hydrazine	302–01–2	1 ppm TWA, Skin	0.1 ppm TWA, Skin A2	0.03 ppm Ceiling (120-min) ††.	0.1 ppm TWA, Skin.
1269 Methylcyclohexanol	25639–42–3	100 ppm TWA	50 ppm TWA		50 ppm TWA.
2118 Nitroethane	79–24–3	100 ppm TWA	100 ppm TWA		100 ppm TWA.
2119 Nitrogen trifluoride	7783–54–2	10 ppm TWA	10 ppm TWA		10 ppm TWA.
1295 Octachloronaphthalene	2234–13–1	0.1 mg/m³ TWA, Skin.	0.1 mg/m³ TWA, 0.3 mg/m³ STEL, Skin.		0.1 mg/m³ TWA, 0.3 mg/m³ STEL, Skin.
2123 Pentachloronaphthalene	1321–64–8	0.5 mg/m³ TWA, Skin.	0.5 mg/m³ TWA		0.5 mg/m³ TWA, Skin.
1341 Propylene dichloride	78–87–5	75 ppm TWA	75 ppm TWA, 110 ppm STEL		75 ppm TWA, 110 ppm STEL.
1385 1,1,2,2-Tetrachloroethane	79–34–5	5 ppm TWA, Skin	1 ppm TWA, Skin	(††)	1 ppm TWA, Skin.
2155 Tetrachloronaphthalene	1335–88–2	2 mg/m³ TWA, Skin.	2 mg/m³ TWA		2 mg/m³ TWA, Skin.
2163 Trichloronaphthalene	1321–65–9	5 mg/m³ TWA, Skin.	5 mg/m³ TWA, Skin		5 mg/m³ TWA, Skin.
1407 1,2,3-Trichloropropane	96–18–4	50 ppm TWA	10 ppm TWA, Skin		10 ppm TWA.

* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes only unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time; OSHA's PELs do not currently apply in Agriculture.

** The ACGIH TLV®-TWA is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time. An A2 designation means that the ACGIH classifies the substance as a suspected human carcinogen.

† NIOSH TWA limits are for 10 hour/day, 40 hour/week exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

†† NIOSH considers this substance a potential occupational carcinogen and recommends that exposures be reduced to the lowest feasible concentration.

Description of the Health Effects

The precise mechanisms by which toxic substances cause liver damage are not fully understood. In general, however, liver toxicity is a graded response (i.e., the severity of the lesion is directly proportional to the intensity/duration of exposure). Although many of the effects caused by exposure to these substances are reversible, some are not.

Liver damage is not a single entity; the manner in which it is manifested depends on the dose, duration, and particular chemical agent involved. For example, acute exposures may cause lipid accumulation in liver cells, cell death, and hepatobiliary dysfunction. In contrast, chronic overexposure leads to cirrhotic changes and the development of neoplasms. Fatty accumulation and necrosis can be either localized or widespread, and chemically induced lesions resulting from chronic exposures can cause marked changes of the entire liver (Plaa 1986/Ex. 1–183).

Typically, the earliest and most sensitive indicators of liver toxicity are alterations in biochemical liver functions, such as changes in specific enzyme activities. These may be accompanied by changes in the morphology of specific organelles in hepatocytes. For example, relatively low doses of halogenated aliphatic hydrocarbons, such as allyl chloride, carbon tetrabromide, and ethylene dichloride, cause an increase in the activity of microsomal mixed-function oxidase enzymes. This increase is ordinarily accompanied by proliferation of the endoplasmic reticulum.

Many compounds that damage the liver, such as 1,1,2,2-tetrachloroethane, also cause an abnormal accumulation of fat, especially of triglycerides, in liver cells. In experimental animals, this effect is manifested as an accumulation of microscopic vacuoles in liver cells. In humans, however, the only grossly detectable manifestation of this effect is increased liver size, which is an indication of severe fat accumulation in the liver.

At sufficiently high doses, most substances that damage the liver cause cell death that leads to tissue necrosis or

gangrene. This necrosis may initially be localized, but, at higher or more sustained exposure levels, the entire liver may be involved. Moderate to severe liver necrosis is usually accompanied by increased concentrations of marker enzymes such as glutamate-pyruvate transaminase or glutamate-oxaloacetate transaminase in the serum; the detection of these substances in the serum of exposed individuals can thus be a useful diagnostic tool.

Dose-Response Characteristics

The development of liver and other organ damage in humans and animals is progressive; it begins with subcellular changes, progresses to the cellular level, and is finally manifested as whole-organ damage. This progression is related to the intensity/duration of dose (i.e., as dose increases, cellular death becomes widespread and eventually causes liver dysfunction). The extent to which liver damage is reversible follows a similar continuum; since the liver can regenerate, minor cellular damage or transient disease states are usually reversible if exposure ceases. However, if exposure continues, the capacity of the liver to regenerate is exceeded and permanent damage results. As is the case for some chemically induced toxic effects, there appears to be a no-observed-effect level below which hepatotoxic effects do not occur.

The following paragraphs describe OSHA's preliminary findings for all of the substances in this group of hepatotoxins. Each discussion reports on the principal acute and chronic health effects associated with exposure to the substance and describes the physical properties and uses of the substance.

ACETYLENE TETRABROMIDE
CAS: 79–27–6; Chemical Formula: $CHBr_2CHBr_2$
H.S. No. 2001

In general industry, construction, and maritime, OSHA's current permissible exposure limit for acetylene tetrabromide is 1 ppm as an 8-hour TWA; there is no limit in agriculture. The ACGIH TLV®-TWA for acetylene tetrabromide is 1 ppm (14 mg/m³); there is no NIOSH REL for this substance. OSHA is proposing an 8-hour TWA PEL of 1 ppm for acetylene tetrabromide in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all regulated sectors.

Acetylene tetrabromide is a pale yellow, dense, non-combustible liquid with a sweet, chloroform-like odor (ACGIH 1986, p. 9; Genium MSDS 1985, No. 562). This substance is used as a solvent for fats, oils, and waxes; as a gauge fluid; as a catalytic polymer additive in synthetic fibers such as flame-resistant polystyrenes, polyurethanes, and polyolefins; as a refractive index liquid in microscopy; and as an ore flotation agent (Proctor, Hughes, and Fischman 1988, p. 54; Hawley's 1987, p. 11; HSDB 1988).

Acetylene tetrabromide is a sensory irritant, a liver toxin, and a central nervous system depressant. The LC_{50} in rats is 540 mg/m³ for 4 hours, and the oral LD_{50} in the same species is 1100 mg/kg (RTECS 1990). The dermal LD_{50} in rats is 5250 mg/kg (RTECS 1990). Guinea pigs exposed to a concentration of approximately 80 ppm acetylene tetrabromide for 90 minutes initially became comatose, appeared to recover, and then died after several days; rats and rabbits exposed to the same concentration for 3 hours lost consciousness after 2 hours but survived (Gray 1950). In another study, rats exposed to a concentration of approximately 80 ppm for 7 hours showed signs of slight eye and nose irritation (Hollingsworth, Rowe, and Oyen 1963). Rats exposed to aerosols of acetylene tetrabromide at concentrations ranging from 210 to 280 ppm for 2 hours showed mild signs of toxicity, and exposure to concentrations of from 420 to 500 ppm caused excitation followed by sleepiness (Arkhangel-Skaya and Yanushkevich 1956). Liquid acetylene tetrabromide instilled into the eyes of rabbits caused slight-to-moderate pain, conjunctival irritation, and reversible corneal injury (Van Haaften 1969). In prolonged contact with the skin of experimental animals, acetylene tetrabromide causes moderate redness, edema, and blistering (Van Haaften 1969; Clayton and Clayton 1981, p. 3517).

Rats, rabbits, mice, and a monkey exposed to a 14-ppm concentration of acetylene tetrabromide for 7 hours/day for 100 days showed edema of the lungs and slight fatty degeneration of the liver at autopsy; repeated exposure to a 4-ppm concentration for 180 days caused slight histopathological changes in the liver and lungs of some animals. At a 1-ppm concentration, no exposure-related effects were seen (Hollingsworth, Rowe, and Oyen 1969). A skin painting study in mice showed an increased incidence of lung tumors and/or forestomach papillomas in the test animals (Van Duuren et al., 1979).

A chemist exposed to an estimated average acetylene tetrabromide concentration of 2 ppm for 7.5 hours, with a peak exposure of 16 ppm for 10 minutes, complained of headache, anorexia, and nausea within a few hours of exposure. Within 5 days, he developed abdominal pain, bilirubinuria, and monocytosis; he also experienced liver damage that was severe and nearly fatal (Van Haaften 1969).

Based on this evidence in humans and animals, OSHA proposes to establish an 8-hour TWA PEL for acetylene tetrabromide of 1 ppm in agriculture; adoption of the proposed limit would establish the same PEL for workplaces in all OSHA-regulated industry sectors. The Agency preliminarily concludes that occupational exposure to acetylene tetrabromide causes sensory irritation and may cause liver damage and central nervous system depression. OSHA believes that, in the absence of a permissible exposure limit, workers in agriculture are potentially at significant risk for these exposure-related effects and that the proposed PEL will substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ALDRIN
CAS: 309–00–2; Chemical Formula: $C_{12}H_8Cl_6$
H.S. No. 2003

OSHA currently has no PEL for aldrin in agriculture; the limit for this substance in general industry, construction, and maritime is 0.25 mg/m³ as an 8-hour TWA, with a skin notation. The ACGIH TLV® is 0.25 mg/m³ TWA, with a skin notation. NIOSH considers this substance a potential human carcinogen and recommends that exposure be reduced to the lowest reliably detectable level. However, NIOSH concurs (Ex. 8–47, Table N8A) with the limit being proposed. OSHA is proposing to establish an 8-hour TWA limit of 0.25 mg/m³ and a skin notation in agriculture; this limit is consistent with OSHA's current limit for this substance in other sectors.

Aldrin is a nonflammable tan to dark-brown solid with a mild odor; it is a chlorinated naphthalene derivative that has been used extensively as an insecticide (Baselt 1980, p. 15; ACGIH 1986, p. 17). Aldrin is often found in the soil around buildings, where it has been used as a termiticide (Clayton and Clayton 1981, p. 3702). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In animals, aldrin causes convulsions; in mice, it has caused liver tumors (Proctor, Hughes, and Fischman 1988, p. 61). The lethal dose in 50 percent (LD_{50}) of rats given aldrin by oral

administration is 67 mg/kg; acutely poisoned animals had tremors and convulsed before death (Clayton and Clayton 1981, p. 3703). Aldrin can be absorbed through the skin in lethal amounts; the dermal LD_{50} for this substance is 150 mg/kg (Clayton and Clayton 1981, p. 3703). Chronic feeding studies in rats involving dietary aldrin doses of 12.5 ppm parts of diet or 0.02 mg/kg/day showed that degenerative liver changes and increases in liver weight had occurred in these animals (Clayton and Clayton 1981, p. 3703).

Aldrin has been tested for carcinogenicity in mice and rats by the oral route of administration. In mice, the results of these studies were positive; mice developed a statistically significant increase in the incidence of malignant liver neoplasms (NIOSH 1978). Based on this evidence, the International Agency for Research on Cancer has concluded that there is limited evidence in animals that aldrin is a carcinogen (IARC 1987, p. 88).

Workers exposed to aldrin at high concentrations exhibit the signs and symptoms of central nervous system effects: headache, nausea, dizziness, vomiting, myoclonic jerking of the limbs, and clonic-tonic convulsions; coma occurs in cases of severe poisoning (Proctor, Hughes, and Fischman 1988, p. 61). Five workers exposed to an aldrin concentration of 8.5 mg/m^3 convulsed and showed myoclonic movements of the limbs (Proctor, Hughes, and Fischman 1988, p. 61). Workers exposed to an unspecified concentration of aldrin dust reported experiencing headaches, dizziness, nausea, and vomiting, but no clinical signs of liver damage were seen in these individuals (Clayton and Clayton 1981, p. 3707). Aldrin can be absorbed through the skin in toxic amounts, and the skin is believed to be a major route of exposure in occupational situations (Clayton and Clayton 1981, p. 3707).

The evidence described above demonstrates that aldrin produces liver damage as well as central nervous system effects and convulsions among exposed animals and humans; this substance can cause these effects by inhalation of the dust, ingestion of airborne particulates, or absorption through the skin when aldrin is present in liquid vehicles.

OSHA therefore preliminarily finds that the absence of an exposure limit in longshoring and agriculture places workers in these occupational environments at significant risk of experiencing these material health impairments. The Agency believes that the establishment of an 8-hour TWA PEL of 0.25 mg/m^3, and a skin notation, is necessary to substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ALLYL CHLORIDE

CAS: 107–05–1; Chemical Formula: $CH_2 = CHCH_2Cl$

H.S. No. 1011

In construction and maritime, OSHA's current PEL for allyl chloride is 1 ppm (3 mg/m^3) as an 8-hour TWA; there is no PEL in agriculture. The ACGIH TLV®s for allyl chloride are 1 ppm as an 8-hour TWA and 2 ppm as a 15-minute STEL. NIOSH has recommended exposure limits of 1 ppm as a 10-hour TWA and 3 ppm as a 15-minute ceiling limit. NIOSH also concurs (Ex. 8–47, Table N1) with the proposed limits. OSHA is proposing an 8-hour TWA limit of 1 ppm and a STEL of 2 ppm for this substance in construction, maritime, and agriculture. These are the limits recently established in general industry.

Allyl chloride is a colorless liquid with an unpleasant, pungent odor. This substance is used as a chemical intermediate and as a catalyst and modifier in the production of resins (HSDB 1986).

Allyl chloride is an irritant, a liver, kidney, and pulmonary toxin, and, at high concentrations, a narcotic. This substance also has reproductive, embryotoxic, and developmental effects in experimental animals. The oral LD_{50} in rats is 64 mg/kg, and the lowest lethal concentration in the same species by inhalation is 290 ppm for 8 hours (RTECS 1990). In rabbits, the median lethal dose by percutaneous absorption is 2066 mg/kg (RTECS 1990). Rats exposed to a 16,000-ppm concentration for 2 hours showed signs of eye and nose irritation and became drowsy, weak, and uncoordinated before developing dyspnea and dying (Proctor, Hughes, and Fischman 1988, p. 63). Guinea pigs exposed to the same concentration for 1 hour showed the same exposure-related effects (Proctor, Hughes, and Fischman 1988, p. 63). Acutely poisoned animals showed liver and kidney damage and pulmonary hemorrhage at autopsy (Proctor, Hughes, and Fischman 1988, p. 63).

Single exposures to allyl chloride lasting only a few minutes at concentrations between 332 ppm and 32,000 ppm caused mucous membrane irritation in various laboratory animals; exposure to an 8-ppm concentration for 5 weeks caused kidney and liver damage (Adams, Spencer, and Irish 1940/Ex. 1–584). Other animal studies have confirmed this substance's liver and kidney toxicity in many species (Torkelson, Wolf, Oyen, and Rowe 1959/Ex. 1–691).

In the prior rulemaking, NIOSH (Ex. 150) commented that a recent bioassay (Santodonato et al. 1985) has shown that allyl chloride is also a tumor initiator in mice. This substance is a mutagen in bacterial test systems, both with and without activation (RTECS 1990). Several studies (RTECS 1990) have shown that, when administered to rats or mice during pregnancy, allyl chloride is embryotoxic and has both reproductive and developmental effects (RTECS 1990).

Exposures of 50 to 100 ppm allyl chloride for five minutes in humans caused eye and nose irritation, and five-minute exposures below 25 ppm have been associated with pulmonary irritation (Shell Chemical Corp. 1974, as cited in Ex. 150). If the liquid is absorbed through the skin of humans, it causes pain in the area of contact that is described as deep-seated (Clayton and Clayton 1981, p. 3569). Humans exposed to concentrations of 1 to 113 ppm showed abnormal liver test results (Hausler and Lenich 1968/Ex. 1–1035), and there is a report that workers exposed to a 3-mg/m^3 (1 ppm) concentration showed signs of kidney dysfunction (Clayton and Clayton 1981, p. 3572).

Chinese factory workers exposed to 0.8 to 2100 ppm allyl chloride for periods ranging from 2.5 months to 5 years developed weakness, paresthesia, pain, and numbness and sensory impairment in the extremities (Proctor, Hughes, and Fischman 1988, p. 63). Workers at another factory who were exposed to 0.06 to 8 ppm for 1 to 4.5 years showed lesser signs and symptoms (Proctor, Hughes, and Fischman 1988, p. 63).

Based on this evidence in humans and animals, OSHA is proposing to add a 2-ppm STEL to the current 8-hour TWA limit of 1 ppm in construction and maritime and is also proposing to extend both limits to agriculture. The Agency preliminarily concludes that both TWA and STEL limits are necessary to protect workers in these sectors from the significant risks of kidney and liver damage and neuropathic effects that are potentially associated with exposure to this substance. OSHA considers these effects material impairments of health and believes that the proposed PELs are necessary to reduce this significant risk. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

CARBON TETRABROMIDE

CAS: 558–13–4; Chemical Formula: CBr_4

H.S. No. 1072

OSHA has no limit for carbon tetrabromide in construction, maritime, or agriculture. The 1987–1988 ACGIH TLV®s for this substance are 0.1 ppm as an 8-hour TWA and 0.3 ppm as a 15-minute STEL. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the 0.1 ppm 8-hour TWA limit and 0.3 ppm 15-minute STEL OSHA is proposing for construction, maritime, and agriculture. These are the limits recently established for this substance in general industry.

At room temperature, pure carbon tetrabromide is a colorless, nonflammable solid; however, the commercial product is usually yellow-brown in color. This substance finds use as a chemical intermediate.

Carbon tetrabromide is considered a highly toxic material (Clayton and Clayton 1981, p. 3479). In addition to liver toxicity, carbon tetrabromide causes irritation of the eyes and upper respiratory tract, and kidney and lung damage (Clayton and Clayton 1981, p. 3479). Even at low levels, carbon tetrabromide is a powerful lacrimator (Clayton and Clayton 1981, p. 3479). In contact with the eyes, the pure material causes irreversible corneal damage; skin contact causes mild irritation and redness (Clayton and Clayton 1981, p. 3479). The lowest lethal dose in rats by oral administration is 1000 mg/kg, and the intravenous LD_{50} in mice is 56 mg/kg (RTECS 1990). Rats exposed to a 0.07 to 74 ppm concentration for 4 hours daily over a 4-month period developed metabolic changes in the liver (Clayton and Clayton 1981, p. 3479). Carbon tetrabromide's hepatotoxic effects include both fatty infiltration and necrosis. Rats exposed to carbon tetrabromide by inhalation for 7 hours per day, 5 days per week for 6 months showed no effects, while repeated exposures to higher concentrations caused fatty changes and degeneration of the liver discernible at autopsy (Torkelson and Rowe 1981a/Ex. 1–974; Clayton and Clayton 1981, pp. 3479–3480).

OSHA is proposing a 0.1-ppm 8-hour TWA limit and a 0.3-ppm 15-minute STEL for carbon tetrabromide in construction, maritime, and agriculture. OSHA preliminarily concludes that these limits are necessary to protect workers in these sectors against the significant risk of hepatotoxic, irritant, and kidney and lung effects associated with exposure to this substance. OSHA considers such effects material health impairments and believes that the proposed PELs will substantially reduce these risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

CHLORDANE

CAS: 57–74–9; Chemical Formula: $C_{10}H_6Cl_8$

H.S. No. 2028

OSHA's PEL for chlordane in general industry, construction, and maritime is 0.5 mg/m³ as an 8-hour time-weighted average (TWA), with a skin designation; there is no PEL in agriculture for this substance. The 1987–1988 ACGIH TLV®–TWA for chlordane is 0.5 mg/m³ and the TLV®–STEL is 2 mg/m³, with a skin designation; NIOSH has no REL but concurs (Ex. 8–47, Table N6A) with the limit being proposed. OSHA is proposing both an 8-hour TWA PEL of 0.5 mg/m³ and a skin designation for chlordane in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all regulated sectors.

Chlordane is an odorless, viscous liquid that ranges from colorless to amber in appearance (Hawley's 1987, p. 256; ACGIH 1986; p. 114). This substance is used almost exclusively as an insecticide against termites. It was formerly used as an acaricide and to control grubs, ants, webworms, armyworms, cutworms, chiggers, and leafhoppers (HSDB 1986; Hawley's 1987, p. 256). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide ACT (FIFRA).

Chlordane is a convulsant and liver toxin in humans and animals and a liver carcinogen in mice (Proctor, Hughes, and Fischman 1988, p. 126). The oral LD_{50} in rats is 283 mg/kg; the LC_{50} in cats is 100 mg/m³ for 4 hours; and the dermal LD_{50} in rabbits is 780 mg/kg (RTECS 1990). Acutely poisoned animals convulse before death (Hayes 1982, p. 229). Animals given daily oral doses of 50 mg/kg chlordane for 2 weeks died, while those given 25 mg/kg for the same period did not (ACGIH 1986, p. 114). At autopsy, rats fed 0.008, 0.15, or 0.32 percent chlordane for 407 days showed liver damage; other organs appeared unaffected (ACGIH 1986, p. 114).

During a 2-year study in rats fed chlordane at 5, 10, 30, 150, or 300 ppm, the following effects were reported: the higher doses of 150 and 300 ppm caused notable growth retardation, marked liver and kidney damage, marked lung damage (300 ppm), and mild lung injury (150 ppm). Liver damage was slight at 30 ppm and minimal at 10 ppm; no liver damage was reported in those animals fed at the 5-ppm level (Clayton and Clayton 1981, p. 3721). Chlordane has been tested in bioassays in mice and rats. In mice, oral administration caused hepatocellular carcinomas in mice of both sexes; in rats, the results were inconclusive. Based on this evidence, the International Agency for Research on Cancer (IARC) has concluded that the evidence for the carcinogenity of chlordane in animals is sufficient (IARC 1974, p. 58).

The signs and symptoms associated with acute oral or dermal exposure of humans to chlordane are loss of appetite, irritability, hyperexcitability, vomiting, tremors, and convulsions, and, if overexposure is severe, death (Clayton and Clayton 1981, p. 3718). Skin and mucous membrane irritation have been reported from contact with technical grade chlordane, but this effect may have been due to contamination of the chlordane with hexachlorocyclopentadiene (Proctor, Hughes, and Fishman 1988, p. 126). Chlordane is rapidly absorbed through the skin and has caused at least one fatality by this route (Proctor, Hughes, and Fishman 1988, p. 126). Workers exposed to chlordane and aldrin at a 5-mg/m³ concentration for 1 to 3 years showed no adverse effects (ACGIH 1986, p. 114). After accidental ingestion, individuals convulse and experience nausea and vomiting; confusion, excitability, incoordination, and coma may also occur (Hayes 1982, p. 230). A formulation plant worker who spilled chlordane on her abdomen and thighs died after experiencing convulsions (Hayes 1982, p. 230). IARC reports that case reports in humans suggest a relationship between exposure to chlordane, alone or in combination with other compounds, and the development of blood dyscrasias (IARC 1974, p. 58).

Based on this evidence in humans and amimals, OSHA preliminarily concludes that agricultural workers exposed to chlordane at the levels permitted by the absence of a limit are at significant risk of experiencing convulsions and liver damage, and perhaps death. The Agency believes that establishing a PEL of 0.5 mg/m³ as an 8-hour TWA, and a skin designation, is necessary to protect workers in agriculture from these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CHLORINATED DIPHENYL OXIDE

CAS: 55720–99–5; Chemical Formula: $C_6H_2Cl_3OC_6H_3Cl_3$ (Approximate)

H.S. No. 2029

OSHA's limit for chlorinated diphenyl oxide in general industry, construction, and maritime workplaces is 0.5 mg/m³

as an 8-hour time-weighted average. There is no limit for this substance in agriculture. In 1987–1988, the ACGIH had a TLV®–TWA of 0.5 mg/m³ and TLV®–STEL 2.0 mg/m³ for chlorinated diphenyl oxide; NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing to establish a permissible exposure limit (PEL) of 0.5 mg/m³ as an 8-hour TWA for chlorinated diphenyl oxide in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all sectors.

The physical form of chlorinated diphenyl oxide varies from a viscous, colorless, oily liquid to a yellowish, waxy semisolid, depending on the chlorine equivalents in the compound's formula. The chlorine equivalents vary from 1 (monochloro-) to 6 (hexachloro-) diphenyl oxide (ACGIH 1986, p. 116; Proctor, Hughes, and Fischman 1988, p. 128). Chlorinated diphenyl oxide is used as a solvent; an intermediate in the organic synthesis of pesticides, wood preservatives, hydraulic fluids, corrosion inhibitors, thermal lubricants, etc.; and as a dielectric fluid in the electrical industry (Proctor, Hughes, and Fischman 1988, p. 128; NIOSH/OSHA Occupational Health Guideline 1981, p. 3).

Exposure to chlorinated diphenyl oxide causes chloracne (acneform dermatitis) and cumulative liver damage in animals (Proctor, Hughes, and Fischman 1988, p. 128). In guinea pigs, the lowest lethal dose by oral administration is 50 mg/kg (RTECS 1989). Application of hexachlorodiphenyl oxide to the skin of rabbits caused marked irritation and epithelial hyperplasia (Clayton and Clayton 1982, p. 2550). Repeated application to the skin of rabbits caused systemic poisoning, manifested at autopsy by liver injury (Clayton and Clayton 1982, p. 2550).

Systemic effects have not been reported in humans exposed to chlorinated diphenyl oxides. However, acneform dermatitis (chloracne) has occurred as a result of industrial exposures (Proctor, Hughes, and Fischman 1988, p. 128). Affected individuals develop comedones and cysts on the face, ears, neck, shoulders, arms, chest, abdomen, and scrotum (Proctor, Hughes, and Fischman 1988, p. 128).

Based on this evidence, OSHA preliminarily concludes that the absence of a limit for chlorinated diphenyl oxide poses a significant risk of chloracne and liver damage to workers in agriculture who are exposed to this substance. The Agency believes that the proposed PEL for chlorinated diphenyl oxide of 0.5 mg/m³ as an 8-hour TWA is necessary to substantially reduce these significant risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CHLOROBENZENE

CAS: 108–90–7; Chemical Formula: C_6H_5Cl

H.S. No. 2033

OSHA's PEL for chlorobenzene in general industry, construction, and maritime is 75 ppm as an 8-hour time-weighted average (TWA); there is no PEL in agriculture. The 1987–1988 ACGIH TLV®–TWA for chlorobenzene is 75 ppm; there is no NIOSH REL for this substance. OSHA is proposing an 8-hour TWA limit of 75 ppm for chlorobenzene in agriculture. Promulgation of this limit will make the PEL for chlorobenzene consistent across all regulated sectors.

Chlorobenzene is a colorless to pale-yellow liquid with an odor similar to that of almonds (ACGIH 1986, p. 123; HSDB 1986). It is used as a chemical intermediate in the manufacture of DDT, aniline, phenol, ortho- and para-chlorobenzene, and dyestuffs, and as a solvent in the manufacture of adhesives, paints, polishes, waxes, diisocyanates, pharmaceuticals, and natural rubber. It is also used as a fiber swelling agent and dye carrier in the textile industry and as a tar and grease remover in cleaning and degreasing operations (HSDB 1986).

Chlorobenzene causes irritation of the eyes and nose and narcosis in both animals and humans; in laboratory animals, chronic exposure causes liver, kidney, and lung damage (ACGIH 1986, p. 123(89); Proctor, Hughes, and Fischman 1988, p.133; Clayton and Clayton 1982, pp. 3605–3610). The oral LD_{50} in rats is 2910 mg/kg (RTECS 1989). The dermal toxicity of chlorobenzene is reported to be low; for guinea pigs, dermal doses of 10 ml/kg were nonlethal (ACGIH 1986, p. 123.3(89)). Subcutaneous injections of 7 to 8 g/kg resulted in the death of injected rats within a few hours of administration; necrosis of the liver and kidneys was observed in these animals at autopsy (Clayton and Clayton 1982, p. 3605). Exposure to an 8000-ppm concentration of chlorobenzene caused eye and nose irritation, followed by severe narcosis in cats after 30 minutes; death occurred within 2 hours of exposure (Proctor, Hughes, and Fischman 1988, p. 133). At a concentration of 1200 ppm for an unspecified time, cats showed narcotic effects; at a concentration of 200 to 660 ppm, exposure caused no significant effects even after several hours (Clayton and Clayton 1982, p. 3605). At autopsy, histopathological changes were seen in the liver, kidneys, and lungs of laboratory animals exposed to a 1000-ppm concentration of chlorobenzene for 7 hours/day, 5 days/week for 44 days (for a total of 32 exposures). When the concentration was reduced to 475 ppm, the changes seen at the higher concentrations occurred but were less severe (Clayton and Clayton 1982, p. 3607). Mice exposed to a 544-ppm concentration of chlorobenzene for 7 hours/day for 3weeks or 22 ppm for 7 hours/day for 3 months suffered from leukopenia and depressed bone marrow activity (Proctor, Hughes, and Fischman 1988, p. 133). Oral toxicity studies in rats that were dosed 5 days/week for 192 days (for a total of 137 doses) showed the following effects: doses of 0.144 g/kg and 0.288 g/kg both caused slight changes in the liver and a significant increase in liver and kidney weights, while a dose of 0.0144 g/kg caused no observable effects (Clayton and Clayton 1982, pp. 3606–3607).

In humans, eye and nose irritation begins on exposure to concentrations of 200 ppm (Proctor, Hughes, and Fischman 1988, p. 133). Contact of the skin with liquid chlorobenzene causes mild skin irritation; prolonged contact may lead to dermatitis (Proctor, Hughes, and Fischman 1988, p. 133). A study of 52 workers exposed to chlorobenzene reported that many of the workers who had been exposed to chlorobenzene (concentration unspecified) for 1 to 2 years experienced headache, dizziness, sleepiness, and dyspeptic disorders. Some of these workers showed spastic muscle contractions and paresthesia of the extremities when tested clinically, and others suffered from vasovegetative instability (Clayton and Clayton 1982, pp. 3610–3611).

Based on this evidence, OSHA preliminarily concludes that agricultural workers exposed to chlorobenzene at the levels permitted by the absence of a limit are at significant risk of experiencing eye and nose irritation; liver, lung, and kidney damage; and narcosis. The Agency believes that establishing a PEL of 75 ppm as an 8-hour TWA is necessary to protect workers in agriculture from these significant occupational risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CHLORODIPHENYL (42% CHLORINE) (PCB)

CAS: 53469–21–9; Chemical Formula: $C_{12}H_7Cl_5$ (Approximate)

H.S. No. 2035

In general industry, construction, and maritime, OSHA's permissible exposure limit (PEL) for chlorodiphenyl (42% chlorine) is 1 mg/m³ as an 8-hour time-weighted average (TWA), with a skin designation. There is no limit for this substance in agriculture. The 1987–1988 ACGIH TLV®–TWA was 1 mg/m³ and the TLV®–STEL was 2 mg/m³, with a skin designation. NIOSH considers this substance a potential human carcinogen and recommends reducing exposure to the lowest feasible concentration (0.001 mg/m³). OSHA is proposing an 8-hour TWA limit of 1 mg/m³, with a skin designation, for chlorodiphenyl (42% chlorine) in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all regulated sectors.

Chlorodiphenyl (42% chlorine) is a polychlorinated biphenyl (PCB); it is a clear, colorless to straw-colored liquid with a mild hydrocarbon odor. The production and sale of PCBs was discontinued by the EPA in 1977, but this substance is still found in transformers and capacitors that have remained in use (HSDB 1988; Merck 1983, p. 1091; ACGIH 1986, p. 128).

Chlorodiphenyl (42% chlorine) is toxic to the liver, is an irritant of the eyes and mucous membranes, and causes acneform dermatitis (chloracne). It is carcinogenic, embryotoxic, and teratogenic in laboratory animals (ACGIH 1986, p. 128; Proctor, Hughes, and Fischman 1988, p. 136; IARC 1987, p. 324). The oral LD_{50} for chlorodiphenyl (42% chlorine) in rats ranges from 4250 to 8700 mg/kg, and the dermal LD_{50} in rabbits ranges from 800 to 1300 mg/kg (IARC 1978, p. 69; RTECS 1990). Monkeys given 300 ppm of this substance in their diet for 90 days showed hair loss, chloracne, subcutaneous edema, liver hypertrophy, and hyperplasia of the gastric mucosa (Gosselin, Smith, and Hodge 1984, p. II–171). Chlorodiphenyl (42% chlorine) is rapidly absorbed through the skin and accumulates in lipid-rich tissues such as those of the brain. Exposure to chlorodiphenyl (42% chlorine) by any route produces skin changes that may lead to acneform dermatitis and sticky eye discharge in laboratory animals (Proctor, Hughes, and Fischman 1988, p. 137; Grant 1986, p. 750). Skin contact results in depletion of the natural oils of the skin, which leads to local redness and skin cracking (AIHA 1965). This substance is toxic to the liver, causing fatty degeneration, cell death, hypertrophy, and induction of microsomal enzymes (Proctor, Hughes, and Fischman 1988, p. 138; IARC 1978, p. 68; Gosselin, Smith, and Hodge 1984, p. II–171 to II–172). Rabbits fed 30 mg/m³ chlorodiphenyl (42% chlorine) for 14 weeks developed enlarged livers, and rats fed 100 ppm for as long as 10 months showed no clinical liver effects but did have enlarged hepatocytes at autopsy (HSDB 1988). Although cats, rabbits, and rats showed no ill effects when exposed for 7hours/day to an 8.6-mg/m³ concentration over a 24-day period, guinea pigs similarly exposed demonstrated poor growth (AIHA 1965).

Workers exposed to 0.1 mg/m³ chlorodiphenyl (42% chlorine) for several months developed mild-to-moderate skin irritation and chloracne, and exposure to a level of 10 mg/m³ was reported to be intolerably irritating (Proctor, Hughes, and Fischman 1988, p. 137). Workers exposed to chlorodiphenyl (42% chlorine) at concentrations below 0.1 mg/m³ have not been known to develop chloracne. Occupationally exposed individuals have shown clinical evidence of liver injury, and deaths caused by toxic hepatitis have occurred in workers exposed to a mixture of PCBs and chlorinated naphthalenes (Proctor, Hughes, and Fischman 1988, p. 137). The International Agency for Research on Cancer (IARC) has concluded that the evidence for chlorodiphenyl-induced carcinogenicity in humans is limited, although studies in humans do support an association between cancer and exposure to these substances (IARC 1987, p. 324). A study of workers in Italy who had been exposed to a mixture of chlorodiphenyls (54% and 42% chlorine) reported an increase in cancer mortality among these workers; male workers had a significant excess of gastrointestinal tract tumors (including one liver tumor) and female workers had a significant excess of hematological tumors (IARC 1987, Supplement 7, p. 323). Several thousand Japanese and Taiwanese citizens who accidentally ingested chlorodiphenyl (42% chlorine) over several months developed swelling of the upper eyelids, excessive eye discharge, follicular keratosis, gastrointestinal problems, various nervous symptoms, peripheral neuropathy, darkening of the skin, and chronic bronchitis; babies born to women who became pregnant during this period also showed many of these signs (IARC 1978, pp. 80–84).

Based on this evidence in humans and animals, OSHA preliminarily concludes that agricultural workers exposed to chlorodiphenyl (42% chlorine) at the levels permitted by the absence of a limit are at significant risk of irritation, chloracne, liver damage, and, perhaps, of cancer. The Agency believes that establishing a PEL of 1 mg/m³ as an 8-hour TWA, and a skin designation, will substantially reduce significant risk for workers in agriculture. OSHA recognizes that a lower limit may be needed for chlorodiphenyl (42% chlorine). As discussed above, however, the principal purpose of this rulemaking is to extend to all workers the protection given to workers in general industry. The need for a change to the limit for this substance will be considered for all workers in a future update of the Agency's PELs.

CHLORODIPHENYL (54% CHLORINE) (PCB)

CAS: 11097–69–1; Chemical Formula: $C_{12}H_5Cl_5$

H.S. No. 2036

In general industry, construction, and maritime, OSHA's permissible exposure limit (PEL) for chlorodiphenyl (54% chlorine) is 0.5 mg/m³ as an 8-hour time-weighted average (TWA), with a skin designation. There is no limit in agriculture for this substance. The 1987–1988 ACGIH TLV®–TWA was 0.5 mg/m³ and the TLV®–STEL was 1 mg/m³, with a skin designation. NIOSH considers this substance a potential human carcinogen and recommends reducing exposure to the lowest feasible concentration (0.001 mg/m³). OSHA is proposing to establish an 8-hour TWA limit of 0.5 mg/m³, with a skin designation, for chlorodiphenyl (54% chlorine) in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all regulated sectors.

Chlorodiphenyl (54% chlorine) is a polychlorinated biphenyl (PCB); it is a pale yellow, viscous liquid with a mild hydro-carbon odor. The production and sale of PCBs was halted by the EPA in 1977, but this substance is still found in transformers and capacitors that have remained in use (ACGIH 1986, p. 129; Merck 1983, p. 1091).

Exposure to chlorodiphenyl (54% chlorine) causes irritation, chloracne, and liver damage. In laboratory animals, this substance is carcinogenic, embryotoxic, and teratogenic. The oral LD_{50} in rats is 1010 mg/kg, and the intravenous LD_{50} in the same species is 358 mg/kg (RTECS 1990). Acutely poisoned rats develop diarrhea, ataxia, loss of pain response, and narcosis (Gosselin, Smith, and Hodge 1984, p. II–171). The minimum lethal dermal dose in rabbits is 1.5 g/kg (Proctor, Hughes, and Fischman 1988, p. 142). Repeated dermal application caused hyperplasia and hyperkeratosis in the epithelium, liver and kidney damage, and atrophy of the thymus (Gosselin, Smith, and Hodge

1984, p. II–17). Rats exposed to a 5.4-mg/m³ concentration of chlorodiphenyl (54% chlorine) for 7 hours/day for 4 months showed an increase in liver weight and, at autopsy, liver damage (Proctor, Hughes, and Fischman 1988, p. 142). Dietary administration of chlorodiphenyl (54% chlorine) in mice and rats has caused a significant excess of benign and malignant liver tumors in several bioassays. Rats also developed interstitial metaplasia and a low, statistically nonsignificant incidence of stomach adenocarcinomas (IARC 1987, Supplement 7, p. 322). The International Agency for Research on Cancer (IARC) has concluded that the evidence for the carcinogenicity of chlorodiphenyl (54% chlorine) in animals is sufficient (IARC 1987, Supplement 7, p. 322). Chlorodiphenyl (54% chlorine) also has caused fetotoxicity, maternal death, abortion, and stillbirth when administered orally to rabbits at a dose of 12.5 mg/kg (Clayton and Clayton 1981, p. 3667). A dose-related increase in the number of malformed offspring occurred in pigs and rats fed chlorodiphenyl (54% chlorine) at a dose of 1 mg/kg/day (Proctor, Hughes, and Fischman 1988, p. 143). Chlorodiphenyl (54% chlorine) is mutagenic in mammalian test systems (RTECS 1990).

Workers exposed to 0.1 mg/m³ chlorodiphenyl for several months developed mild-to-moderate irritation of the skin and chloracne; however, exposure to chlorodiphenyls at concentrations below 0.1 mg/m³ did not produce chloracne (Proctor, Hughes, and Fischman 1988, p. 140). Exposure to a 10-mg/m³ concentration is reported to be intolerably irritating (Proctor, Hughes, and Fischman 1988, p. 140). Workers who have PCB-induced chloracne also often show clinical evidence of liver disease, and several workers have died of toxic hepatitis after exposure to PCBs and chlorinated naphthalenes (Proctor, Hughes, and Fischman 1988, p. 140). Studies in humans have shown that workers exposed to chlorodiphenyl (54% chlorine) in electrical capacitor filling and sealing operations have experienced a slight increase in the incidence of cancer, particularly in melanomas of the skin; another study showed an excess number of deaths due to cancer of the liver and biliary passages in chlorodiphenyl-exposed workers (IARC 1987, Supplement 7, p. 323). A study of workers in Italy who had been exposed to a mixture of chlorodiphenyls (54% and 42% chlorine) reported a significant increase in cancer mortality among these workers; male workers had a significant excess of gastrointestinal tract tumors (including one liver tumor) and female workers had a significant excess of hematological tumors (IARC 1987, Supplement 7, p. 323). IARC has concluded that the available human studies suggest an association between cancer and exposure to PCBs and notes that the increased risk of hepatobiliary cancer is consistent across different studies (IARC 1987, Supplement 7, p. 324).

Based on this evidence in humans and animals, OSHA preliminarily concludes that agricultural workers exposed to chlorodiphenyl (54% chlorine) at the levels permitted by the absence of a limit are at significant risk of irritation, chloracne, liver damage, and, perhaps, of cancer. The Agency believes that establishing a PEL of 0.5 mg/m³ as an 8-hour TWA, and a skin designation, will substantially reduce significant risk for workers in agriculture. OSHA recognizes that a lower limit may be needed for chlorodiphenyl (54% chlorine). As discussed above, however, the principal purpose of this rulemaking is to extend to all workers the protection given to workers in general industry. The need for a change to the limit for this substance will be considered for all workers in a future update of the Agency's PELs.

o-CHLOROSTYRENE

CAS: 2039–87–4; Chemical Formula: C_8H_7Cl

H.S. No. 1089

In construction, maritime, and agriculture, OSHA has no limit for o-chlorostyrene. The ACGIH TLV®s are an 8-hour TWA of 50 ppm and a STEL of 75 ppm; NIOSH has no REL for this substance but concurs (Ex. 8–47, Table N1) that the proposed limits are appropriate. OSHA is proposing a 50-ppm TWA PEL and a 75-ppm STEL for o-chlorostyrene in construction, maritime, and agriculture. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

o-Chlorostyrene is a colorless liquid at room temperature. This substance is used as a chemical intermediate and in the manufacture of polymers (Sittig 1985, p. 241).

o-Chlorostyrene is an irritant of the eyes and skin on acute exposure, and chronic exposure causes kidney as well as liver damage (New Jersey Fact Sheet 1986, pp. 1, 2). In an unpublished report, the Dow Chemical Company (1973a, as cited in ACGIH 1986/Ex.1–3, p. 136) describes the results of an o-chlorostyrene inhalation study in rats, rabbits, guinea pigs, and dogs. Dow exposed the animals to an average o-chlorostyrene concentration of 101 ppm for 7 hours daily, 5 days per week, for a total of 130 exposures in 180 days. No adverse effects were observed in any species in terms of appearance, growth, behavior, mortality, hematology, BUN, alkaline phosphatase, SGPT, BSP, organ weights, or gross pathology (Dow Chemical Company 1973a, as cited in ACGIH 1986/Ex. 1–3, p. 136). Microscopic examination of animal tissue revealed a somewhat higher incidence of pathological changes in the liver and kidneys (Dow Chemical Company 1973a, as cited in ACGIH 1986, Ex. 1–3, p. 136).

A paper by Coulter, Kehde, and Hiscock (1971) reports that o-chlorostyrene has toxicological properties that are similar to those of styrene; since human volunteers exposed for 1 to 7 hours to the vapor of styrene at concentrations of 100 ppm have shown narcotic effects (Stewart, Dodd, Baretta, and Schaffer 1968, Ex. 1–380), exposure to o-chlorostyrene should be maintained at concentrations below this level at all times.

Based on this evidence, OSHA is proposing PELs for o-chlorostyrene in construction, maritime, and agriculture of 50 ppm as an 8-hour TWA and 75 ppm as a 15-minute STEL. The Agency preliminarily concludes that both of these limits will protect workers in these sectors from the significant risks of liver and kidney damage and narcosis to which they could potentially be exposed in the absence of an OSHA limit. OSHA believes that these health effects constitute material health impairments and that the proposed TWA and STEL limits are necessary to substantially reduce these significant occupational risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

CYCLOHEXANONE

CAS: 108–94–1; Chemical Formula: $C_6H_{10}O$

H.S. No. 1108

OSHA's limit for cyclohexanone in construction and maritime is 50 ppm as an 8-hour TWA. The ACGIH 1987–1988 TLV® for this substance is 25 ppm as an 8-hour TWA, with a skin notation. NIOSH has a REL of 25 ppm as a 10-hour TWA for cyclohexanone and also concurs (Ex. 8–47, Table N1) with the limit OSHA is proposing in construction, maritime, and agriculture, which is an 8-hour TWA PEL of 25 ppm, with a skin notation. This is the limit recently established for this substance in general industry.

Cyclohexanone is a white to pale yellow, oily liquid with an odor similar to that of acetone and peppermint. This

substance is used as a solvent in metal degreasing operations and in the production of acetates, resins, rubber, waxes, shellacs, nitrocellulose, and pesticides (ACGIH 1986, p. 159; HSDB 1986). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In addition to liver damage, exposure to cyclohexanone causes eye, nose, and upper respiratory tract irritation and, at high concentrations, narcosis in experimental animals. The oral LD_{50} in rats is 1535 mg/kg, and the LC_{50} in the same species is 8000 ppm for 4 hours (RTECS 1990). The lowest reported dermal LD_{50} in rabbits is 948 mg/kg (RTECS 1990). Cyclohexanone has been studied in several experimental animal species. A concentration of 2000 ppm inhaled for four hours was lethal to one of six rats; at 4000 ppm, all of the exposed animals died (Smyth, Carpenter, Weil et al. 1969/Ex. 1–442). Rabbits showed marked irritation and some corneal injury when undiluted cyclohexanone was instilled into the eye (Carpenter and Smyth 1946/Ex. 1–859); applied to the skin of rabbits, this substance caused mild irritation (RTECS 1990). Guinea pigs exposed to the vapor at a concentration of 4000 ppm for 6 hours showed narcotic symptoms, lacrimation, salivation, depression of body temperature and heart rate, and corneal opacity (Specht, Miller, Valaer, and Sayers 1940/Ex. 1–1179). At autopsy, rabbits exhibited degenerative changes of the liver and kidneys after 50 daily 6-hour inhalation exposures to 190 ppm (Treon, Crutchfield, and Kitzmiller 1943b/Ex. 1–394). Exposures to a 309-ppm concentration of cyclohexanone on the same regimen caused conjunctival congestion, while exposures to a 3000-ppm concentration of cyclohexanone were lethal to some of the exposed rabbits (Treon, Crutchfield, and Kitzmiller 1943b/Ex. 1–394). When the cyclohexanone concentration was increased to approximately 3082 ppm, the rabbits lost coordination, developed difficult breathing, and showed signs of narcosis (Treon, Crutchfield, and Kitzmiller 1943b/Ex. 1–394).

In humans, Nelson and co-workers (1943/Ex. 1–66) reported that irritation caused by exposure to cyclohexanone was intolerable at 50 ppm; however, 25 ppm was not objectionable to most subjects in 3- to 5-minute exposures (Nelson, Enge, Ross et al. 1943/Ex. 1–66). Marked eye and upper respiratory tract irritation occurred at a concentration of 75 ppm (Clayton and Clayton 1981, p. 4782).

OSHA is proposing to add a skin notation for cyclohexanone based on this substance's ability to cause systemic toxicity via absorption through the skin. In the prior Air Contaminants rulemaking, one commenter (Ex. 3–678) stated that, in his opinion, there was no evidence for cyclohexanone's dermal toxicity, and thus that no skin notation was necessary. In response to this commenter, OSHA pointed to the dermal LD_{50} in rabbits of 948 mg/kg (RTECS 1990) and to the Agency's policy of establishing skin notations for substances having dermal LD_{50}s in rabbits of 1000 mg/kg or less.

Based on this evidence, OSHA is proposing an 8-hour TWA PEL for cyclohexanone of 25 ppm, and a skin notation, in construction, maritime, and agriculture. The Agency has preliminarily determined that this limit and notation will protect workers in construction, maritime, and agriculture from the significant risks of liver and kidney damage, skin and respiratory tract irritation, and percutaneous absorption associated with exposure to this substance. OSHA considers these effects material health impairments and believes that the proposed PEL is necessary to substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

1,1-DICHLOROETHANE

CAS: 75–34–3; Chemical Formula: CH_3CHCl_2

H.S. No. 1126

OSHA's PEL for dichloroethane in general industry, construction, and maritime is 100 ppm as an 8-hour TWA. There is no PEL in agriculture. The ACGIH TLV®s for this substance are 200 ppm as an 8-hour TWA and 250 ppm as a 15-minute STEL, respectively. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. In agriculture, OSHA is proposing an 8-hour TWA PEL of 100 ppm for 1,1-dichloroethane. Promulgation of this limit will make OSHA's PEL for dichloroethane consistent across all regulated sectors.

1,1-Dichloroethane, also called ethylidene chloride, is a colorless, mobile, combustible liquid with an odor like that of chloroform (ACGIH 1986, p. 184; Hawley's 1987, p. 494). This substance is used as a chemical intermediate and also finds limited use as a solvent. It is also used as a fumigant and insecticide (HSDB 1990). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Dichloroethane is a narcotic and liver and kidney toxin; exposure to high concentrations may also cause lung damage. Reproductive effects and tumors have been reported in a few animal studies involving dichloroethane (RTECS 1990). The oral LD_{50} in rats is 725 mg/kg (RTECS 1990). Rats died after exposure to dichloroethane concentrations of 32,000 ppm for 2.5 hours, but reducing the exposure interval to 30 minutes resulted in the survival of all exposed animals (AIHA 1971). Rats, guinea pigs, rabbits, and dogs exposed to 1000 ppm for 7 hours/day for 5 days/week for 6 months showed no gross or microscopic alterations at autopsy; however, a single exposure to concentrations of 8,000 ppm and above for 7 hours caused liver and kidney changes discernible at autopsy (AIHA 1971). Rats inhaling 6000 ppm for 7 hours/day on gestation days 6–15 had litters with an increased incidence of retarded fetal development, including delayed sternabral ossification (Schwetz et al. 1974). An NCI gavage bioassay (NCI 1978) in rats and mice failed to show conclusive evidence of carcinogenicity in these species, although marginal increases in mammary adenocarcinomas and hemangiosarcomas were seen in female rats and female mice developed a statistically significant increase in the incidence of endometrial stromal polyps.

In humans, exposure to very high concentrations of this substance causes deep narcosis, as evidenced by dichloroethane's former use as a human anesthetic (Proctor, Hughes, and Fischman 1988, p. 188). However, no other information on effects in humans is available.

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 100 ppm in agriculture. The Agency believes that this PEL is necessary to protect workers in this sector from experiencing the narcotic and hepatotoxic effects potentially associated with exposure to this substance. OSHA considers these effects material impairments of health and believes that the PEL will substantially reduce the significant risk that agricultural workers will experience these effects. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DIMETHYL ACETAMIDE

CAS: 127–19–5; Chemical Formula: $CH_3CON(CH_3)_2$

H.S. No. 2065

In general industry, construction, and maritime, OSHA's permissible exposure limit for dimethyl acetamide is 10 ppm as an 8-hour TWA, with a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no OSHA limit for dimethyl acetamide in agriculture, and NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. The ACGIH has an 8-hour TLV®–TWA of 10 ppm, with a skin notation, for dimethyl acetamide. OSHA is proposing to establish a permissible exposure limit of 10 ppm as an 8-hour TWA, and a skin notation, for this substance in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all regulated sectors.

Dimethyl acetamide is a colorless liquid with a faint ammonia-like odor (Genium MSDS 1989, No. 458). Dimethyl acetamide finds use as a solvent for plastics, resins, and gums; as a catalyst; as a paint remover; and as a high-purity solvent for crystallization and purification (Hawley's 1987, p. 409). This substance has also been used in human medicine as a neoplastic agent (HSDB 1985).

Dimethyl acetamide causes skin irritation and liver damage in animals and humans; this substance has also been shown to be a teratogen in animals. The oral LD_{50} in rats is 5000 mg/kg; for mice, it is 4620 mg/kg (RTECS 1987). The dermal LD_{50} in rabbits is 2240 mg/kg (RTECS 1987). Rabbits experienced mild skin irritation after contact of the liquid with the skin for 24 hours. Dermal application of 9600 mg/kg dimethyl acetamide caused behavioral symptoms and pulmonary and liver effects in mice, indicating that this substance can be absorbed through the skin in toxic amounts (RTECS 1987). Dimethyl acetamide caused corneal necrosis when applied to the rabbit eye in undiluted form (Smyth 1962). Rats showed nasal irritation, an increase in blood cholesterol, and liver hypertrophy after being exposed to a 288-ppm concentration for 6 hours/day for 2 weeks; there was also evidence of testicular atrophy 2 weeks after exposure ceased (Kelley et al. 1984). Dogs exposed to repeated dermal applications of 4 mg/kg for 6 weeks exhibited severe fatty infiltration of the liver at autopsy (Kelley et al. 1984). Rats repeatedly exposed to a concentration of 195 ppm for 6 months showed focal necrosis of the liver at post mortem; when the concentration was reduced to 40 ppm for 6 months, no adverse effects were seen (Horn 1961). A reproductive study in male rats involving inhalation exposure to 40, 120, or 400 ppm dimethyl acetamide for 6 hours/day, 5 days/week for 43 exposures failed to find reproductive effects in any of the animals tested; however, significant increases in liver weight and in liver/body weight ratios were seen in animals in the 120- and 400-ppm groups (Wang, Kier, and Pounds 1989). Teratogenic effects (such as encephalocele and diffuse subcutaneous edema) were observed among fetuses of rats that had been dermally exposed on gestation days 10 and 11 to a total dimethyl acetamide dose of 2400 mg/kg (Stula and Krauss 1977). Developmental abnormalities also occurred in the fetuses of rabbits given oral dimethyl acetamide doses of 1839 mg/kg during days 6 to 18 of gestation (RTECS 1987).

The signs and symptoms of oral exposure to this substance in humans include depression, lethargy, hallucinations, and jaundice (Proctor, Hughes, and Fischman 1988, p. 206). Patients given oral doses of 400 mg/kg daily showed depression, lethargy, confusion, and disorientation after 3 days (Weiss et al. 1962). Skin absorption is believed to have contributed substantially to the development of the jaundice seen in workers repeatedly exposed to a 20 to 25 ppm concentration of dimethyl acetamide (Johnson 1961, in ACGIH 1986, p. 205). According to the ACGIH, "the dermal factor is considered in practice to be so significant that no air concentration, however low, will provide protection if skin contact with dimethyl acetamide is permitted" (ACGIH 1986, p. 205).

Based on the evidence presented above, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing the eye and skin irritation and liver damage associated with exposure to dimethyl acetamide. The Agency believes that the proposed 8-hour TWA PEL of 10 ppm, with a skin notation, is necessary to substantially reduce these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DIOXANE

CAS: 123–91–1; Chemical Formula: $O(CH_2CH_2)_2O$

H.S. No. 1145

In construction and maritime, OSHA's PEL for dioxane is 100 ppm as an 8-hour TWA, with a skin notation. There is no PEL in agriculture. The ACGIH has an 8-hour TLV®–TWA of 25 ppm for this substance, with a skin notation. NIOSH has a recommended exposure limit of 1 ppm as a 30-minute ceiling for dioxane but concurs (Ex. 8–47, Table N6A) that the PEL being proposed by OSHA is appropriate. OSHA is proposing a 25-ppm 8-hour TWA PEL for dioxane, with a skin notation, in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Dioxane is a colorless liquid with an ethereal odor. It is an ingredient in fumigants, adhesives, cosmetics, deodorants, polishes, and emulsions. It is also used as a wetting and dispersing agent in textile processing, dye-baths, and stain and printing compositions (HSDB 1985). Dioxane also finds use as a solvent and a stabilizer in chlorinated solvents (HSDB 1985; Proctor, Hughes, and Fischman 1988, p. 219). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Dioxane is an irritant of the eyes, skin, and mucous membranes as well as a liver and kidney toxin and carcinogen in experimental animals. The oral LD_{50} in rats is 4200 mg/kg, and the LC_{50} in the same species is 46 g/m^3 (approximately 13,000 ppm) for 2 hours (RTECS 1990). In rabbits, the median lethal dose by percutaneous absorption is 7600 mg/kg (RTECS 1990). Before death, acutely poisoned animals showed weakness, depression, incoordination, and coma (Clayton and Clayton 1982, p. 3947). Autopsy revealed histologic changes to the liver and kidneys of experimental animals (Clayton and Clayton 1982, p. 3948). Applied to the eyes of rabbits, dioxane caused mild, transient injury of the cornea, graded 4 on an ascending severity scale of 1 to 10 (Grant 1974, p. 417). The liquid causes irritation on repeated contact with the skin of rabbits; the degree of irritation is described as mild (Clayton and Clayton 1982, p. 3948; RTECS 1990). A 2-year drinking water study conducted by the Dow Chemical Company (1972b, as cited in ACGIH 1986/Ex. 1–3, p. 217), in which male and female rats were given water containing 1.0, 0.1, or 0.01 percent dioxane, showed that animals given the highest dose developed liver and nasal tumors, in addition to pathological changes in the liver and kidney. Rats in the 0.1-percent group showed renal tubular sloughing and hepatocellular degeneration but no significant increase in neoplasms. Because this study demonstrated hepato- and nephrotoxic effects at doses 10 times lower than the dose causing cancer in animals, the proposed permissible exposure limit reflects a level that will prevent dioxane's liver and kidney effects; this

level will also reduce carcinogenic effects. A study by Torkelson et al. (1974/Ex. 1–111) in four species of animals exposed to multiple daily airborne exposures of dioxane at 50 ppm showed no gross or histopathologic organ changes; this study demonstrates that the proposed 25-ppm level should protect against the risk of liver and kidney effects.

There is evidence in humans that dioxane is an irritant and a toxin to the liver and kidney. Human volunteers reported experiencing mild eye, nose, and upper respiratory tract irritation at dioxane vapor concentrations of 300 ppm for 15 minutes; when the concentration was increased to 1600 ppm for 10 minutes, the volunteers reported burning and tearing of the eyes (Proctor, Hughes, and Fischman 1988, p. 219). A 1-minute exposure to a 5500-ppm concentration induced vertigo (Proctor, Hughes, and Fischman 1988, p. 219). Five fatalities occurred when humans were severely overexposed (concentration not specified) for a 5-week period; before death, these individuals experienced stomach pain, vomiting, loss of appetite, scanty urine, anuria, and coma (Proctor, Hughes, and Fischman 1988, p. 220). Autopsy revealed liver and kidney damage and edema of the lungs and liver (Proctor, Hughes, and Fischman 1988, p. 220). Similar signs and symptoms were seen in another case that resulted in death: the individual involved is estimated to have been exposed for 1 week to concentrations ranging from 208 to 605 ppm (and perhaps higher) and to have had concurrent dermal exposure (Proctor, Hughes, and Fischman 1988, p. 220). A mortality study of workers exposed to dioxane at concentrations described as low was inconclusive in terms of dioxane's carcinogenicity in humans (IARC 1982, p. 121).

In the prior rulemaking, several commenters noted dioxane's carcinogenic potential in animals and urged OSHA to designate dioxane as a carcinogen (Exs. 8–47, Tr. 3–96, 3–97; Tr. 9–217, 9–218). OSHA is aware that the International Agency for Research on Cancer (IARC) has classified dioxane as a Group 2B (possible human) carcinogen based on its finding that the evidence of this substance's carcinogenicity in animals is sufficient to warrant this designation. As discussed earlier in this preamble, OSHA's use of a particular target organ classification for a substance is not meant to suggest that the chemical in question is without other adverse health effects. In the case of dioxane, for example, OSHA is aware that exposure causes sensory irritation, liver damage, kidney damage, and the following cancers in experimental animals: liver adenomas and carcinomas in rats, hepatomas in guinea pigs, nasal cavity carcinomas in rats, and gall bladder carcinomas in guinea pigs (IARC 1982, p. 121). The classification of dioxane in the liver toxin section of this preamble is intended only to reflect the fact that the level of the proposed PEL, i.e., 25 ppm, is believed to be a level that will protect against dioxane's hepatotoxic effects.

Thus, OSHA preliminarily concludes that dioxane poses a significant risk of liver and kidney damage, and perhaps of cancer, to exposed workers. The Agency believes that an 8-hour TWA limit of 25 ppm for dioxane, with a skin notation, is necessary to protect workers in construction, maritime, and agriculture against these significant risks of material health impairment and that this limit will substantially reduce this significant risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ETHYLENE DICHLORIDE

CAS: 107–06–2; Chemical Formula: $ClCH_2CH_2Cl$

H.S. No. 1168

The OSHA PEL for ethylene dichloride (EDC) in construction and maritime is 50 ppm as an 8-hour TWA. In 1980, the ACGIH reduced its TLV® for ethylene dichloride to 10 ppm as an 8-hour TWA. NIOSH has a recommended exposure limit (REL) of 1 ppm TWA and 2 ppm as a 15-minute ceiling limit and has designated EDC a potential human carcinogen. NIOSH also concurs (Ex. 8–47, Table N6A) with the limit being proposed. OSHA is proposing an 8-hour TWA limit of 1 ppm and a STEL of 2 ppm for ethylene dichloride in construction, maritime, and agriculture. These are the limits recently established for these substances in general industry.

Ethylene dichloride is a colorless liquid with a chloroform-like odor. EDC is registered in the United States for agricultural use as a post-harvest grain fumigant and a fumigant for use in orchards, agricultural premises, and mushroom houses (HSDB 1985). This substance also is used as a cleaner of leather and rubber goods and as a solvent, particularly in the metal cleaning industries (HSDB 1985). Its major uses are in the production of vinyl chloride and as an antiknock additive in gasolines (EPA 1987). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Ethylene dichloride causes narcosis in animals on acute exposure (EPA 1987) and has also been shown to cause mutations in bacterial test systems with microsomal activitation (EPA 1987; RTECS 1990). In contact with the skin of rabbits, liquid EDC causes mild irritation; in contact with the eye, this substance causes severe irritation (RTECS 1990). The oral LD_{50} in rats is 670 mg/kg, and the LC_{50} in the same species is 1000 ppm for 7 hours (RTECS 1990). The dermal LD_{50} in rabbits is 2800 mg/kg (RTECS 1990). Animals acutely poisoned by inhalation showed weakness, incoordination, restlessness, irregular breathing, and coma before death (EPA 1987). Dogs, monkeys, rabbits, rats, mice, and guinea pigs were exposed to varying concentrations of EDC for 7 hours/day, 5 days/week for 8 months; maximal no-effect levels were 400 ppm (dogs), 200 ppm (monkeys and rabbits), and 100 ppm (rats, mice, and guinea pigs) (Hayes 1982, p. 151). Other studies (Spencer et al. 1951; Hoffmann et al. 1971) have shown similar results.

Several studies indicate that the current OSHA PEL is insufficient to protect workers in construction, maritime, and agriculture against the toxic effects of exposure to EDC. A paper by Kozik (1957/Ex. 1–182) reported that workers generally exposed to ethylene dichloride at levels of 10 to 15 ppm but occasionally exposed to levels of 40 ppm experienced increased morbidity, diseases of the liver and bile ducts, and nervous system effects. Kozik investigated the effect of EDC on the speed of visual-motor reaction and on the number of errors made. There was no difference in reaction speed between the 17 gluers (exposed) and 10 machinists (control) before and after work. However, more errors were committed by the gluers (15 out of 17) than by controls (4 out of 10). These errors reflected disturbances of equilibrium and mobility of the nerve processes during a complicated reaction. Kozik also noted that, although the reaction errors had ceased by the beginning of the next working day in the machinists, they persisted in the gluers. The author also compared the muscular strength and endurance of the upper extremities in workers in the two groups. Both were decreased in the gluers, and both strength and endurance decreased with job longevity in these workers. The author attributed these distinct shifts in the functional state of the motor apparatus of the upper extremities to exposure to EDC. Eighty-three gluers were examined for diseases of the motor apparatus of the upper extremities. Neuromyalgia, myofasciculitis,

tendovaginitis, or angioneurosis was found in 67.7 percent of the subjects. Diseases of the liver and bile ducts were found in 19. The author concluded that "prolonged exposure to EDC vapors in concentrations close to the maximum permissible concentration or only slightly exceeding it causes pronounced alterations on the part of the liver and nervous system, which are typical of EDC poisoning." In addition, Brzozowski and associates (1954/Ex. 1–63) reported abnormal changes in the blood of 50 percent of workers (8 of 16) exposed to EDC levels of 10 ppm and above (Brzozowski, Czajka, Dutkiewicz et al. 1954/Ex. 1–63).

In the prior rulemaking, many commenters were of the opinion that a permissible exposure limit of 10 ppm, rather than the proposed 1-ppm limit, would provide adequate protection against EDC's hepatotoxic, central nervous system, and hematopoietic effects (Exs. 3–624, 3–677, 3–678, 3–741, 3–874, and 3–1174). In response to these commenters, OSHA noted (54 *FR* 2484–2485) that many studies report serious EDC-related effects among workers exposed to airborne concentrations in the 10- to 15-ppm range. For example, the aircraft workers in the Kozik (1957/Ex. 1–182) study (average 8-hour TWA exposures of 10 to 15 ppm) experienced increased morbidity and lost more workdays than did non-EDC-exposed workers at the same factory. These workers experienced high rates of gastrointestinal disease and liver and gallbladder diseases; these symptoms and diseases are typical EDC exposure effects. Another study (Cetnarowicz 1959) examined refinery workers exposed to EDC at levels ranging from 10 to 200 ppm and found that these workers experienced many of the same symptoms as those experienced by the aircraft workers. Clinical analyses confirmed that the liver and gastrointestinal tract were the principal target organs affected by EDC exposure. Rosenbaum (1947) also reported that EDC exposures below 25 ppm (not further specified) caused functional nervous system disorders, including headache, insomnia, and fatigue, and also slowed the heartbeat rate in affected workers. In the general industry rulemaking, OSHA concluded that the evidence presented in these studies was consistent, biologically plausible, and convincing and noted that the symptoms and signs of EDC's effects had been confirmed both clinically (palpitation of enlarged livers, X-ray evidence of pyloric spasms) and by laboratory analysis (elevated urobilinogen levels, positive Takata-Ara liver function tests, negative glucose tolerance tests).

In the earlier Air Contaminants rulemaking, there was considerable discussion of EDC's carcinogenicity, with some commenters (Exs. 3–677, 3–741, and 3–874) arguing that inhalation exposures to EDC in animals had not produced a statistically significant increase in tumors in exposed rats or mice. In response to these commenters, OSHA noted that NIOSH, EPA, and the International Agency for Research on Cancer (IARC) have all concluded that EDC is carcinogenic in animals. IARC summarized its findings as follows:

> 1,2-Dichloroethane [EDC] was tested in one experiment in mice and in one in rat by oral administration. In mice, it produced benign and malignant tumors of the lung and malignant lymphomas in animals of both sexes, hepatocellular carcinomas in males and mammary and uterine adenocarcinomas in females. In rats, it produced carcinomas of the forestomach in male animals, benign and malignant mammary tumors in females, and hemangiosarcomas in animals of both sexes (IARC 1979, Vol. 20, p. 442).

These effects are severe, and evidence demonstrates that they occur at concentrations of about 10 ppm. A no-effect level has not been established for EDC. Accordingly, a substantial safety factor is appropriate and the 1 ppm level is necessary based on hepatotoxic effects alone; this limit provides, at most, a safety factor of 10. There is also evidence of EDC's carcinogenicity; however, OSHA is not regulating this substance on the basis of its carcinogenicity at this time. Nevertheless, carcinogenicity is an additional reason for promulgating a limit that is a factor of 10 below the lowest observed hepatotoxic effect level.

Based on this evidence, the Agency preliminarily concludes that an 8-hour TWA limit of 1 ppm and a 15-minute STEL of 2 ppm are necessary to protect workers in construction, maritime, and agriculture against the significant risks of liver damage, gastrointestinal toxicity, and cancer that are potentially associated with exposure to ethylene dichloride. OSHA believes that the proposed limits are necessary to substantially reduce these significant occupational risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

HAFNIUM

CAS: 7440–58–6; Chemical Formula: Hf

H.S. No. 2087

In general industry, construction, and maritime, OSHA's permissible exposure limit for hafnium is 0.5 mg/m^3 as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.5 mg/m^3 for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the PEL being proposed. OSHA is proposing an 8-hour TWA PEL in agriculture of 0.5 mg/m^3 for hafnium. Promulgation of this limit will make the PEL for hafnium consistent across all OSHA-regulated sectors.

Hafnium is a gray, ductile metal with a brilliant silver-like luster. It is used for control rods in water-cooled nuclear reactors and in light bulb filaments, electrodes, special glasses, and vacuum tubes (ACGIH 1986, p. 293; Hawley's 1987, p. 583).

Hafnium and its compounds are irritants of the eye and skin in humans and animals. Studies also indicate that hafnium and its compounds cause liver damage in animals (Haley 1962). The LD_{50} (route unreported) for hafnium in mice is 76 mg/kg (RTECS 1990). Rabbits showed transient eye irritation when 1 mg of hafnium chloride was placed into their eyes. In contact with unabraded skin, hafnium chloride caused transient edema and erythema in rabbits; application of this substance to abraded skin caused ulceration (Haley 1962). Rats fed hafnium in the diet at a 1 percent level showed slight changes in the liver at autopsy (Chemical Safety Data Sheet 1966, in Proctor, Hughes, and Fischman 1988, p. 267). There are no data on the toxicity of hafnium in humans.

Based on this evidence in animals, OSHA preliminarily concludes that hafnium and its compounds potentially cause eye and skin irritation and liver damage. OSHA believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these effects. The Agency believes that establishing an 8-hour TWA limit of 0.5 mg/m^3 will substantially reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

HEXACHLORONAPHTHALENE

CAS: 1335–87–1; Chemical Formula: $C_{10}H_2Cl_6$

H.S. No. 2089

In general industry, construction, and maritime, OSHA currently has an 8-hour TWA limit for hexachloronaphthalene of 0.2 mg/m^3, with a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. OSHA has no PEL for hexachloronaphthalene in agriculture. The ACGIH's current TLV®–TWA for this substance is 0.2 mg/m^3,

with a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the PEL being proposed. OSHA is proposing a TWA PEL of 0.2 mg/m³, with a skin notation, for hexachloronaphthalene in agriculture. Promulgation of this limit will make the PEL for hexachloronaphthalene consistent across all OSHA-regulated sectors.

Hexachloronaphthalene is a light yellow or white waxy solid with an aromatic odor. In commercial use, the chloronaphthalenes occur as mixtures of naphthalenes having varying degrees of chlorination. Hexachloronaphthalene is used in flameproofing and waterproofing; to insulate electrical equipment; and as a lubricant additive (ACGIH 1986, p. 302; Hazardous Substance Fact Sheet 1985, p. 1).

Hexachloronaphthalene causes liver damage and a severe form of dermatitis called chloracne (Proctor, Hughes, and Fischman 1988, p. 270). In rats, repeated exposure to an average concentration of 8.9 mg/m³ of a penta- and hexachloronaphthalene mixture resulted in poor growth, jaundice, and death. There was marked fatty degeneration and centrilobular necrosis of the liver at autopsy. Minor liver injury also occurred at repeated exposures to a concentration of 1.16 mg/m³ (Irish 1963, in Clayton and Clayton 1982, p. 3670–3671). Rabbits intraperitoneally injected with a 15 mg/day dose of a penta- and hexachloronaphthalene mixture for 12 to 26 days developed liver disease (HSDB 1985).

Acute yellow atrophy of the liver has been reported in workers repeatedly exposed to a mixture of penta- and hexachloronaphthalene at concentrations averaging 1 to 2 mg/m³. These workers also experienced symptoms of nausea, indigestion, weight loss, and jaundice (Hygienic Guide Series 1966; Elkins 1959). Long-term skin contact with the fume, dust, or hot vapor of the higher chlorinated naphthalenes causes chloracne, which is slow to develop and may take months to disappear (Irish 1963, in Clayton and Clayton 1982, p. 3672–3673).

Based on this evidence in humans and animals, OSHA preliminary concludes that exposure to hexachloronaphthalene causes chloracne and liver damage and that, in the absence of a limit, workers in agriculture are at risk of experiencing these adverse health effects. The Agency believes that the proposed 8-hour TWA limit of 0.2 mg/m³, and a skin notation, are necessary to substantially reduce these significant risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

HYDRAZINE

CAS: 302–01–2; Chemical Formula: $H_2N–NH_2$

H.S. No. 1205

In construction and maritime, OSHA's current limit for hydrazine is 1ppm as an 8-hour TWA, with a skin notation. There is no limit in agriculture. OSHA is retaining the skin notation and proposing an 8-hour TWA PEL of 0.1 ppm for hydrazine in construction, maritime, and agriculture. Because of hydrazine's potential carcinogenic hazard, NIOSH (1978e/Ex. 1–263; Ex. 8–47) recommends that workplace exposures not exceed 0.03 ppm, as measured over 2 hours; this level represents the lowest detectable concentration over this sampling interval. The 1987–88 ACGIH TLV® for hydrazine is 0.1 ppm as an 8-hour TWA, with a skin notation, and the ACGIH also designates this substance a potential human carcinogen. Promulgation of the proposed limit will make the PEL for hydrazine consistent across all regulated sectors.

Hydrazine is an odorless, fuming, oily liquid with an ammonia-like odor. Hydrazine is used as a high-energy rocket fuel, a reducing agent, an oxygen scavenger for boiler waters, and a chemical intermediate (ACGIH 1986, p. 310(89)). It is also used in the preparation of agricultural chemicals and pharmaceuticals (Hawley's 1987, p. 612).

Hydrazine has many toxic effects in humans and experimental animals. Humans exposed to hydrazine vapors experience immediate irritation of the nose and upper respiratory tract as well as nausea and dizziness (Proctor, Hughes, and Fischman 1988, p. 273). Severe overexposure causes a temporary blindness that may persist for 24 hours. In contact with the skin or eyes, liquid hydrazine causes severe burns (Proctor, Hughes, and Fischman 1988, p. 273). Skin sensitization has also been reported in humans (ACGIH 1986, p. 310.1(89)). Hydrazine has been absorbed through the skin in humans to cause systemic effects, manifested as weakness, tremor, and vomiting; in one fatal case of occupational overexposure, nephritis and liver damage were seen at autopsy (Proctor, Hughes, and Fischman 1988, p. 273).

In experimental animals, hydrazine causes moderate to severe irritation of the eyes, skin, and mucous membranes, induces convulsions, produces hemolysis, damages the liver, and causes cancer (ACGIH 1986, pp. 310(89), 310.1(89); RTECS 1990; Proctor, Hughes, and Fischman 1988, p. 272). The oral LD_{50} in rats is 60 mg/kg, and the LC_{50} in the same species is 570 ppm for 4 hours (RTECS 1990). The dermal LD_{50} in rabbits is 91 mg/kg (RTECS 1990). Animals acutely poisoned by dermal absorption show fatty degeneration of the liver, anemia, destruction of erythrocytes, loss of appetite, weakness, vomiting, convulsions, and weakness (ACGIH 1986, p. 310(89)). Lethal exposures by inhalation cause pathological changes in the liver, kidneys, and lungs that are visible at autopsy (ACGIH 1986, p. 310(89)). A hepatotoxic response in mice, anemia and weight loss in dogs, and a dose-related depression in growth rate in rats occurred after a 6-month exposure to 1 ppm of hydrazine for 6 hours per day, 5 days per week, or to a 0.2-ppm concentration continuously (Haun and Kinkead 1973/Ex. 1–824).

The ACGIH has assigned an A2 designation (suspected human carcinogen) to hydrazine, based on a study by MacEwen, Vernot, and Haun (1979/Ex. 1–193) showing significant increases in nasal tumors in rats exposed to 1 or 5 ppm hydrazine, in thyroid adenocarcinomas in rats exposed to 5 ppm, and in lung adenomas among mice exposed to 1 ppm. NIOSH also considers hydrazine a potential human carcinogen (Ex. 8–47, Table N6B), as does the International Agency for Research on Cancer (IARC 1987, p. 224). The animal studies conducted by Haun and Kinkead (1973/Ex. 1–824) and by MacEwen, Vernot, and Haun (1979/Ex. 1–193) clearly demonstrate that exposure to hydrazine at the current 1-ppm PEL presents a significant risk of liver disease, respiratory cancer, and adverse blood effects; animals exposed to airborne concentrations at this level have exhibited all of these responses.

In the prior rulemaking, some commenters (Exs. 8–16, 194; Tr. 9–218; Tr. 3–309) questioned the classification of hydrazine in the liver target organ section of the preamble, believing that it should be placed in the cancer section instead. However, as discussed elsewhere in this preamble, OSHA does not intend this classification scheme to have regulatory implications and has adopted it only to facilitate generic rulemaking.

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 0.1 ppm, with a skin notation, for hydrazine. OSHA preliminarily concludes that this limit will substantially reduce the significant risks of liver disease, cancer, and hematopoietic effects that have been demonstrated to occur in animals at exposures above the proposed PEL.

All of these effects clearly constitute material health impairments, and OSHA believes that the proposed PEL will substantially reduce this significant risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

METHYLCYCLOHEXANOL

CAS: 25639–42–3; Chemical Formula: $CH_3C_6H_{10}OH$

H.S. No. 1269

In construction and maritime, OSHA currently has an 8-hour TWA limit of 100 ppm for methylcyclohexanol. There is no PEL in agriculture. The Agency is proposing an 8-hour TWA of 50 ppm as the PEL for this substance in construction, maritime, and agriculture; this is the 1987–1988 ACGIH TLV® for methylcyclohexanol. NIOSH has no REL for this substance but concurs (Ex. 8–47, Table N1) with OSHA's proposed limits for methylcyclohexanol. This is the limit recently established for this substance in general industry.

Methylcyclohexanol is a colorless, viscous liquid with an aromatic odor; in industrial use, it takes the form of a mixture of isomers in which the meta and para forms predominate. Methylcyclohexanol finds use as a degreasing agent, a solvent for esters and ethers, a blending agent for soaps and detergent, and a solvent for gums, oils, resins, and waxes (ACGIH 1986, p. 385; Clayton and Clayton 1982, p. 4650). It is also an ingredient in soap-based spot removers and a chemical intermediate for lubricating oil additives (HSDB 1985).

In addition to liver injury, exposure to methylcyclohexanol causes eye, nose, and mucous membrane irritation and, at high concentrations, narcosis (Proctor, Hughes, and Fischman 1988, p. 338). The oral LD_{50} in rats is 1660 mg/kg (RTECS 1988). The median lethal dose in rabbits by dermal application is 6.8 to 9.4 g/kg (Clayton and Clayton 1982, p. 4651). Animals acutely poisoned via dermal exposure showed tremors, hypothermia, and narcosis (Clayton and Clayton 1982, p. 4651). The vapor causes signs of irritation in rabbits at concentrations of approximately 500 ppm (Grant 1986, p. 614). Repeated inhalation exposures to the vapor caused salivation, eye irritation, and lethargy in rabbits exposed to a 500-ppm concentration, but exposures to 230 ppm caused no observable effects. Fifty 6-hour exposures to a concentration of 120 ppm caused microscopic changes in the liver and kidney tissue of rabbits that were visible at autopsy (Treon, Crutchfield, and Kitzmiller 1943b/Ex. 1–394).

In humans, headaches and eye and respiratory irritation have been reported after prolonged exposures to high (not further specified) concentrations of methylcyclohexanol (Fillipi 1914, as cited in ACGIH 1986/Ex. 1–3, p. 385). Several workers exposed to a solvent that contained methylcyclohexanol at unspecified concentrations had a significantly reduced number of leukocytes and one worker had slight lymphocytosis (Clayton and Clayton 1982, p. 4652).

Based on this evidence of methylcyclohexanol's toxicity in humans and animals, OSHA is proposing an 8-hour TWA limit of 50 ppm for this substance in construction, maritime, and agriculture. OSHA preliminarily concludes that this limit will protect workers in these sectors against the significant risks of hepatic and renal damage and narcosis that are associated with exposures to this substance at concentrations above the proposed PEL. The Agency believes that the proposed limit will substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

NITROETHANE

CAS: 79–24–3; Chemical Formula: $CH_3CH_2NO_2$

H.S. No. 2118

The OSHA PEL for nitroethane in general industry, construction, and maritime is 100 ppm as an 8-hour TWA. There is no limit in agriculture for nitroethane. The ACGIH has a TLV®–TWA of 100 ppm for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. The Agency is proposing an 8-hour TWA limit of 100 ppm for nitroethane in agriculture. Promulgation of this limit will make the PEL for nitroethane consistent across all OSHA-regulated sectors.

Nitroethane is a colorless, oily liquid with a faint chloroform-like, fruity odor (ACGIH 1986, p. 434; HSDB 1985). Nitroethane is used as a solvent, a fuel additive, an intermediate in chemical synthesis, and in propellant research (Hawley's 1987, p. 828; ACGIH 1986, p. 434).

Nitroethane is an irritant of the eyes and mucous membranes in both animals and humans; in animals, it is a hepatotoxin and, at high concentrations, a narcotic. The oral LD_{50} in rats is 1100 mg/kg, and the LC_{50} in mice is 19,500 mg/m³ for 2 hours (RTECS 1987). Death occurred in rabbits exposed to 5000 ppm nitroethane for a period of 3 hours. Rabbits exposed to half of that concentration (2500 ppm) for the same duration survived. Exposure to nitroethane concentrations above 5000 ppm caused irritation of the eyes and mucous membranes, labored breathing, pulmonary rales, and, in some rabbits, pulmonary edema. Some animals also exhibited brief convulsions. Mild to severe liver damage and some kidney damage was observed at autopsy in animals exposed to lethal concentrations of nitroethane (Machle, Scott, and Treon 1940; AIHA 1978). Exposure of monkeys and various laboratory animal species to a 500-ppm concentration of nitroethane caused no observable effects (Mackle, Scott, and Treon 1940).

Toxicity data on humans exposed to nitroethane are limited. Nitroethane is reported to cause irritation of the eyes and mucous membranes (Merck 1983, p. 857). There are no reports of chronic effects in humans but, based on effects seen in animals, exposure to high concentrations may cause liver damage. Prolonged skin contact with nitroethane causes irritation and dermatitis (Proctor, Hughes, and Fischman 1988, p. 372).

Based on this evidence, OSHA is proposing a 100 ppm 8-hour TWA limit to protect workers in agriculture from the significant risks of irritation and liver damage caused by exposure to higher concentrations of nitroethane. The Agency preliminarily concludes that this limit is necessary to substantially reduce a significant risk of material health impairment in exposed workers. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

NITROGEN TRIFLUORIDE

CAS: 7783–54–2; Chemical Formula: NF_3

H.S. No. 2119

In general industry, construction, and maritime, OSHA's permissible exposure limit for nitrogen trifluoride is 10 ppm as an 8-hour TWA. There is no limit for this substance in agriculture. The ACGIH has an 8-hour TLV®–TWA of 10 ppm for nitrogen trifluoride; NIOSH has no REL but concurs with the limit being proposed (Ex. 8–47, Table N3A). OSHA proposes to establish an 8-hour TWA limit of 10 ppm for this substance in agriculture. Promulgation of this limit will make the PEL for nitrogen trifluoride consistent across all OSHA-regulated sectors.

Nitrogen trifluoride is a colorless gas with a moldy odor. This substance is used as an oxidizer for high energy fuels, in the preparation of tetrafluorohydrazine, and in the fluorination of fluorocarbon olefins

(ACGIH 1986, p. 437; Braker and Mossman 1980, p. 538).

Acute exposure to nitrogen trifluoride causes methemoglobinemia, and chronic exposure causes kidney and liver injury. The 1-hour LC_{50}s in rats and monkeys are 6700 ppm and 7500 ppm, respectively (RTECS 1987). Rats exposed to a 10,000-ppm concentration of nitrogen trifluoride for 60 to 70 minutes died; methemoglobin levels had reached 60 to 70 percent (Dost, Reed, and Wang 1970). Acutely poisoned animals showed the following signs and symptoms: severe respiratory distress, cyanosis, incoordination, collapse, and convulsions (Torkelson et al. 1962). Dogs exposed to a 9600-ppm concentration of nitrogen trifluoride for 60 minutes showed Heinz body anemia; reduced hematocrit, hemoglobin, and red blood cell counts; and signs of anoxia (Vernot, Haun, MacEwen, and Egan 1973). Rabbits given nine intraperitoneal injections of nitrogen trifluoride showed grossly enlarged spleens, liver damage, and myocardial degeneration at autopsy (ACGIH 1986, p. 437). Autopsy showed mild to moderate liver and kidney changes in rats exposed to a 100-ppm concentration of nitrogen trifluoride for 7 hours/day for 4.5 months; no hematologic changes were seen in these subchronically exposed animals (Torkelson et al. 1962).

There are no reported cases of industrial poisoning with nitrogen trifluoride.

Based on this evidence, OSHA preliminarily concludes that nitrogen trifluoride causes methemoglobinemia and may cause liver and kidney injury in exposed workers. The Agency believes that the proposed limit is necessary to reduce the significant risk posed to agriculture workers exposed to this substance. OSHA believes that establishing an 8-hour TWA limit of 10 ppm is necessary to substantially reduce the risk that agricultural workers will experience these material health impairments. Promulgation of the proposed limit will make the PEL for nitrogen trifluoride consistent across all OSHA-regulated sectors.

OCTACHLORONAPHTHALENE
CAS: 2234–13–1; Chemical Formula: $C_{10}Cl_8$
H.S. No. 1295

In construction and maritime, OSHA currently has an 8-hour TWA limit of 0.1 mg/m³, with a skin notation, for octachloronaphthalene. There is no PEL in agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) that these limits are appropriate. The 1987–1988 ACGIH TLV®s for octachloronaphthalene are 0.1 mg/m³ and 0.3 mg/m³ as 8-hour TWA and 15-minute STEL limits, respectively. In construction and maritime, the Agency is retaining the 8-hour TWA limit and the skin notation and is proposing to add a STEL of 0.3 mg/m³ for this substance. OSHA is also proposing to extend both limits to agriculture. Promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

Octachloronaphthalene is a nonflammable, pale yellow, waxy solid that contains 70 percent chlorine. The chlorinated naphthalenes are highly toxic to the liver by several routes of administration: ingestion, inhalation, and percutaneous absorption (Clayton and Clayton 1982, p. 3670). The liver damage caused by these substances is termed acute yellow atrophy, and there is general agreement that the toxicity of the naphthalenes increases with the compound's degree of chlorination (Sikes, Wise, and Bridges 1952, Ex. 1–804; Clayton and Clayton 1982, p. 3671). In animals, exposure to the chloronaphthalenes causes acne-like lesions that itch severely (Patty 1963g/Ex. 1–845), and human volunteers exposed dermally to the penta- and hexachloronaphthalenes also showed this response (Proctor, Hughes, and Fischman 1988, p. 384). However, human volunteers exposed dermally to octachloronaphthalene failed to develop acne (Proctor, Hughes, and Fischman 1988, p. 384). Several deaths and many nonfatal cases of liver disease have been reported in workers occupationally exposed to the heated vapors of chlorinated naphthalene (specific chloronaphthalene compound not identified) (AIHA 1966).

Based on this evidence, OSHA is retaining the 8-hour TWA PEL of 0.1 mg/m³ and the skin notation and is proposing to add a STEL of 0.3 mg/m³ for octachloronaphthalene in construction and maritime; the Agency is also proposing to extend both limits to agriculture. The Agency preliminarily concludes that the TWA and STEL limits are necessary to protect workers in construction, maritime, and agriculture against the significant risks of serious liver damage associated with exposure to this substance. OSHA considers liver damage a material health impairment. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

PENTACHLORONAPHTHALENE
CAS: 1321–64–8; Chemical Formula: $C_{10}H_3Cl_5$
H.S. No. 2123

OSHA's permissible exposure limit for pentachloronaphthalene in general industry, construction, and maritime is 0.5 mg/m³ as an 8-hour TWA; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit in agriculture. The ACGIH has a TLV®-TWA limit of 0.5 mg/m³ for pentachloronaphthalene. NIOSH has no REL but concurs with the limit being proposed (Ex. 8–47, Table N3A). OSHA is proposing to establish a TWA PEL of 0.5 mg/m³, with a skin notation, for pentachloronaphthalene in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Pentachloronaphthalene is a pale yellow solid with an aromatic odor similar to that of mothballs. Pentachloronaphthalene is used in synthetic waxes and electrical insulating materials, as a lubricant, and as a coating for wood, paper, and textiles (Hazardous Substance Fact Sheet 1986, p. 1; HSDB 1984). The chloronaphthalenes are used in commercial products as mixtures of naphthalenes of varying degrees of chlorination; the penta-, hexa-, hepta-, and octachloronaphthalenes are considered more toxic than the mono- or trichloronaphthalenes.

Pentachloronaphthalene causes chloracne and liver damage in humans and animals. When this substance was applied to rabbit skin, the skin underwent epithelial hyperplasia, inflammatory and degenerative changes, and, finally, regenerative processes (Adams et al. 1941, in Clayton and Clayton 1981, p. 3671). Rats fed a mixture of penta- and hexachloronaphthalene at a rate of 3 g/day for 1 month showed weight loss, severe liver injury, and were sick; nine out of 10 of the animals died (Drinker et al. 1937, in Clayton and Clayton 1981, p. 3670). Exposure to the vapors of a penta- and hexachloronaphthalene mixture at an average concentration of 1.16 mg/m³ caused liver injury in rats; at 8.88 mg/m³, there was poor growth, severe liver injury, and death (Irish 1963).

In humans, exposure to chlorinated naphthalenes containing pentachloronaphthalene as the major constituent has caused numerous cases of chloracne and many cases of liver damage; several of these cases have been fatal (ACGIH 1986, p. 460). Nine reports of toxic hepatitis as a result of exposure to chlorinated naphthalenes were reported by von Oettingen (von Oettingen 1955). Pentachlorinated naphthalenes were also reported to be

the cause of seven cases of toxic hepatitis, two of them fatal (Cotter 1944, in ACGIH 1986, p. 460). Workers handling chlorinated naphthalenes have developed chloracne; both long-term contact with the material or short-term contact with its hot vapors can cause chloracne (Irish 1963). Chloracne is characterized by papules, large comedones and pustules, mainly on the face, neck, arms, and legs. Symptoms, including headache, vertigo, anorexia, and systemic effects, often accompany the chloracne-related skin lesions (Kleinfeld et al. 1972; Greenberg et al. 1939).

Based on this evidence in humans and animals, OSHA preliminarily concludes that pentachloronaphthalene causes skin and liver damage in exposed individuals. In the absence of a limit for this substance, OSHA believes that workers in agriculture are at significant risk of experiencing these adverse effects. OSHA believes that establishing a PEL of 0.5 mg/m³ as an 8-hour TWA, and a skin notation, for pentachloronaphthalene is necessary to substantially reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PROPYLENE DICHLORIDE

CAS: 78–87–5; Chemical Formula: $CH_3CHClCH_2Cl$

H.S. No. 1341

OSHA's limit for propylene dichloride in construction and maritime is 75 ppm as an 8-hour TWA. The 1987–1988 ACGIH TLV®s for propylene dichloride are 75 ppm (8-hour TWA) and 110 ppm (15-minute STEL). NIOSH has no REL for this substance. In construction and maritime, OSHA is retaining an 8-hour TWA of 75 ppm and proposing to add a STEL of 110 ppm. The Agency is also proposing to extend both limits to agriculture. These are the limits recently established for this substance in general industry.

Propylene dichloride is a colorless, flammable, mobile liquid with an odor like that of chloroform. Propylene dichloride (also called 1,2-dichloropropane) is an ingredient of soil fumigants, and a solvent for oils, fats, drycleaning, and metal degreasing (EPA 1987). It is also a chemical intermediate, a scavenging agent for gasoline, and an insecticide for fruits, grains, and livestock (ACGIH 1986, p. 501). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The primary hazards associated with exposure to propylene dichloride are inhalation-induced toxicity to liver tissue, central nervous system depression, and skin and eye irritation. The oral LD_{50} in rats is 1947 mg/kg, and the LC_{50} in the same species is 14 g/m³ for 8 hours (RTECS 1990). The dermal LD_{50} in rabbits is 8750 mg/kg (RTECS 1990). Repeated inhalation exposures to 1000 ppm have been reported to kill dogs (after 24 exposures), guinea pigs (after 22 exposures), and rats (in some cases after only seven exposures); however, some animals survived more than 100 seven-hour exposures. Necropsy showed severe liver damage; the hepatotoxicity of propylene dichloride appears to be greater than that of carbon tetrachloride and less than that of ethylene dichloride (Heppel, Neal, Highman, and Porterfield 1946/Ex. 1–510). Animals of these same species (rats, dogs, and guinea pigs) survived 128 to 140 7-hour exposures to 400 ppm propylene dichloride for 5 days/week without histologic effects, while mice died from similar exposures; surviving mice displayed hepatomas (Heppel, Highman, and Peake 1948/Ex. 1–605). The National Toxicology Program recently completed a carcinogenicity bioassay in mice and rats (NTP 1986c). In male and female mice, a statistically significant and dose-related increase in hepatocellular tumors was seen (IARC 1986, p. 140). The results were inconclusive in female rats and negative in male rats (IARC 1986, p. 140). Based on this evidence, the International Agency for Research on Cancer concluded that there was limited evidence that propylene dichloride is carcinogenic in animals (IARC 1986, p. 140).

Workers inhaling high levels (not further specified) of propylene dichloride showed signs of severe liver damage, hemolytic anemia, and developed acute kidney failure; in these three cases, the onset of symptoms was delayed for more than 24 hours (Proctor, Hughes, and Fischman 1988, p. 424). Short-term exposures to 400- or 500-ppm concentrations caused no apparent effects in workers (Proctor, Hughes, and Fischman 1988, p. 424). In repeated or prolonged contact with the skin, propylene dichloride causes defatting and may lead to dermatitis (Proctor, Hughes, and Fischman 1988, p. 424). Propylene dichloride accidentally sprayed into the eye of a worker caused transient corneal damage (Grant 1986, p. 328).

Because of propylene dichloride's toxicity in humans and animals, OSHA is retaining the 8-hour TWA PEL of 75 ppm, proposing to add a 15-minute STEL of 110 ppm in construction and maritime, and proposing to extend both limits to agriculture. The Agency preliminarily concludes that this combined limit will protect workers in construction, maritime, and agriculture against the significant risks of sensory irritation, hepatotoxic effects, central nervous system depression, and cancer potentially associated with exposures to this substance. OSHA believes that the TWA and STEL will reduce this risk substantially. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

1,1,2,2-TETRACHLOROETHANE

CAS: 79–34–5; Chemical Formula: $CHCl_2CHCl_2$

H.S. No. 1385

In construction and maritime, OSHA's PEL for 1,1,2,2-tetrachloroethane is 5 ppm as an 8-hour TWA, with a skin notation. The 1987–1988 ACGIH TLV® for this substance is 1 ppm as an 8-hour TWA, with a skin notation. NIOSH considers 1,1,2,2-tetrachloroethane a potential human carcinogen but concurs (Ex. 8–47, Table N6A) with the limit being proposed. OSHA is proposing a PEL of 1 ppm as an 8-hour TWA limit, with a skin notation, for tetrachloroethane in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

1,1,2,2-Tetrachloroethane (also called acetylene tetrachloride) is a colorless, nonflammable, heavy, mobile liquid with a sweet, chloroform-like odor. This substance is used as a chemical intermediate, a solvent, and an insecticide (IARC 1979, p. 479). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

1,1,2,2-Tetrachloroethane is toxic both by inhalation and skin absorption (Proctor, Hughes, and Fischman 1988, p. 460). In addition to liver damage, exposure to this substance causes kidney damage and central nervous system depression (Clayton and Clayton 1981, p. 3514). The oral LD_{50} in rats is 800 mg/kg, and the lowest lethal concentration in the same species is 1000 ppm for 4 hours (RTECS 1990). The dermal LD_{50} in rabbits is 6.4 g/kg (Clayton and Clayton 1981, p. 3514). Rats survived a 4-hour exposure to a 500-ppm concentration but died when the concentration was increased to 1000 ppm for the same interval (Clayton and Clayton 1981, p. 3514). Rabbits exposed to a 14.6-ppm concentration for 3 to 4

hours daily for 7 to 11 months showed mild liver damage (Clayton and Clayton 1981, p. 3515). In monkeys exposed to 1000 or 4000 ppm for 2 hours/day for 190 days, marked vacuolation of the liver was seen at autopsy (HSDB 1989). 1,1,2,2-Tetrachloroethane has been tested in carcinogenicity bioassays in mice and rats. In mice of both sexes, this substance caused a significant increase in the incidence of hepatocellular carcinomas; in rats, however, no significant increase was seen (IARC 1987, p. 354).

Workers acutely overexposed to 1,1,2,2-tetrachloroethane on the job have died after experiencing nausea, vomiting, abdominal pain, jaundice, tenderness in the area of the liver, delirium, convulsions, and coma (Proctor, Hughes, and Fischman 1988, p. 460). Two men exposed to 116 ppm for 20 minutes reported dizziness and vomited; when the concentration was increased to 146 ppm, they became dizzy after 10 minutes, experienced mucous membrane irritation after 12 minutes, and reported fatigue after 20 minutes (Proctor, Hughes, and Fischman 1988, p. 460). Occupational exposures to 1,1,2,2-tetrachloroethane at concentrations ranging from 20 to 65 ppm caused nausea, vomiting, abdominal pain, and tremor (Proctor, Hughes, and Fischman 1988, p. 460).

Based on this evidence, OSHA preliminarily concludes that the exposed workers in construction, maritime, and agriculture are at risk of experiencing the hepatotoxic effects of exposure to 1,1,2,2-tetrachloroethane. OSHA preliminarily finds that the proposed 1 ppm 8-hour TWA limit and skin notation are necessary to substantially reduce this significant risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

TETRACHLORONAPHTHALENE

CAS: 1335–88–2; Chemical Formula: $C_{10}H_4Cl_4$

H.S. No.: 2155

OSHA's limit for tetrachloronaphthalene in general industry, construction, and maritime is an 8-hour TWA of 2 mg/m³, with a skin notation. There is no limit for this substance in agriculture. The ACGIH TLV®–TWA for tetrachloronaphthalene is 2 mg/m³; NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 2 mg/m³, and a skin notation, for tetrachloronaphthalene in agriculture; adoption of this limit would make the PEL for this substance consistent across all OSHA-regulated industries.

Tetrachloronaphthalene is a colorless to pale yellow solid with an aromatic odor (ACGIH 1986, p. 562). The chloronaphthalenes in commercial use are synthetic waxes and lubricants that are composed of varying mixtures of the tri-, tetra-, penta-, or hexa-chloronaphthalenes. Chloronaphthalenes are used as electrical insulators, condenser dielectrics, and separators in batteries. They also find use in flame- and waterproof coatings for wood, paper, and textiles and were formerly used in pesticides (HSDB 1989). In agriculture, chloronaphthalenes have been used as lubricants for feed pelleting and other farm machinery (Clayton and Clayton 1981, p. 3671).

Tetrachloronaphthalene causes liver damage in humans and animals. There are no acute toxicity data for tetrachloronaphthalene alone. Some animals in a group of rats fed 0.5 mg/kg/day of a mixture of tetra- and pentachloronaphthalene for 2 months became sick and others died; at autopsy, signs of liver injury were apparent (Drinker et al., in Clayton and Clayton 1981, p. 3670). Rats exposed for 16 hours/day by inhalation to a 1.3-mg/m³ concentration of tri- and tetrachloronaphthalene for up to 4.5 months showed no adverse effects other than possible liver enlargement; at a concentration of 19.97 mg/m³, however, liver damage was seen in these animals at autopsy (Drinker et al., in Clayton and Clayton 1981, p. 3670). Thousands of farm animals have been killed by hyperkeratosis, a disease caused by the ingestion of chloronaphthalene-containing lubricants that have contaminated pelleted feed (Gosselin, Smith, and Hodge 1984, p. II–172).

Workers exposed to chloronaphthalenes by inhalation of the vapor or dust have developed toxic hepatitis and necrosis of the liver; several deaths have been attributed to the industrial use of the chloronaphthalenes (Drinker et al. 1937; Greenburg, Mayers, and Smith 1937; Mayers and Smith 1942). Workers exposed for 4 months to an unknown concentration of tetra- and penta-chloronaphthalene developed headaches, fatigue, anorexia, and vertigo; the dermatitis and chloracne developed by these workers, however, appears to have been caused by penta-, rather than tetra-, chloronaphthalene (Kleinfeld, Messite, and Swencicki 1972). Tests involving human volunteers have shown that the tri- and tetrachloronaphthalenes do not cause chloracne when in contact with the skin (Shelley and Kligman 1957).

Based on this evidence in humans and animals, OSHA preliminarily concludes that tetrachloronaphthalene is a liver toxin that poses a potentially significant risk to workers in agriculture. Accordingly, OSHA is proposing to extend the Agency's 2-mg/m³ 8-hour TWA PEL and a skin notation to workplaces in this sector; this action will provide agricultural workers with the same protection afforded workers in other OSHA-regulated sectors.

TRICHLORONAPHTHALENE

CAS: 1321–65–9; Chemical Formula: $C_{10}H_5Cl_3$

H.S. No. 2163

OSHA's limit for trichloronaphthalene in general industry, construction, and maritime is 5 mg/m³ as an 8-hour TWA, with a skin notation. There is no limit in agriculture. The ACGIH TLV®–TWA for this substance is 5 mg/m³, with a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 5 mg/m³, and a skin notation, for trichloronaphthalene in agriculture; this action would make OSHA's limit for this substance consistent across all OSHA-regulated sectors.

Trichloronaphthalene is a colorless, white, or pale yellow solid with an aromatic odor (ACGIH 1986, p. 600). The chloronaphthalenes in commercial use are synthetic waxes and lubricants that are composed of varying mixtures of the tri- and tetra-, or penta- and hexa-, chloronaphthalenes. Chloronaphthalenes are used in insulation for electric wires, as additives for lubricants, as supports for storage batteries, and in coatings for foundry use. These substances are also used in solvents and as immersion liquids in microscopy (HSDB 1989).

Trichloronaphthalene is a liver toxin in humans and animals. There are no acute toxicity data for trichloronaphthalene alone. Some of a group of rats fed a mixture of tetra- and trichloronaphthalene at a dose of 0.5 mg/kg/day for 2 months died, while others became sick; at autopsy, all animals showed signs of liver damage (Drinker et al. 1957). Daily exposure of rats to the vapors of a mixture of tetra- and trichloronaphthalene at a concentration of 1.3 mg/m³ for as long as 4.5 months produced only slight enlargement of the liver; increasing the concentration to 10.97 mg/m³, however, resulted in liver damage visible at autopsy (Drinker et al. 1957). Thousands of farm animals died after ingesting chloronaphthalene-contaminated feed; the feed was contaminated by the

lubricants used to oil the feed-pelleting machinery (Gosselin, Smith, and Hodge 1984, p. II–172).

A worker exposed to a 3-mg/m³ concentration of trichloronaphthalene (mixed with tetrachloronaphthalene) developed toxic hepatitis but subsequently recovered (Mayers and Smith 1942, in ACGIH 1986, p. 600). No industrial fatalities have been attributed to trichloronaphthalene exposure (ACGIH 1986, p. 600). Although the specific threshold for trichloronaphthalene-induced eye irritation is not known, this substance is regarded as an eye irritant (Parmeggiani 1983, p. 466). Tests in human volunteers have shown trichloronaphthalene not to be acnegenic, although some of the chloronaphthalenes do cause chloracne (Shelley and Kligman 1957).

Based on this evidence in humans and animals, OSHA preliminarily concludes that trichloronaphthalene is a liver toxin that poses a potentially significant risk to workers in agriculture. Accordingly, OSHA is proposing to extend the Agency's 5 mg/m³ 8-hour TWA PEL, and a skin notation, to workplaces in this sector; this action will provide agricultural workers with the same protection provided to workers in other OSHA-regulated sectors.

1,2,3-TRICHLOROPROPANE
CAS: 96–18–4; Chemical Formula: $CH_2ClCHClCH_2Cl$
H.S. No. 1407

In construction and maritime, OSHA's PEL for 1,2,3-trichloropropane is 50 ppm as an 8-hour TWA. There is no PEL in agriculture. The 1987–1988 ACGIH TLV® is 10-ppm TWA, with a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N6A) with the limit OSHA is proposing for construction, maritime, and agriculture, which is a 10-ppm 8-hour TWA. This is the limit recently established for this substance in general industry.

1,2,3-Trichloropropane is a colorless to straw-colored, combustible liquid with an odor similar to that of chloroform. This substance finds use as a degreasing agent, a paint and varnish remover, and a solvent. It is also used in chemical synthesis (HSDB 1988).

In addition to liver toxicity, 1,2,3-trichloropropane causes irritation of the eyes, skin, and mucous membranes and narcosis in experimental animals. The oral LD_{50} in rats is 320 mg/kg; in mice, the LC_{50} is 3400 mg/m³ for 2 hours (RTECS 1990). The dermal LD_{50} in rabbits is 1770 mg/kg (RTECS 1990). Acutely poisoned animals surviving for 1 week to 10 days after exposure died of liver damage (Proctor, Hughes, and Fischman 1988, p. 491). Instilled into the eyes of rabbits, 1,2,3-trichloropropane caused severe irritation (RTECS 1990; Proctor, Hughes, and Fischman 1988, p. 491). Applied to the skin of rabbits, the liquid caused defatting and irritation described by the investigators as "intense" (McOmie and Barnes 1949). Five of six rats exposed to a 1000-ppm concentration of 1,2,3-trichloropropane died after four-hour exposures. Rats and guinea pigs exposed at 800, 2100, or 5000 ppm for 30 minutes showed central nervous system depression, which progressed, at the higher exposure levels, to narcosis and convulsions (Lewis 1979). Daily 10-minute exposures at 2500 ppm for 10 days killed 7 of 10 mice (McOmie and Barnes 1949). Animals exposed once for four hours to 1,2,3-trichloropropane at concentrations of 125, 340, 700, or 2150 ppm showed dose-related signs of irritation, which included, at 700 or 2150 ppm, labored respiration, inactivity, and eye and nose irritation; at autopsy, however, no organ or other damage was apparent (McOmie and Barnes 1949).

Drew, Patel, and Lin (1978/Ex. 1–313) noted changes in rat liver enzymes after a single four-hour exposure to 500 ppm, and Russian studies indicate that morphologic changes and metabolic lesions of the liver, kidney, and lungs occurred in mice exposed continuously to 1,2,3-trichloropropane concentrations of 0.007 to 0.3 ppm (Sidorenko, Tsulaya, Bonashevskaya, and Shaipak 1979/Ex. 1–669; Sidorenko, Tsulaya, Koreneveskaya, and Bonashevskaya 1976/Ex. 1–668; Tsulaya, Bonashevskaya, Zykova et al. 1977/Ex. 1–450).

A National Toxicology Program (NTP) prechronic study, in which rats were gavaged daily with 1,2,3-trichloropropane at 8, 16, 32, 63, 125, or 250 mg/kg body weight for 120 days, showed good survival in all but the highest dose group (NTP 1983a). Statistically significant changes in the liver and kidneys, as well as necrosis and irritation of the nasal passages, occurred in the 63- and 125-mg/kg dose groups. Decreases in red blood cell counts and hematocrits were also seen, even in the 16-mg/kg dose group. 1,2,3-Trichloropropane did not affect testicular weight, sperm count, or morphology. The NTP found this substance to be genetically active in three bioassays. Hardin, Bond, Sikov et al. (1981/Ex. 1–699) did not find 1,2,3-trichloropropane to be fetotoxic or teratogenic.

Human volunteers found exposure to 1,2,3-trichloropropane objectionable because of eye and upper respiratory tract irritation, and many found 50 ppm an unacceptable level for a full-shift exposure (Silverman, Schulte, and First 1946/Ex. 1–142). At a 100-ppm concentration, all of the volunteers noted eye and nose irritation and reported that the odor was unpleasant (Silverman, Schulte, and First 1946/Ex. 1–142).

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 10 ppm for 1,2,3-trichloropropane. The Agency preliminarily concludes that a 10-ppm 8-hour TWA limit is necessary to protect workers in construction, maritime, and agriculture against the significant risks of liver and kidney damage and eye and throat irritation that are potentially associated with exposures to this substance at levels above the proposed PEL. OSHA is aware that a lower PEL may be appropriate for trichloropropane. However, as discussed above, the primary purpose of the current rulemaking is to extend the protection afforded to workers in some sectors to those in all sectors. In a future PEL update rulemaking, OSHA will carefully consider the need for a lower PEL for this substance.

Introduction

Kidney damage is the basis for proposing to establish or revise the PELs for six of the substances in this group. These chemicals, their CAS and HS numbers, and their current PELs in construction and maritime are shown in Table C4–2. Their 1987–1988 ACGIH TLV®s and NIOSH RELs are also shown. The PELs OSHA is proposing for these kidney toxins in construction, maritime, and agriculture appear in the last column of Table C4–2. Promulgation of the proposed PELs will make their limits consistent across general industry.

TABLE C4-2.—LIST OF SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED PRIMARILY ON AVOIDANCE OF KIDNEY TOXICITY

H.S. No./chemical name	CAS No.	Current OSHA PEL in construction and maritime *	1987-1988 ACGIH TLV® **	NIOSH REL*	Proposed OSHA PEL in construction, maritime, and agriculture *
1129 1,3-Dichloropropene	542-75-6-		1 ppm TWA, Skin		1 ppm TWA, Skin.
1132 Dicyclopentadiene	77-73-6		5 ppm TWA		5 ppm TWA.
1166 Ethyl silicate-	78-10-4	100 ppm TWA	10 ppm TWA		10 ppm TWA.
1195 Hexachlorobutadiene	87-68-3		0.02 ppm TWA, Skin A2		0.02 ppm TWA.
1203 Hexone (Methyl isobutyl ketone).	108-10-1	100 ppm TWA	50 ppm TWA, 75 ppm STEL	50 ppm TWA	50 ppm TWA, 75 ppm STEL.
1419 Uranium, soluble compounds.	7440-61-1	0.2 mg/m³ TWA	0.2 mg/m³ TWA, 0.6 mg/m³ STEL.		0.05 mg/m³ TWA.

* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes only unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time; OSHA's PELs do not currently apply in Agriculture.

** The ACGIH TLV®-TWA is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time. An A2 designation means that the ACGIH classifies the substance as a suspected human carcinogen.

* NIOSH TWA limits are for 10 hour/day, 40 hour/week exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

Description of the Health Effects

The precise mechanism by which these chemicals damage the kidneys is unknown. Typically, these substances are selectively toxic to cells in the renal tubules, perhaps because impaired transport causes the chemical to collect in these cells. In addition to its function in the excretion of wastes, the kidney plays an important role in the regulation of total body homeostasis. This organ regulates extracellular volume, controls electrolyte and acid-base balance, and forms several hormones that control systemic metabolism. Depending on their particular site of action, nephrotoxicants can interfere with hydration, the proper excretion of the body's wastes, electrolytic balance, metabolism, or the maintenance of correct acid-base balances. Like the hepatotoxic effects previously described, the least severe lesions caused by nephrotoxic compounds are graded and reversible. The earliest changes are usually alterations in the activities of specific enzymes in the tubular cells. These changes may be accompanied by minor morphological alterations of the cells that are visible only with an electron microscope. Higher doses or more sustained exposures are required to cause cellular necrosis that is visible with light microscopy. Because of the reserve capacity of the kidneys, a significant degree of tubular cell necrosis must occur before it is reflected by measurable alterations in kidney function. Thus, indicators of impaired renal function that can be measured in humans, such as proteinuria, glucosuria, and increased BUN, are relatively insensitive indicators of early kidney damage. Other indicators of significant kidney damage include increased kidney weight, swelling of the tubular epithelium, fatty degeneration of tubular epithelium, and the presence of tubular casts in the urine.

Dose-Response Characteristics

Kidney damage, like liver damage, is progressive; only at the earlier stages are nephrotoxic effects reversible. With continued exposure, the damage becomes more extensive, until it reaches the point at which it cannot be repaired. The toxicity of the kidney-damaging chemicals included in this group also increases as dose increases. For most nephrotoxins, there appears to be a no-observed-effect level. Workplace exposures to concentrations of these substances at levels at or below the proposed limits are unlikely to cause observable kidney effects in most workers. OSHA has preliminarily determined that the nephrotoxic risks being protected against are significant at the current PELs or in the absence of a PEL and believes that kidney damage constitutes a material health impairment within the meaning of the Act. The PELs being proposed are those already established in general industry or other sectors, and promulgation will thus make the PELs for these substances consistent across all regulated sectors.

1,3-DICHLOROPROPENE

CAS: 542-75-6; Chemical Formula: $CHCl{=}CH{-}CH_2Cl$

H.S. No. 1129

In construction, maritime, and agriculture, OSHA has no PEL for 1,3-dichloropropene. NIOSH has no REL but concurs (Ex. 8-47, Table N6A) with the proposed limit. The 1987-1988 ACGIH TLV® for this substance is 1 ppm as an 8-hour TWA, with a skin notation. The Agency is proposing an 8-hour TWA limit of 1 ppm, with a skin notation, for this dichloropropene in construction, maritime, and agriculture. Promulgation of this PEL will make the limit for this substance consistent across all regulated sectors.

This compound occurs in two forms: as the cis- and the trans-isomer. It is a colorless to straw-colored liquid that is used in organic synthesis, as a soil fumigant, and as a pesticide on a variety of crops (HSDB 1989). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In male and female rats, the acute oral LD_{50}s for a 92-percent mixture of the cis- and trans-isomers of 1,3-dichloropropene were 713 and 470 mg/kg, respectively; post-mortem examination showed liver and kidney damage and evidence of possible lung injury (Torkelson and Oyen 1977/Ex. 1-532). The dermal LD_{50} in rabbits for a 92-percent undiluted mixture was 504 mg/kg, but a 10-percent solution administered by gavage at a dose of 125 or 250 mg/kg was lethal to some of the animals (Torkelson and Oyen 1977/Ex. 1-532). Contact with the liquid was irritating to the eyes and skin of rabbits (Torkelson and Oyen 1977/Ex. 1-532).

Inhalation exposures to 1,3-dichloropropene vapor concentrations above 2700 ppm produced eye and nasal irritation and severe lung, nasal, kidney, and liver damage in rats (Torkelson and Oyen 1977/Ex. 1-532). Exposure to 1000 ppm caused eye and nasal irritation, lacrimation, and, if prolonged, unconsciousness; rats exposed to 1000 ppm for two hours died, but those exposed for one hour survived (Torkelson and Oyen 1977/Ex. 1-532). Guinea pigs exposed to 400 ppm for a single 7-hour period died, while rats exposed similarly survived but had obvious lung congestion (Torkelson and Oyen 1977/Ex. 1-532). Rats, rabbits, guinea pigs, and dogs were exposed 7

hours/day, 5 days/week for 6 months to 1-ppm or 3-ppm concentrations of 1,3-dichloropropene (Torkelson and Oyen 1977/Ex. 1–532). No adverse effects were observed in any of the animals exposed at 1 ppm. Of the animals exposed at 3 ppm, only male rats showed adverse effects; these animals had reversible cloudy swelling of the renal tubular epithelium (Torkelson and Oyen 1977/Ex. 1–532).

In humans, acute exposures to 1,3-dichloropropene cause skin, eye, and respiratory irritation (Torkelson and Oyen 1977/Ex. 1–532). There are no data on the effects in humans of chronic exposure to this substance.

OSHA is proposing an 8-hour TWA limit of 1 ppm, with a skin notation, for 1,3-dichloropropene in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in construction, agriculture, and maritime against the significant risks of eye and mucous membrane irritation and lung, kidney, and liver damage that are associated with exposure to this substance. A skin notation is proposed to protect against 1,3-dichloropropene's ability to cause systemic toxicity when absorbed through the skin. Promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DICYCLOPENTADIENE

CAS: 77–73–6; Chemical Formula: $C_{10}H_{12}$

H.S. No. 1132

OSHA has no limit for dicyclopentadiene (DCPD) in construction, maritime, or agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the proposed limit. The 1987–1988 ACGIH TLV® for dicyclopentadiene is 5-ppm as an 8-hour TWA. In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA PEL of 5 ppm. This is the limit recently established for this substance in general industry.

At room temperature, DCPD is a solid that has a disagreeable odor. This substance is used in the production of chlorinated hydrocarbon pesticides, to stabilize organophosphorus pesticides, and to produce elastomers, paints, varnishes, and flame retardants (Clayton and Clayton 1981, p. 3242; ACGIH 1986, p. 194). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The health effects associated with exposure to DCPD include severe eye and moderate skin irritation (RTECS 1990), as well as renal damage and possible pulmonary damage. By the oral and intraperitoneal routes, DCPD is extremely toxic, with an oral LD_{50} value of 353 mg/kg and an intraperitoneal LD_{50} value of 200 mg/kg in rats (RTECS 1990); fatalities occurred among rats within 60 minutes of exposure to the saturated vapor (Kinkead, Pozzani, Geary, and Carpenter 1971/Ex. 1–606). The LC_{50} in rats is 359 ppm for 4 hours, and the dermal LD_{50} in rabbits is 5080 mg/kg (RTECS 1990). Kinkead and associates (1971/Ex. 1–606) report that rats exposed repeatedly for 10 days survived concentrations of 72 or 146 ppm but had convulsions and succumbed at the 332-ppm level; autopsy revealed lung hemorrhage and blood in the intestines; in the females, hemorrhage of the thymus also occurred (Kinkead, Pozzani, Geary, and Carpenter 1971/Ex. 1–606). Chronic exposures of 7 hours/day for 89 days produced kidney damage and some pulmonary effects in rats exposed to concentrations of 35 or 74 ppm; the no-effect level for these endpoints in rats was determined to be below 19.7 ppm. Dogs exposed to dicyclopentadiene concentrations of 9, 23, or 32 ppm on the same regimen exhibited only minimal effects (Kinkead, Pozzani, Geary, and Carpenter 1971/Ex. 1–606).

Human sensory response tests resulted in findings of mild eye and throat irritation within 7 minutes' exposure to DCPD vapor at a concentration of 1 ppm and of olfactory fatigue within 24 minutes; a 30-minute exposure to a 5.5-ppm concentration produced no olfactory fatigue (ACGIH 1986/Ex. 1–3, p. 194). Subjective complaints of headache during the first two months of occupational exposure disappeared during the following 3 months of exposure, suggesting that humans develop a tolerance for this substance (ACGIH 1986/Ex. 1–3, p. 194).

Based on this evidence in animals and humans, OSHA is proposing an 8-hour TWA PEL of 5 ppm for dicyclopentadiene. The Agency preliminarily concludes that this limit will protect workers in construction, maritime, and agriculture against the significant risks of kidney injury, pulmonary effects, and irritation that are associated with occupational exposure to DCPD at the levels permitted in the absence of an OSHA limit in these sectors. OSHA believes that the proposed PEL is necessary to substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ETHYL SILICATE

CAS: 78–10–4; Chemical Formula: $Si(OC_2H_5)_4$

H.S. No. 1166

OSHA's permissible exposure limit for ethyl silicate in construction and maritime is 100 ppm as an 8-hour TWA. There is no PEL in agriculture. NIOSH has no REL for ethyl silicate but concurs (Ex. 8–47, Table N1) with the selection of the proposed limit. The 1987–1988 ACGIH TLV® for this substance was an 8-hour TWA of 10 ppm. OSHA is proposing a 10 ppm 8-hour TWA PEL for ethyl silicate in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Ethyl silicate is a colorless liquid with a faint odor. It is used as an adherent in controlled-release pesticides, as a weatherproofing agent on mortar and cement and in paints and other coatings. It is also used as a chemical intermediate (ACGIH 1986, p. 264; HSDB 1986). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In addition to kidney damage, ethyl silicate causes both irritation and systemic toxicity. The oral LD_{50} in rats is 6270 mg/kg, and the lowest lethal concentration in rats is 1000 ppm for 4 hours (RTECS 1990). The dermal LD_{50} in rabbits is 5878 mg/kg (RTECS 1990). In guinea pigs and rats, a 60-minute exposure of 2000 ppm was reported as the maximal duration/concentration that did not cause serious disturbances; 500 ppm was the maximal no-effect exposure level for an exposure of several hours' duration (Smyth and Seaton 1940b/Ex. 1–376). Thirty-day exposures to 400 ppm ethyl silicate for 7 hours/day caused significant mortality in rats and damage to the lungs, liver, and kidney in the surviving animals. Exposures of rats, guinea pigs, and mice to 88, 50, or 23 ppm for 90 days (7 hours/day, 5 days/week) resulted only in decreased kidney weights in mice exposed at the 88-ppm level (Pozzani and Carpenter 1951/Ex. 1–166). In another study, Kasper, McCord, and Fredrick (1937/Ex. 1–1155) showed that animals exposed to 164 ppm ethyl silicate for 17 8-hour days showed less weight gain than did controls. Rowe and associates (1948/Ex. 1–359) reported that three 7-hour exposures to a 1000-ppm concentration were fatal to 4 of 10 rats; similar exposures to 500 ppm caused pronounced kidney changes and slight lung irritation. Four to 10 similar exposures at 250 ppm caused slow weight loss and some lung and renal changes; at 125 ppm, slight to moderate kidney damage was observed (Rowe, Spencer, and Bass 1948/Ex. 1–359).

Smyth and Seaton (1940b/Ex. 1–376) reported that exposure to a concentration of 1200 ppm causes lacrimation in humans and that 250 ppm causes eye and nose irritation. Very brief exposures to high concentrations of ethyl silicate cause severe irritation in humans. At a 3000-ppm concentration, the irritation caused even by brief exposures is reported to be intolerable. At a 1200-ppm concentration, exposure causes a stinging sensation and tearing; at 700 ppm, mild stinging of the eyes occurred. Even at a concentration of 250 ppm, slight irritation of the eyes was reported (Proctor, Hughes, and Fischman 1988, p. 256).

In construction, maritime, and agriculture, OSHA is proposing a PEL for ethyl silicate of 10 ppm as an 8-hour TWA. The Agency preliminarily concludes that this limit is necessary to protect workers in construction, maritime, and agriculture from the significant risk of renal damage that is associated with exposures to this substance at concentrations above the proposed PEL. OSHA considers these effects material impairments of health and believes that the proposed PEL will reduce this significant risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

HEXACHLOROBUTADIENE

CAS: 87–68–3; Chemical Formula: $CCl_2=CCl—CCl=CCl_2$

H.S. No. 1195

In construction, maritime, and agriculture, OSHA has no exposure limit for hexachlorobutadiene (HCBD). NIOSH has no REL for hexachlorobutadiene but concurs (Ex. 8–47, Table N6A) with the limit being proposed. The ACGIH has a TLV®–TWA of 0.02 ppm, and a skin notation, for this substance, which it also classifies as a suspected human carcinogen (A2). OSHA is proposing an 8-hour TWA PEL of 0.02 ppm for HCBD in construction, maritime, and agriculture. Promulgation of this limit will make the PEL for this substance consistent across all regulated sectors.

Hexachlorobutadiene is a heavy, clear liquid. This substance is used as a chemical intermediate in the production of lubricants and of rubber compounds. It also finds use in the recovery of chlorine-containing gas in chlorine plants and as a gyroscope fluid (IARC 1979, p. 180).

In addition to kidney injury, exposure to hexachlorobutadiene causes respiratory and other irritation, as well as cancer. The LD_{50}s reported for mice, rats, and guinea pigs are 110, 90, and 90 mg/kg, respectively (RTECS 1990), and the dermal LD_{50} in rabbits is 1211 mg/kg (RTECS 1990). Rats survived exposures to a 161-ppm concentration for 0.88 hour or to 34 ppm for 3.3 hours; most guinea pigs and cats similarly exposed died (Kociba, Schwetz, Keyes et al. 1977/Ex. 1–494). Another inhalation study in rats showed eye and nose irritation, respiratory difficulty, and damage to kidney tissue and the adrenal cortex after two 4-hour exposures at 250 ppm; twelve 6-hour exposures to 100 ppm caused eye and nose irritation, respiratory difficulty, weight loss, anemia in the female animals, and kidney and adrenal damage; fifteen 6-hour exposures to 25 ppm caused retarded weight gain in females, respiratory difficulty, and kidney damage; fifteen 6-hour exposures to a 10-ppm concentration caused retarded weight gain in females but no systemic injury; and fifteen 6-hour exposures at 5 ppm resulted in no adverse effects (Gage 1970/Ex. 1–318).

Reproductive studies in male and female rats demonstrated multiple toxicological effects, including kidney damage in both sexes and increased liver weight in males, at the high-dose level of 20 mg/kg/day. Dietary administration of 20, 2, or 0.2 mg/kg daily had no effect on conception percentages, gestational survival, neonatal survival, neonatal sex ratios, neonatal morphology, or neonatal body weights (except for the high-dose neonates) (Schwetz, Smith, Humiston et al. 1977/Ex. 1–368). Results of lifetime dietary studies suggest that the no-effect level for hexachlorobutadiene in rats is 0.2 mg/kg/day, that a clear dose-response relationship exists for HCBD-induced toxicity affecting primarily the kidney, and that carcinogenic effects (i.e., renal neoplasms) result from ingestion of 20 mg/kg/day (Kociba, Schwetz, Keyes et al. 1977/Ex. 1–494). These authors also reported that HCBD-induced neoplasms occurred in these animals only at HCBD doses higher than those causing discernible renal injury. The International Agency for Research on Cancer (IARC) has concluded that there is limited evidence that hexachlorobutadiene is carcinogenic in animals (IARC 1979, p. 189). NIOSH also (Ex. 8–47, Table N6A) considers hexachlorobutadiene a potential human carcinogen.

Based on this evidence, OSHA is proposing an 8-hour TWA limit of 0.02 ppm for hexachlorobutadiene in construction, maritime, and agriculture. Assuming a 10-m^3 per day breathing volume per 8-hour workshift and a 70-kg body weight for humans, the proposed limit would correspond to a daily hexachlorobutadiene intake of approximately 0.03 mg/kg, which is about 10 times below the observed no-effect level in rats fed hexachlorobutadiene. The Agency preliminarily concludes that this 0.02-ppm limit will protect workers in construction, maritime, and agriculture from the significant risks of kidney damage; eye, skin, and pulmonary irritation; and renal neoplasms that are potentially associated with exposure to HCBD at levels above the proposed limit. OSHA considers these effects material impairments of health and believes that the proposed PEL is necessary to substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

HEXONE (METHYL ISOBUTYL KETONE)

CAS: 108–10–1; Chemical Formula: $CH_3COCH_2CH(CH_3)_2$

H.S. No. 1203

In construction and maritime, OSHA's 8-hour TWA limit for hexone (methyl isobutyl ketone, or MIBK) is 100 ppm. The ACGIH has established a TLV®–TWA of 50 ppm and a TLV®–STEL of 75 ppm for this substance. NIOSH has a TWA REL of 50 ppm for MIBK but concurs (Ex. 8–47, Table N1) that the limits OSHA is proposing are appropriate. OSHA is proposing a 50-ppm 8-hour TWA limit and a 75-ppm STEL for hexone in construction, maritime, and agriculture. These are the limits recently established for this substance in general industry.

Hexone is a clear liquid with a characteristic ketone odor. This substance is used as a nitrocellulose and cellulose ether solvent and a solvent for paints, varnishes, protective coatings, fats, waxes, oils, gums, and resins (ACGIH 1986, p. 402; HSDB 1986). It also finds use in drycleaning preparations and in the synthesis of methyl isobutyl carbinol (HSDB 1986).

In addition to renal toxicity, hexone causes irritation of the eyes, mucous membranes, and skin and, at high concentrations, is a narcotic in animals (Proctor, Hughes, and Fischman 1988, p. 337). The oral LD_{50} in rats is 2080 mg/kg, and the LC_{50} in the same species is 8000 ppm for 4 hours (RTECS 1990). A 4-hour exposure to 4000 ppm MIBK killed all exposed rats, but a similar exposure to 2000 ppm was not fatal to these animals (Smyth, Carpenter, and Weil 1951/Ex. 1–439). Guinea pigs exposed to a MIBK concentration of 10,000 ppm immediately showed signs of irritation (Specht, Miller, Valaer, and Sayers 1940/Ex. 1–1179).

MacEwen, Vernot, and Haun (1971/Ex. 1–194) exposed rats, mice, dogs, and monkeys to 100 or 200 ppm MIBK for two weeks and noted no signs of intoxication; however, rats exposed to 100 ppm had heavier kidneys and higher kidney-to-body-weight ratios at autopsy, and, at 200 ppm, the livers of exposed animals were heavier as well. Postmortem examination revealed nephrosis of the proximal tubules. The same authors (MacEwen, Vernot, and Haun 1971/Ex. 1–194), exposed rhesus monkeys, dogs, and rats continuously for 90 days to MIBK concentrations of 100 ppm. These authors observed no significant changes in clinical chemistry or blood test results, although the rats had heavier kidneys and livers, reversible hyaline droplet degeneration of the proximal tubules of the kidneys, and some necrosis of the tubules at autopsy.

Silverman, Schulte, and First (1946/Ex. 1–142) determined that the maximum dose of MIBK tolerable to human volunteers for eight hours was 100 ppm; at 200 ppm, these subjects found the odor of MIBK objectionable and the vapor irritating. Linari and co-workers (1964/Ex. 1–1159) reported that more than half of all workers exposed to 500 ppm of MIBK for 20 to 30 minutes daily, and perhaps to 80 ppm for the remainder of the shift, experienced weakness, loss of appetite, headache, burning eyes, nausea, vomiting, and sore throat; several of these workers also reported insomnia, somnolence, heartburn, and intestinal pain. Some workers had enlarged livers and others had colitis. Clinical test results on these workers were normal (Linari, Perrelli, and Varese 1964/Ex. 1–1159).

In a follow-up study on this same group of centrifuge operation workers, Armeli and co-workers (1968/Ex. 1–1028) determined that reduction of MIBK levels (during the 15 to 30 minutes of centrifuge operation) to 100 to 105 ppm, and (for the remainder of the shift) to 50 ppm had also significantly reduced the symptomatology reported earlier by these workers. However, liver enlargement persisted in two workers, and a few workers continued to report gastrointestinal and nervous system effects (Armeli, Linari, and Martorano 1968/Ex. 1–1028).

Elkins (1959f/Ex. 1–734) noted that exposure to 100 ppm during boot-waterproofing operations caused workers to develop headache and nausea; another similarly exposed group experienced only irritation at 100 ppm. Human volunteers developed headaches, eye irritation, sore throat, and nausea and experienced weakness when they were exposed for an unspecified time to a hexone concentration of 80 to 500 ppm (Proctor, Hughes, and Fischman 1988, p. 337). Hexone causes defatting of the skin on repeated or prolonged contact (AIHA 1966).

OSHA is proposing an 8-hour TWA limit of 50 ppm and a 15-minute STEL of 75 ppm for hexone in construction, maritime, and agriculture. The Agency preliminarily concludes that these limits will protect workers in construction, maritime, and agriculture from the significant risks of headache, nausea, and irritation, as well as the potential kidney and liver effects that are associated with exposures to hexone. OSHA considers these effects material impairments of health and believes that the proposed PELs are necessary to substantially reduce these risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

URANIUM (SOLUBLE COMPOUNDS)
CAS: Varies; Chemical Formula: Varies

In construction and maritime, OSHA's PEL for the soluble compounds of uranium is an 8-hour TWA of 0.2 mg/m^3, measured as uranium. There is no PEL in agriculture. The ACGIH TLV®s for the soluble uranium compounds are 0.2 mg/m^3 as an 8-hour TWA and 0.6 mg/m^3 as a 15-minute STEL. NIOSH has no REL but concurred (Ex. 8–47, Table N1) with the proposed limit when it was established recently in general industry. In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA of 0.05 mg/m^3.

The soluble compounds of uranium most common in industrial use are uranium hexafluoride, uranyl acetate, uranyl chloride, uranyl fluoride, uranyl nitrate, and uranyl sulfate. The physical and chemical properties and uses of these compounds vary with the particular substance.

Exposure of animals to the soluble compounds of uranium may cause both chemical poisoning and radiation injury (Clayton and Clayton 1981, p. 1996). The soluble uranium compounds are chemically more toxic than the insoluble compounds, but both the soluble and insoluble compounds have the same potential to cause radiation injury (Clayton and Clayton 1981, p. 2000; Klaassen, Amdur, and Doull 1986, p. 695). The most significant damage occurs to the kidneys and lungs, but the eyes and central nervous system also may be adversely affected. Data suggest that exposure to uranium also causes carcinogenic and mutagenic effects. The intraperitoneal LD_{50} for uranyl nitrate is 400 mg/kg in mice (Sax and Lewis 1989, p. 3446). Exposure to the dusts and mists of the soluble uranium compounds is fatal to almost all animals exposed by inhalation to 20 mg/m^3 daily for 1 month; death is caused by acute renal failure and pulmonary insufficiency. Exposure levels of 0.2 mg/m^3 are occasionally fatal, while levels of 0.05 mg/m^3 produce no detectable effects (Clayton and Clayton 1981, p. 2001; Proctor, Hughes, and Fischman 1988, p. 501). Rats, monkeys, and dogs exposed to an airborne concentration of 5 mg/m^3 soluble uranium compounds for 6 hours/day, 5 days/week for 5 years showed no evidence of acute uranium toxicity, although autopsy 6.5 years after cessation of exposure revealed an increase in the incidence of pulmonary neoplasms in the dogs and monkeys (AIHA 1978). Dogs fed 0.2 mg/kg uranyl nitrate for 1 year tolerated the exposure but exhibited growth deficits and abnormal urinary protein and glucose levels indicative of renal damage (Clayton and Clayton 1981, p. 2001). Male rats fed 0.1 percent uranyl nitrate and female rats fed 0.5 percent uranyl nitrate for 2 years showed no adverse growth effects; exposure to 0.05 percent for 2 years caused no effects (Clayton and Clayton 1981, p. 2001). Rabbits exposed to soluble uranium salts developed neurological symptoms and pathological changes in the cerebral and cerebellar cortices; exposed dogs developed epithelial degeneration of the choroid plexi (HSDB 1986). All soluble uranium compounds are lethal when applied in a single dose to rabbits' skin; some are also lethal when placed in the conjunctival sac (Clayton and Clayton 1981, p. 2000). Most soluble uranium compounds also damage the eye itself on contact (Grant 1986, p. 965). Exposure to 0.18 mg/ml uranyl nitrate produced chromatid and chromosomal damage in Chinese hamster cells in vitro (HSDB 1986).

As in animals, exposure of humans to the soluble compounds of uranium may cause both chemical poisoning and radiation injury. Acute exposure to the dusts of the soluble compounds irritates the mucous membranes, eyes, and respiratory tract, and acute overexposure via any route of administration causes kidney damage (Proctor, Hughes, and Fischman 1988, p. 502). Several acute exposures have been documented. Two deaths occurred after exposure to gaseous uranium hexafluoride: One worker died 10 minutes after a 5-minute exposure, and the other died 70 minutes after a brief exposure. Both cases were complicated by exposure to steam, which caused

third-degree burns and liberated hydrofluoric acid (Clayton and Clayton 1981, pp. 2008–2009). In another incident, exposure to gaseous uranium hexafluoride (and its hydrolysis products, including hydrofluoric acid) caused chemical conjunctivitis with corneal necrosis, cough, and shortness of breath, accompanied by increased density of bronchovascular markings and hilar shadows on chest X-ray, laryngeal hemorrhages, and albuminuria, microhematuria, an elevated blood urea nitrogen level, and increased urinary solids (which are indicative of kidney injury and renal insufficiency). With treatment, recovery was complete (Clayton and Clayton 1981, p. 2009). Uranium miners in the United States, Czechoslovakia, and Canada have an excess incidence of deaths from respiratory cancer and pulmonary insufficiency, which are presumed to be caused by radiation injury from radon gas, a byproduct of uranium decay (Rom 1983, p. 688). A study of the risk of respiratory deaths among uranium miners in the U.S. showed the following dose-response: miners exposed for 5 to 9.9 years had a 2-fold increase in risk; miners exposed for 10 to 24.9 years had a 3.6-fold increase in risk; and those exposed for greater than 24.9 years had a 3.75-fold increase in risk. Smoking was shown both to increase the risk of death from respiratory disease and to shorten the neoplastic latency period (Clayton and Clayton 1981, pp. 2010–2011). In contact with the skin, uranyl nitrate causes burns (Klaassen, Amdur, and Doull 1986, p. 628). Uranium miners have elevations of all types of chromosome aberrations; an increase in chromosomal aberrations in cultured lymphocytes is correlated with exposure to radon daughters (Rom 1983, p. 689). Uranyl nitrate is mutagenic to lymphocytes at 100 μg/l (RTECS 1989).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to the soluble compounds of uranium is associated with kidney and liver damage and, perhaps, of cancer. The Agency believes that the proposed PEL of 0.05 mg/m³ is necessary to protect workers in construction, maritime, and agriculture from the significant risk of incurring these exposure-related effects, which OSHA considers material impairments of health within the meaning of the Act. In addition, promulgation of this limit will make OSHA's PEL for these substances consistent across all regulated sectors.

Preliminary Conclusion for Both Liver and Kidney Toxins

The health effects associated with occupational exposures to the hepato- and nephrotoxins shown in Tables C4–1 and C4–2 can be acute or chronic, reversible or irreversible, temporarily disabling or threatening to life. Workers in construction, maritime, and agriculture who experience chemically induced hepatotoxic or nephrotoxic effects may have enlarged livers, high blood pressure, hormonal imbalances, and/or organ necrosis. In addition, exposure to these substances is associated with a host of other adverse health effects, ranging from pulmonary irritation to cancer, and OSHA preliminarily concludes that the proposed limits are necessary to substantially reduce the risk of these effects as well. In addition, promulgation of these limits will make OSHA's PELs for these substances consistent across all regulated sectors.

5. Substances for Which Proposed Limits are Based on Avoidance of Ocular Effects

Introduction. Seven of the chemicals for which OSHA is proposing limits have the potential to cause serious ocular effects among workers in a variety of occupational settings. Some of the chemicals in this group are also sensory irritants and have other target organ effects; these substances have been placed in this section of the proposal because their limits are based primarily on their ability to cause permanent damage to the cornea, lens, or optic nerve of the eyes of exposed workers.

Table C5–1 lists these ocular toxins, along with their CAS numbers, an OSHA-assigned HS number that is used to facilitate indexing of the rulemaking docket, their 1987–1988 ACGIH TLV®s, and their NIOSH RELs (if any). In addition, Table C5–1 shows OSHA's current PELs for these substances in construction and maritime. (OSHA currently has no PELs in agriculture.) The right-hand column in Table C5–1 shows the PEL recently promulgated for each substance in the Agency's final Air Contaminants standard for general industry; these are the limits being proposed for these substances today in construction, maritime, and agricultural workplaces.

TABLE C5–1.—SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON AVOIDANCE OF OCULAR EFFECTS

H.S. No./chemical name	CAS No.-	Current OSHA PEL in construction and maritime	1987–1988 ACGIH TLV ®***	NIOSH REL /+/	Proposed OSHA PEL in construction, maritime, and agriculture
1172 N-Ethylmorpholine	100–74–3	20 ppm TWA, Skin.	5 ppm TWA, Skin		5 ppm TWA, Skin.
1209 Hydrogen sulfide	7783–06–4	10 ppm TWA	10 ppm TWA, 15 ppm STEL	10 ppm Ceiling (10 min).	10 ppm TWA, 15 ppm STEL.
2094 Hydroquinone	123–31–9	2 mg/m³ TWA	2 mg/m³ TWA	2 mg/m³ Ceiling (15 min).	2 mg/m³ TWA.
1252 Methyl alcohol	67–56–1	200 ppm TWA-	200 ppm TWA, 250 ppm STEL, Skin.	200 ppm TWA, 800 ppm Ceiling (15 min).	200 ppm TWA, 250 ppm STEL, Skin.
1266 Methyl silicate	681–84–5	5 ppm Ceiling	1 ppm TWA-		1 ppm TWA.
1282 Naphthalene	91–20–3	10 ppm TWA	10 ppm TWA, 15 ppm STEL		10 ppm TWA, 15 ppm STEL.
2138 Quinone (p-Benzoquinone)	106–51–4	0.5 ppm TWA	0.1 ppm TWA		0.1 ppm TWA.

*OSHA's TWA limits are for 8-hour exposures, its STELs are for 15 minutes unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time.

**OSHA's PELs do not currently apply in Agriculture [or in longshoring except in circumstances where the General Duty clause (Sec. 5(a)(1) of the Act) is cited].

***The ACGIH TLV®-TWA is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

+ NIOSH TWA limits are for 10 hour/day, 40 hour/week exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

For N-ethylmorpholine, OSHA's current PEL in construction, shipyards, and marine terminals is 20 ppm, with a skin notation; the Agency is proposing to reduce the 8-hour TWA PEL to 5 ppm and to retain the skin designation for this substance. For three of these substances—hydrogen sulfide, methyl alcohol, and naphthalene—OSHA is proposing to add short-term exposure limits to existing 8-hour TWA PELs, and for another substance, methyl silicate, OSHA is proposing to revise the current 5 ppm ceiling limit to a 1 ppm 8-hour TWA PEL. For hydroquinone, OSHA is retaining the PEL that currently exists in construction and maritime and proposing to extend it to agriculture, and for guinone, the Agency is proposing to reduce the limit to 0.1 ppm.

Description of the health effects. Damage to the eye caused by exposure to the chemicals in this group can occur in the form of corneal, lens, retinal, ganglion cell layer, or optic nerve effects. Depending on the severity of the exposure, individual susceptibility, and the particular chemical and circumstances involved, this damage may be transient, temporarily disabling, or permanently blinding.

Corneal effects. The cornea and conjunctiva are the outer surfaces of the eye and are thus directly exposed to external insults. Since the cornea must maintain transparency to remain functional, scar formation after injury to the cornea can destroy visual function completely. Recent evidence suggests that the transparency of the cornea is maintained by thin inner and outer boundary layers and that the death of these layers leads to loss of transparency (Potts 1986/Ex. 1–174). The corneal epithelium (outer layer) sometimes regenerates, depending on the depth of the burn or insult and the nature of the toxicant.

The vapors of some chemicals, including methyl silicate, produce painful corneal epithelial injuries that are associated with a delay in the onset of symptoms. These substances can continue to cause pain and loss of corneal epithelial cells for several hours after exposure. Typically, there is no discomfort during the actual exposure to the vapor, but several hours later, the eyes begin to burn, vision blurs, and conjunctival hyperemia, tearing, photophobia, and squinting occur (Grant 1986/Ex. 1–975). Possible mechanisms of action are enzyme inhibition, denaturing of other proteins, alteration of the DNA, and interference with the mitotic process; after a period of exposure, the affected cells die. Although the damaged epithelium sometimes regenerates after this type of injury, the damage can also involve the corneal stroma and endothelium, leading to scarring, vascularization, opacity, and loss of vision. The poor warning properties characteristic of these substances (i.e., their failure to cause immediate irritation and pain) make the establishment of protective exposure limits particularly important.

Exposure to the vapors of some of the substances in this group produces painless edema of the corneal epithelium, which can be accompanied by the delayed onset of visual haloes. A chemical that produces these effects is N-ethylmorpholine, a catalyst used to manufacture urethane foam. Painless edema generally occurs in workers who have been exposed for several hours to airborne concentrations that do not produce discomfort during the exposure itself. The visual effect produced by such exposures consists of the appearance of colored haloes around lights, an effect that is caused by the diffraction of light through the swollen epithelial cells of the eye. Visual haloes are severely distracting and restrict activity substantially, and the mechanism underlying this effect is not well understood (Grant 1986/Ex. 1–975).

Lens effects. The lens is a transparent, avascular tissue surrounded by a thin, collagenous capsule. The major portion of the lens is composed of long, thin fibers that form closely packed, onion-like layers. Transparency is dependent on several factors: a highly ordered cellular arrangement; fiber size, shape, and uniformity; molecular structure; and regularity of fiber packing (Potts 1986/Ex. 1–174). Interference with lens metabolism, with transport across cell boundaries, or with the integrity of the lens capsule itself can cause a loss of lens transparency and lead to decreased visual acuity (Potts 1986/Ex. 1–174). All such changes in lens transparency are referred to as cataracts.

Retinal effects. The retina is a compact neural structure that is responsible for converting the ocular light image to neural impulses. Because the retina is an internal structure, it is not generally affected by exposure to dust, splashes of liquids, or vapors. However, exposure to certain internally absorbed substances, such as methyl alcohol, may cause changes or lesions in the retina, including retinal edema or hemorrhage. Exposure to a few of the substances in this group can cause acute narrowing of the retinal arteries themselves, which can lead, in turn, to damage of the optic nerve and loss of vision.

Effects on ganglion cell layer and optic nerve. Below the retinal surface layer lies the ganglion cell layer, which is composed of the cell bodies of neurons that extend to the midbrain via the optic nerve. Ganglion cells may be damaged directly when a chemical acts on the cell bodies themselves or secondarily when a toxin destroys the optic nerve. Depending on the severity of the exposure, loss of visual acuity or vision may ensue.

Dose-Response Relationships and Ocular Effects

For most of the chemicals shown in Table C5–1, limits have been proposed on the basis of research in animals, health surveys, and case reports of injuries occurring in occupationally exposed populations. These studies indicate that exposures to concentrations of these substances at levels above the no-observed-effect level cause pain and may damage the eyes of exposed workers. For some of these substances, the available human data are limited, and evidence from animal studies or knowledge of a chemical's structural analogy to another chemical known to have ocular effects provides the basis for the proposed exposure limit. Animal models are generally good predictors of ocular effects in humans because the eyes of rodents, especially those of guinea pigs and rabbits, closely resemble human eyes. Thus, animal studies on the effects of chemical exposure on the eye can be relied on to predict accurately how the chemicals that produce these effects in animals will behave in workers exposed in industrial situations. For the seven chemicals in this group of ocular toxins, the available toxicologic data and OSHA's preliminary findings are described below.

N-ETHYLMORPHOLINE

CAS: 100–74–3; Chemical Formula: $C_6H_{13}NO$

H.S. No. 1172

In construction, shipyards, marine terminals, and long-shoring, the current OSHA PEL for N-ethylmorpholine is 20 ppm as an 8-hour TWA, with a skin notation. OSHA has no PEL for this substance in agriculture, and NIOSH has no REL. OSHA is retaining the skin notation and proposing an 8-hour TWA PEL of 5 ppm for this substance in construction, maritime, and agriculture. The proposed PEL and skin notation are consistent with the current ACGIH TLV® for N-ethylmorpholine; in the prior rulemaking, NIOSH concurred (Ex. 8–47, Table N1) that this limit and notation were appropriate. Promulgation of this PEL for N-ethylmorpholine would make

the PEL for this substance consistent across all OSHA-regulated sectors.

N-Ethylmorpholine is a colorless, flammable liquid with an ammonia-like odor; it is used as a catalyst in urethane manufacture, an intermediate in the production of dyestuffs, pharmaceuticals, rubber accelerators, and emulsifiers, and as a solvent in many applications (HSDB 1990).

N-Ethylmorpholine's acute toxicity in animals is relatively low; the oral LD_{50} in rats is 1780 mg/kg, and the LC_{50} in mice is 18,000 mg/m ^{3} for 2 hours (RTECS 1990). However, the vapors of this substance have high toxicity for the eye (Smyth, Carpenter, Weil, and Pozzani 1954). When N-ethylmorpholine was instilled into rabbit eyes, it caused the cornea to become hazy within 5 minutes and the epithelium covering the eye to slough off (Smyth, Carpenter, Weil, and Pozzani 1954). The injury to rabbits' eyes caused by N-ethylmorpholine was graded 7 on an ascending severity scale of 1 to 10 (Smyth, Carpenter, Weil, and Pozzani 1954).

Overexposure to fairly low concentrations of the vapors from this substance causes corneal edema, blue-gray vision, and colored haloes, as well as irritation of the eyes, skin, mucous membranes, and upper respiratory tract. Typically, vision becomes misty and haloes appear a few hours after workers have been exposed to N-ethylmorpholine vapors for a period of hours. Distortion of vision can occur even at levels considerably lower than those that cause irritation (Mastromatteo 1965/Ex. 1–146).

Reversible corneal edema has been observed in workers exposed to 40 ppm or more of N-ethylmorpholine for several hours (Dernehl 1966). Workers routinely exposed to 3- to 4-ppm concentrations but never to concentrations above 11 ppm complained of haloes and foggy vision as well as drowsiness (ACGIH 1986/Ex. 1–3, p. 263). The irritant effects of N-ethylmorpholine were also seen in a controlled-exposure experiment involving volunteer subjects. Ten volunteers exposed for 2.5 minutes to a concentration of 100 ppm experienced irritation of the eyes, nose, and throat; those exposed for 2.5 minutes to a 50-ppm concentration experienced slight irritation; and no irritation was reported after exposure for 2.5 minutes to 25 ppm (ACGIH 1986/Ex. 1–3, p. 263).

OSHA's current 20-ppm PEL for N-ethylmorpholine in construction and maritime is not adequate to protect exposed workers against the occurrence of corneal edema and other adverse visual effects. Because corneal edema is both painless as it is developing and does not manifest symptoms for several hours after exposure, workers are likely to be unaware of the danger of exposure. This lack of warning properties is particularly hazardous because the effects on visual function of repeated exposure of the eyes to substances that cause corneal edema are not known.

Based on this evidence, OSHA preliminarily finds that the proposed PEL of 5 ppm as an 8-hour TWA is necessary to protect workers in construction, maritime, and agriculture from N-ethylmorpholine's injurious effects on the eyes. In addition, the Agency is retaining the skin notation because this substance can be absorbed through the skin in systemically toxic amounts (Genium MSDS 1986, No. 589). OSHA believes that the proposed PEL is necessary to reduce the significant risk of material health impairments, which are manifested as corneal edema, visual distortion, and other adverse visual effects, that are associated with occupational exposure to this substance. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

HYDROGEN SULFIDE

CAS: 7783–06–4; Chemical Formula: H_2S
H.S. No. 1209

OSHA's current limit for hydrogen sulfide in construction and maritime is 10 ppm as an 8-hour TWA; there is no PEL for this substance in agriculture. The ACGIH TLV®s for hydrogen sulfide are 10 ppm as an 8-hour TWA and 15 ppm as a 15 minute STEL; NIOSH has a 10-ppm, 10-minute REL for this substance. OSHA is retaining the 8-hour TWA of 10 ppm in construction and maritime and proposing to add a STEL of 15 ppm in these two sectors; the Agency is also proposing to extend both the 8-hour TWA and the STEL to agriculture. Promulgation of these PELs will make OSHA's limits for hydrogen sulfide consistent across all regulated sectors.

Hydrogen sulfide is a colorless, flammable gas with the odor of rotten eggs. It is widely used as a chemical intermediate, an analytical reagent, and in the manufacture of heavy water in the utilities sector. In agriculture, it is used as a disinfectant (HSDB 1985). It is also generated inadvertently by the fermentation of animal manure. Many farm workers have been exposed to this substance while working in the vicinity of liquid manure storage pits and have been asphyxiated as a consequence (Osbern and Crapo 1981). Hydrogen sulfide also is encountered in natural oil and gas deposits and in sewers, caissons, tunnels, and other construction sites (Grant 1986, p. 495). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Hydrogen sulfide's effects on experimental animals are similar to those seen in exposed workers: conjunctivitis, pulmonary irritation, acute poisoning, and death by chemical asphyxiation (Klaassen, Amdur, and Doull 1986, p. 242; Grant 1986, p. 496). The LC_{50} in mice is 673 ppm for 1 hour (RTECS 1990). A number of experiments have demonstrated that exposure to hydrogen sulfide concentrations of 50 to 100 ppm for several hours (or sometimes longer) causes damage to the corneal epithelium of dogs, cats, rabbits, and guinea pigs; animals are believed to be somewhat less sensitive than humans to hydrogen sulfide's ocular effects but may be more sensitive to its pulmonary effects (Grant 1986, p. 496).

When inhaled at concentrations exceeding 500 ppm, exposure to hydrogen sulfide has caused respiratory paralysis and death. Acutely poisoned individuals who recover may experience headaches, fatigue, dizziness, and nystagmus; eventually, however, recovery is usually complete (Milby 1962; NRC 1979). The 1986 ACGIH Documentation (Ex. 1–3, p. 318) cites several reports (Brieger 1964; Kranenburg and Kessener 1935; Masure 1950, in Carson 1963; Elkins 1950a/Ex. 1–953) of the occurrence of adverse ocular effects, including conjunctivitis, caused by exposure to 20 ppm or less of hydrogen sulfide. A study by Poda and Aiken (1966/Ex. 1–115) reports that the adoption of a voluntary limit of 10 ppm in two heavy-water plants eliminated exposure problems at those facilities. An early study by Flury and Zernik (1931f) reports that the conjunctivitis caused by the exposure of volunteers to 10 to 15 ppm of hydrogen sulfide for six hours endured for several days; however, this substance is not known to have caused irreversible eye damage.

In the earlier Air Contaminants rulemaking, OSHA received several comments related to the health effects and proposed limits for hydrogen sulfide (Exs. 3–1163, 3–216, 8–37, 8–47, 129; Tr. XI, pp. 114, 225). One commenter, the Edison Electric Institute (EEI) (Tr. XI, p. 225), described the exposures of utility workers to hydrogen sulfide, and another, the Montana Sulphur and Chemical Corporation (Ex. 3–216), stated that, in its opinion, the evidence for the proposed limits was not "compelling."

Because Montana Sulphur and Chemical itself had never, in its long experience of manufacturing and handling this "notoriously toxic" substance, had a case of eye irritation that required medical treatment, it urged OSHA (Ex. 3–216) to promulgate a STEL for hydrogen sulfide in the range of 25 to 30 ppm rather than at the proposed level of 15 ppm.

In response to this commenter, OSHA noted (54 *FR* 2494) that the proposed STEL of 15 ppm was based on the best available evidence (i.e., a report that workers exposed to this substance at this level had not experienced eye irritation, while workers exposed to higher levels had experienced such effects), as well as reports in the industrial hygiene literature that levels below 20 ppm cause eye irritation (Brieger 1964; Kranenburg and Kessener 1935; Masure 1950, in Carson 1963; Elkins 1950a/ Ex.1–853). For example, the author of the best-known general source on the toxicology of the eye (Grant 1986) states that "where the concentration [of hydrogen sulfide] is regularly kept below 10 ppm in air, it is rare to have any irritation of the eyes" (p. 496). OSHA believes that the proposed STEL will ensure that concentrations are maintained close to the 8-hour TWA and that excursions above irritant levels are thus minimized.

Based on this evidence, OSHA preliminarily finds that the current 10-ppm 8-hour TWA limit alone does not adequately protect workers in construction and maritime against the adverse ocular effects associated with exposure to concentrations of hydrogen sulfide above 10 ppm. Furthermore, agricultural workers are not presently protected by any limit. OSHA believes that the eye irritation and conjunctivitis associated with such exposures represent a significant risk of material health impairment to workers in construction, maritime, and agriculture because these workers may experience pain and other ocular effects and be forced to seek medical treatment after such exposures. OSHA is accordingly retaining the 8-hour TWA limit of 10 ppm for hydrogen sulfide in construction and maritime and is proposing to establish a short-term limit of 15 ppm in these two sectors. The Agency is also proposing to extend both limits to agriculture. The Agency preliminarily finds that these limits are necessary to provide protection from the significant risk of exposure-related ocular effects, including conjunctivitis, corneal edema, and distortion of vision, associated with occupational exposure to hydrogen sulfide. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

HYDROQUINONE

CAS: 123–31–9; Chemical Formula: $C_6H_4(OH)_2$

H.S. No. 2094

OSHA's current limit for hydroquinone in general industry, construction, and maritime is 2 mg/m³ as an 8-hour TWA. The Agency has no PEL for hydroquinone in agriculture. The ACGIH TLV®–TWA for this substance is 2 mg/m³, and the NIOSH REL is 2 mg/m³ as a 15-minute ceiling. OSHA is proposing a PEL for hydroquinone of 2 mg/m³ as an 8-hour TWA in agriculture. Promulgation of this limit will make the PEL for hydroquinone consistent across all OSHA-regulated sectors.

Hydroquinone is a white, crystalline solid that is used primarily in photographic developers. It is also used as an antioxidant or stabilizer in polymers, oils, fuels, and paints, and as a dye intermediate. Hydroquinone derivatives are reported to be effective antimitotic and tumor-inhibiting agents (ACGIH 1986, p. 319; Clayton and Clayton 1981, p. 2589).

Exposure to hydroquinone causes skin and eye irritation, skin depigmentation, respiratory symptoms, and ocular damage. The oral LD_{50} in rats is 320 mg/kg; the dermal LD_{50} in an unspecified species of mammal is 5970 mg/kg (RTECS 1990). Before death, acutely poisoned animals exhibit an increase in motor activity, an increase in the formation of methemoglobin, hypersensitivity to external stimuli, labored breathing, hyperactive reflexes, and cyanosis. These initial signs of poisoning are followed by clonic convulsions, exhaustion, loss of reflexes, lowered body temperature, paralysis, coma, and death (Clayton and Clayton 1981, p. 2590). Pigment accumulation in the conjunctiva and cornea has been induced in rabbit eyes after 2 to 4 months of daily application of powdered hydroquinone to the eyes. Scarring and gray opacification were also noted in this study (Grant 1986, p. 499).

Industrial exposure to hydroquinone occurs during the manufacture and use of this substance and involves not only the dust of hydroquinone but its vapor. Acute exposure both to quinone and hydroquinone causes conjunctival irritation. In a study of workers manufacturing hydroquinone, eye injuries developed gradually over a period of years in workers exposed for fewer than 5 years. Although quinone was probably the main cause of these injuries, hydroquinone was considered a contributory cause. No systemic effects were associated with these exposures (Anderson and Oglesby 1958; Sterner, Oglesby, and Anderson 1947). Skin irritation, allergic sensitization, depigmentation, and dermatitis have reportedly occurred following repeated skin contact with creams containing 5 percent or more hydroquinone (NIOSH 1978).

A group of 24 workers in a Swedish movie film laboratory who used hydroquinone-containing and other color film developing agents developed occupational dermatitis. Patch testing confirmed that hydroquinone was the allergenic agent in one of these workers (Liden, Brehmer-Andersson 1988). Another photography laboratory worker developed vitiligo (depigmentation of the skin) caused by hydroquinone. This worker had been dipping x-ray films into hydroquinone with his bare hands for 10 years before he developed this condition (Das and Tandon 1988).

Chronic exposure to quinone vapor and hydroquinone dust causes a brownish discoloration of the conjunctiva and cornea, small opacities of the cornea, and structural changes of the cornea that result in the loss of visual acuity. The structural corneal damage consisted of changes in the curvature of the lens; such curvature can lead to progressive loss of visual acuity and blindness (Anderson and Oglesby 1958; Sterner, Oglesby, and Anderson 1947). Exposure to hydroquinone dust concentrations as high as 20 to 30 mg/m³ produced no systemic effects, and volunteers showed no signs of toxicity after ingesting 300 to 500 mg hydroquinone daily for up to 5 months (Carlson and Brewer 1953; Sterner, Oglesby, and Anderson 1947). In addition to ocular and skin effects, exposure to hydroquinone causes respiratory symptoms. Thirty-three workers exposed to hydroquinone, trimethyl hydroquinone, and retinene-hydroquinone, in a chemical plant showed a higher prevalence of respiratory symptoms than unexposed workers from the same plant, and the exposed group also showed significantly lower pulmonary function values. Because these workers had significantly higher levels of immunoglobulin G and elevated levels of immunoglobulin E compared to in-plant controls, the authors concluded that exposure to hydroquinone causes ventilatory impairment, perhaps by an immunological mechanism (Choudat, Neukirch, Brochard, Barrat, Marsac, Conso, and Philbert 1988).

Based on this evidence, OSHA is proposing a 2 mg/m³ 8-hour TWA limit to protect workers in agriculture from

the significant risk of skin and respiratory irritation, skin depigmentation, and ocular damage associated with exposure to hydroquinone. OSHA considers these effects material impairments of health and preliminarily concludes that this limit is necessary to substantially reduce the significant risk of these impairments in exposed agricultural workers. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

METHYL ALCOHOL

CAS: 67–56–1; Chemical Formula: CH_3OH

H.S. No. 1252

OSHA's current 8-hour TWA limit for methyl alcohol in construction and maritime is 200 ppm as an 8-hour TWA. There is no PEL for this substance in agriculture. The ACGIH TLVs® for methyl alcohol are an 8-hour TWA of 200 ppm, a STEL of 250 ppm, and a skin notation. The NIOSH REL for methyl alcohol is 200 ppm as a TWA and 800 ppm as a STEL; however, after reviewing the health evidence for methyl alcohol, NIOSH concurred (Ex. 8–47, Table N1) with the PEL OSHA is proposing. OSHA is retaining the 8-hour TWA PEL of 200 ppm in construction and maritime, proposing to add a STEL of 250 ppm for this substance in these two sectors, and additionally proposing both limits in agriculture. In addition, OSHA is proposing to add a skin notation to the limit for methyl alcohol.

Methyl alcohol is a mobile, highly polar, flammable liquid that is widely used as an industrial solvent in enamels, paints, stains, varnish removers, cleaners, and antifreezes (Clayton and Clayton 1982, p. 4529).

Methyl alcohol has severe ocular effects in animals, both when in contact with the eye itself and when ingested. The oral LD_{50} in rats is 5628 mg/kg, and the LD_{50} in the same species is 64,000 ppm for 4 hours (RTECS 1990). The dermal LD_{50} in rabbits is 15,800 mg/kg (RTECS 1990). Three of six rabbits developed a moderate degree of corneal opacity when undiluted methyl alcohol was instilled into their eyes, and all six animals developed conjunctivitis as a result of eye contact with the liquid (Clayton and Clayton 1982, p. 4531). Dogs killed by the inhalation of acute doses of methyl alcohol (37,000 ppm for 8 hours) showed, at autopsy, hyperemia of the choroid and edema of the ocular tissue, as well as early signs of retinal degeneration (Tyson and Schoenberg 1914). Methyl alcohol has also been shown to have embryotoxic, developmental, and reproductive effects in animals of several species. In monkeys, the lowest lethal concentration by inhalation is 1000 ppm and the lowest lethal dermal dose is 393 mg/kg (RTECS 1990).

Workers exposed to methyl alcohol at concentrations between 200 and 375 ppm experience severe recurrent headaches and, at higher levels, the visual capacity of exposed individuals is diminished. The lowest reported lethal concentration in humans is 868 mg/kg (RTECS 1990). The metabolites believed to be chiefly responsible for methyl alcohol's toxic effects are formic acid and formaldehyde, which cause acidosis (Rom 1983, p. 523). If the poisoning is severe, impairment of vision and death may occur; these effects have occurred after severe ingestion, inhalation, and dermal absorption (NIOSH 1976; Henson 1960). Chronic exposure to airborne concentrations of methyl alcohol of 1200 to 8300 ppm can cause impaired vision (NIOSH 1976). Splashed into the eyes, methyl alcohol causes reversible corneal injury; in prolonged contact with the skin, it causes defatting, scaling, and dermatitis (NIOSH 1976).

In the prior rulemaking, several commenters submitted information to the record on methyl alcohol (Exs. 150, 194, 3–661, 3–902, and 3–896). Both the Eastman Kodak Company (Ex. 3–661) and the Chevron Corporation (Ex. 3–896) objected to the STEL of 250 ppm because they believe that no STEL is warranted for methyl alcohol. However, OSHA rejected the reasoning of these commenters (see 54 FR 2495), noting that, in addition to those studies specifically cited in the preamble, many other studies report that exposure to methyl alcohol at levels that cause headaches is possible even when the 8-hour limit of 200 ppm is being observed.

Based on this evidence, OSHA preliminarily finds that the proposed 250-ppm STEL is necessary because the current 8-hour PEL of 200 ppm alone cannot protect workers in construction, maritime, and agriculture from exposure to short-term peaks at levels that cause eye irritation and severe, recurrent headaches. OSHA is proposing to add a skin notation to the PEL for methyl alcohol because a dermal dose of only 393 mg/kg has caused death in monkeys; this dose is well below the dermal LD_{50} of 1000 mg/kg adopted by OSHA as the cutoff for skin designations (see Section C.15 of this preamble) (RTECS 1990). The Agency preliminarily concludes that the proposed 8-hour TWA and 15-minute STEL will together reduce the significant risk of headaches and blurred vision presented by short-term occupational exposures to methyl alcohol at concentrations above these levels. In addition, the proposed skin notation will reduce the likelihood that exposed workers in construction, maritime, and agriculture will experience the systemic toxicity caused by absorption of this substance through the skin that has been demonstrated to occur in experimental animals. The Agency preliminarily finds that the headaches, blurred vision, other ocular effects, and systemic toxicity associated with exposure to methyl alcohol constitute material impairments of health. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

METHYL SILICATE

CAS: 681–84–5; Chemical Formula: $(CH_3O)_4Si$

H.S. No. 1266

OSHA currently has a ceiling limit of 5 ppm for methyl silicate in construction and maritime; there is no PEL for this substance in agriculture. The ACGIH TLV® for this substance is 1 ppm as an 8-hour TWA. OSHA is proposing an 8-hour TWA PEL of 1ppm for workplaces in construction, maritime, and agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) that this limit, which was recently established in general industry, is appropriate.

Methyl silicate is a colorless liquid or a crystalline solid. It is used as an intermediate and to coat television tubes.

The toxic action of methyl silicate has been compared to that of mustard gas, both in terms of severity and the delayed onset of signs and symptoms (Grant 1986, p. 627). When a single drop of methyl silicate was placed into the eye of a rabbit, the resulting injury was graded 9 out of a possible 10 on an ascending scale of severity (Grant 1986, p. 627). The corneal epithelium of the eye of the rabbit became permanently opacified (Grant 1986, p. 627). –Rabbits exposed to 1000 ppm of methyl silicate in dry air experienced delayed eye burns (ACGIH 1986/Ex. 1–3, p. 409). Exposure of these animals to a methyl silicate concentration of approximately 15,000 ppm for five minutes caused eye burns, but exposure to this level for four minutes caused no appreciable effect. Guinea pigs showed maximum no-effect levels of 135 ppm for 15 minutes, 90 ppm for one hour, and 20 ppm for 8 one-hour periods. The latency period for ocular changes was 16 hours for serious effects and up to three days for mild involvement (ACGIH 1986/Ex. 1–3, p. 409).

It is estimated that exposing humans to methyl silicate at concentrations of

200 to 300 ppm for 15 minutes will produce ocular lesions and that exposure to 1000 ppm for this period will produce injury requiring hospitalization (ACGIH 1986/Ex. 1–3, p. 409). There is one report of blindness caused by a splash of this substance into the eyes (Grant 1986, p. 628).

Based on this evidence, OSHA preliminarily concludes that the proposed 8-hour TWA limit of 1 ppm is necessary to protect workers in construction, maritime, and agriculture from the ocular effects associated with exposure to this substance. The basis for OSHA's reasoning is that evidence has shown that (1) exposures of 200–300 ppm for 15 minutes produce only mild eye effects in humans (ACGIH 1986/Ex. 1–3, p. 409) and (2) that exposure to a concentration of 90 ppm for 1 hour, or to 20 ppm for 8 one-hour periods, produces no damage to the eyes of guinea pigs. The proposed 8-hour TWA PEL of 1 ppm, which is more than a factor of 20 below the lowest-observed-effect level, will ensure that workers in construction, maritime, and agriculture are protected from the significant risks of such material health impairments. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

NAPHTHALENE

CAS: 91–20–3; Chemical Formula: $C_{10}H_8$

H.S. No. 1282

OSHA's current exposure limit for naphthalene in construction and maritime is 10 ppm as an 8-hour TWA. The proposal retains this TWA, proposes to add a short-term limit of 15 ppm, and additionally proposes to extend both the TWA and the STEL to agriculture. These limits agree with the ACGIH's TLV®s. There is no limit in agriculture. NIOSH has no REL; however, in the prior rulemaking, NIOSH (Ex. 8–47, Table N1) concurred with the PELs selected by OSHA for this substance, which are those recently established in general industry.

Naphthalene is a white, crystalline substance that imparts the characteristic "moth-ball" odor to commercial mothball products (HSDB 1985). Naphthalene is an important chemical intermediate that is also used in dyes, explosives, lubricants, tanning agents, and pesticides (HSDB 1985).

In animals, naphthalene causes hemolysis (destruction of the red blood cells), tumors of the blood, and developmental effects (RTECS 1990), as well as ocular toxicity (Grant 1986). Cataracts and severe eye irritation have been induced in experimental animals by administration of this substance (Grant 1986). The oral LD_{50} in rats is 490 mg/kg (RTECS 1990).

In humans, the inhalation of naphthalene vapor causes eye irritation, headache and nausea (Proctor, Hughes, and Fischman 1988, p. 358). The lethal dose in humans has been reported as 50 mg/kg (NIOSH 1977i/Ex. 1–1182). In contact with the eye, naphthalene causes conjunctivitis, corneal injury, chorio-retinitis, scotoma, and decreased visual acuity (Proctor, Hughes, and Fischman 1988, p. 358). Eight of 21 workers exposed to unspecified levels of naphthalene for 5 years developed opacities of the lens of the eye (Grant 1986). Ingestion of large amounts of naphthalene causes severe hemolytic anemia and hemoglobinuria (Stokinger and Mountain 1963/Ex. 1–765). Exposure to a 15-ppm concentration of the vapor is reported to cause marked eye irritation (Grant 1986).

In the prior rulemaking, the American Iron and Steel Institute commented that, in its opinion, no STEL was warranted for naphthalene. OSHA responded (54 FR 2496) to this comment by pointing to the study by Robbins (1951/Ex. 1–799), which clearly shows that excursions as high as 15 ppm cause severe eye irritation and concluded that the proposed STEL was thus both necessary and appropriate.

Accordingly, OSHA is retaining the 8-hour TWA of 10 ppm in construction and maritime, proposing to add a 15-minute STEL of 15 ppm in these sectors, and proposing to extend both PELs to agriculture. The Agency preliminarily concludes that these limits will protect workers in construction, maritime, and agriculture from the significant risks of eye irritation and cataracts, which constitute material health impairments that are potentially associated with exposure to levels above the current limit. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

QUINONE

CAS: 106–51–4; Chemical Formula: $C_6H_4O_2$

H.S. No. 2138

In general industry, construction, and maritime, OSHA's permissible exposure limit for quinone is 0.5 ppm as an 8-hour TWA. There is no limit for this substance in agriculture. The ACGIH has a current TLV®–TWA limit of 0.1 ppm for this substance. NIOSH has no REL but concurs (EX. 8–47, Table N3A) with the limit being proposed. OSHA is proposing to establish a PEL of 0.1 ppm for quinone in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Quinone is a yellow crystalline solid with a penetrating, chlorine-like odor. This substance is used as an intermediate in the manufacture of fungicides, hydroquinone, and dyes, in photography, and as an oxidizing agent and analytical reagent (ACGIH 1986, p. 509; Hawley's 1987, p. 991).

Quinone causes severe eye and skin irritation in humans; in animals it causes lung and kidney damage and convulsions. The oral LD_{50} in rats is 130 mg/kg (RTECS 1990). Large oral or subcutaneous doses of quinone cause local irritation, convulsions, respiratory difficulty, a decrease in blood pressure, and death due to paralysis of the medullary centers (Clayton and Clayton 1981, p. 2593). A major cause of death in acutely poisoned animals is asphyxia due to pulmonary damage and quinone's effects on hemoglobin (Deichman and Keplinger 1963, in Clayton and Clayton 1981, p. 2593). Signs of kidney injury have also been observed in severely poisoned animals (Deichman and Keplinger 1963, Clayton and Clayton 1981, p. 2593). Quinone has been tested for carcinogenicity in mice by skin application and inhalation and in rats by subcutaneous injection. The International Agency for Research on Cancer (IARC) has concluded, however, that the evidence in animals is insufficient to evaluate quinone's carcinogenicity (IARC 1977, Vol. 15, p. 260).

Quinone causes ocular and cutaneous lesions in humans; however, no cases of systemic poisoning have been reported. In contact with the skin, quinone causes discoloration, severe irritation, erythema, swelling, and papules and vesicles; prolonged contact can cause necrosis of the skin (Clayton and Clayton 1981, p. 2594). Eye irritation becomes noticeable on exposure to concentrations above 0.1 ppm; acute exposures cause conjunctival irritation, corneal edema, ulceration, and scarring (AIHA Hygienic Guide Series 1978). Exposure to quinone vapor produced characteristic chronic injuries in workers in a hydroquinone manufacturing facility. The effects of exposure developed gradually; no serious effects occurred in individuals exposed to the vapor for less than 5 years. Signs and symptoms in those exposed for longer than 5 years included pigmentary changes of the conjunctiva and cornea, corneal opacities, and loss of visual acuity (Sterner, Oglesby, and Anderson 1947). Although the pigmentary changes induced by quinone are reversible, the other ocular effects

caused by exposure to this substance sometimes progress even after exposure has ceased (Proctor, Hughes, and Fischman 1988, p. 431).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to quinone causes severe skin and eye injury, as well as pulmonary damage. The Agency believes that, in the absence of a limit, workers in agriculture are at risk of experiencing quinone's adverse effects and that establishing an 8-hour TWA of 0.1 ppm for quinone in agriculture is necessary to reduce these risks of material health impairment. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Preliminary Conclusions. OSHA believes that adoption of the limits being proposed for this group of chemicals, which have the potential to cause adverse ocular effects ranging from transient discomfort to permanent blindness, will substantially reduce the risk of visual impairment associated with exposure to these substances. The toxicological bases for the proposed limits include evidence derived from occupationally exposed workers and results obtained in animal models that have been shown to be excellent predictors of human responses. The risks being protected against have serious consequences, both in terms of the health and functional capacity of the exposed workers themselves and the safety and well-being of these workers and their co-workers.

The available health evidence for the substances described in this section forms a reasonable basis for proposing the revision or addition of these limits. At the time of the final rule, OSHA will promulgate new or revised limits for these substances if the Agency determines that these limits will reduce significant risk. Promulgation of the proposed limits will also make OSHA's limits for these substances consistent across all regulated sectors.

6. Substances for Which Proposed Limits Are Based on Avoidance of Respiratory Effects

Introduction. For workplaces in construction, maritime, and agriculture, limits are being proposed for a total of 44 substances or materials for which exposure has been shown to cause adverse respiratory effects. The substances in this group cause acute pulmonary edema, alveolar damage, or chronic respiratory damage through the general mechanisms of cellular damage or fibrosis. At sufficient doses, these effects can be permanent, disabling, and life-threatening.

Some of the materials in this group are composites of naturally occurring minerals; for these substances, the Agency is proposing limits based on the most hazardous component. For several materials (coal dust, crystalline tripoli, silica, and graphite), OSHA is proposing that the TWA be measured as the respirable quartz fraction of the dust, because it is exposure to this fraction that presents the greatest risk to exposed workers in these sectors.

Table C6–1 lists the substances in this group, along with their current PELs in construction and maritime and their CAS and HS numbers. The 1987–1988 TLV®s, NIOSH RELs, and limits being proposed for these substances in construction, maritime, and agriculture are also shown on Table C6–1. With the exception of limits for asphalt fumes, fibrous glass, and mineral wool, the limits being proposed are those in effect in general industry. For these three substances, OSHA is proposing PELs in general industry as well as in construction, maritime, and agriculture.

TABLE C6–1.—SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON AVOIDANCE OF RESPIRATORY EFFECTS

H.S. No./chemical name	CAS No.	Current OSHA PEL in construction and maritime*	1987–1988 ACGIH TLV® **	NIOSH REL***	Proposed OSHA PEL in construction, maritime, and agriculture*
1017 Aluminum (pyro powders)	7429–90–5		5 mg/m³ TWA		57774 mg/m³ TWA.
1028 Asphalt fumes	8052–42–4		5 mg/m³ TWA	5 mg/m³ Ceiling (15-min) (Total particulate).	5 mg/m³ TWA.
1034 Bismuth telluride (Se-doped).	1304–82–1		5 mg/m³ TWA		5 mg/m³ TWA.
2027 Carbon black	1333–86–4	3.5 mg/m³ TWA	3.5 mg/m³ TWA	3.5 mg/m³ TWA; if PAHs are present, 0.1 mg/m³ TWA.	3.5 mg/m³ TWA.
1080 Chlorine dioxide	10049–04–4	0.1 ppm TWA	0.1 ppm TWA, 0.3 ppm STEL		0.1 ppm TWA, 0.3 ppm STEL
2038 Chromium (II) compounds (as Cr).	7440–47–3	0.5 mg/m³ TWA	0.5 mg/m³ TWA		0.5 mg/m³ TWA.
2038A Chromium (III) compounds (as Cr).	7440–47–3	0.5 mg/m³ TWA	0.5 mg/m³ TWA		0.5 mg/m³ TWA.
1093 Chromium metal (as Cr)	7440–47–3	1 mg/m³ TWA	0.5 mg/m³ TWA		1 mg/m³ TWA.
1096 Coal dust, < 5% quartz	None	2.4 mg/m³ TWA	2 mg/m³ TWA		2 mg/m³ TWA.
1097 Coal dust, > 5% quartz	None	10 mg/m³/% SiO_2+2.	0.1 mg/m³ TWA		0.1 mg/m³ TWA.
2041 Cotton dust	None	1 mg/m³ TWA	0.2 mg/m³ TWA	200 µg/m³ TWA lint-free cotton dust.	0.5 mg/m³ TWA.
1161 Ethyl acrylate	140–88–5	25 ppm TWA, Skin.	5 ppm TWA, 25 ppm STEL, Skin		5 ppm TWA 25 TWA.
1177 Ferrovanadium dust	12604–58–9	1 mg/m³ TWA, 3 mg/m³ STEL.	1 mg/m³ TWA	1 mg/m³ TWA, 3 mg/m³ STEL.	1 mg/m³ TWA.
1178 Fibrous glass	None		10 mg/m³ TWA	5 mg/m³ TWA total fibrous glass.	1 f/cc TWA.
1190 Grain dust (oat, wheat, barley).	None		10 mg/m³ TWA		10 mg/m³ TWA.
1191 Graphite, natural, respirable <1% quartz.	7782–42–5	15 mppcf TWA	2.5 mg/m³ TWA		2.5 mg/m³ TWA.
1213 Indium & compounds	7440–74–6	0.1 mg/m³ TWA	0.1 mg/m³ TWA		0.1 mg/m³ TWA.
1215 Iron oxide (dust and fumes)	1309–37–1	10 mg/m³ TWA	5 mg/m³ TWA		10 mg/m³ TWA.

TABLE C6-1.—SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON AVOIDANCE OF RESPIRATORY EFFECTS—Continued

H.S. No./chemical name	CAS No.	Current OSHA PEL in construction and maritime*	1987-1988 ACGIH TLV* **	NIOSH REL***	Proposed OSHA PEL in construction, maritime, and agriculture*
1272 Methylene bis(4-Cyclohexylisocyanate).	5124-30-1		0.01 ppm Ceiling		0.01 ppm Ceiling.
1276 Mica, respirable dust containing < 1% quartz.	12001-26-2	20 mppcf TWA	3 mg/m³ TWA		3 mg/m³ TWA.
1277 Mineral wool fiber	None		10 mg/m³ TWA		1 f/cc TWA.
1283 Nickel (soluble compounds)	7440-02-0	1 mg/m³ TWA	0.1 mg/m³ TWA	0.015 mg/m³ TWA+ (inorganic compounds).	0.1 mg/m³ TWA.
1289 Nitrogen dioxide	10102-44-0	5 ppm Ceiling	3 ppm TWA, 5 ppm STEL	1 ppm Ceiling (15-min).	1 ppm STEL.
1300 Oxygen difluoride	7783-41-7	0.05 ppm TWA	0.05 ppm Ceiling		0.05 ppm Ceiling.
1301 Ozone	10028-15-6	0.1 ppm TWA	0.1 ppm TWA, 0.3 ppm STEL		0.1 ppm TWA, 0.3 ppm STEL.
1303 Paraquat, respirable dust	4685-14-7	0.5 mg/m³ TWA, Skin.	0.1 mg/m³ TWA		0.1 mg/m³ TWA, Skin.
1352 Silica, amorphous, diatomaceous earth.	61790-53-2	20 mppcf	10 mg/m³ TWA		6 mg/m³ TWA.
1353 Silica, amorphous, precipitate and gel.	112926-00-8		10 mg/m³ TWA		6 mg/m³ TWA.
1354 Silica, crystalline cristobalite	14464-46-1	250/% SiO_2+5 (as mppcf).	0.05 mg/m³ TWA	50 μg/m³ TWA	0.05 mg/m³ TWA.
2142 Silica, crystalline quartz (total dust).	14808-60-7		0.3 mg/m³ TWA	50 μg/m³TWA (respirable free silica).	0.3 mg/m³ TWA.
1355 Silica, crystalline quartz, respirable.	14808-60-7	250/% SiO_2+5 (as mppcf).	0.1 mg/m³ TWA	50 μg/m³ TWA	0.1 mg/m³ TWA.
1356 Silica, crystalline tridymite (as respirable quartz dust).	15468-32-3	250/% SiO_2+5 (as mppcf).	0.05 mg/m³ TWA	50 μg/m³ TWA	0.05 mg/m³ TWA.
1357 Silica, crystalline tripoli (as respirable quartz dust).	1317-95-9	250/% SiO_2+5 (as mppcf).	0.1 mg/m³ TWA	50 μg/m³ TWA	0.1 mg/m³ TWA.
1358 Silica, fused	60676-86-0	250/% SiO_2+5 (as mppcf).	0.1 mg/m³ TWA (respirable dust)		0.1 mg/m³ TWA.
1363 Soapstone, total dust	None	20 mppcf TWA	6 mg/m³ TWA		6 mg/m³ TWA.
1363A Soapstone, respirable dust	None		3 mg/m³ TWA		3 mg/m³ TWA.
1375 Sulfur dioxide	7446-09-5	5 ppm TWA	2 ppm TWA, 5 ppm STEL	0.5 ppm TWA	2 ppm TWA, 5 ppm STEL.
1378 Sulfur tetrafluoride	7783-60-0		0.1 ppm Ceiling		0.1 ppm Ceiling.
1381 Talc (containing no asbestos).	14807-96-6	20 mppcf TWA	2 mg/m³ TWA (respirable dust)		2 mg/m³ TWA.
1395 Tin oxide	21651-19-4		2 mg/m³ TWA		2 mg/m³ TWA.
1409 Trimellitic anhydride	552-30-7		0.005 ppm TWA		0.005 ppm TWA.
1430A Wood dust, hard	None		1 mg/m³ TWA		5 mg/m³ TWA, 10 mg/m³ STEL.
1403B Wood dust, soft	None		5 mg/m³ TWA, 10 mg/m³ STEL		5 mg/m³ TWA, 10 mg/m³ STEL.
1430C Wood dust (Western Red Cedar).	None				2.5 mg/m³ TWA.
2169 Yttrium	7440-65-5	1 mg/m³ TWA	1 mg/m³ TWA, 3 mg/m³ STEL		1 mg/m³ TWA.

* OSHA's PELs do not currently apply in agriculture; OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TLV®-TWA is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times in any working day, with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10 hour/day, 40 hour/week exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

+ NIOSH considers this substance a potential occupational carcinogen.

Description of the Health Effects. The respiratory system is a major route of occupational exposure for toxic substances. Because of the vital nature of pulmonary function, respiratory toxicants present a serious health hazard both from acute and chronic exposures. Acute respiratory disease can be life threatening.

Chronic pulmonary disease can result from long-term exposure to respiratory toxicants and is potentially crippling because it greatly reduces the quality of life and the productivity of its victims. In addition, the onset of respiratory disease can be insidious, because it may be indicated only by the gradual development of a few nonspecific signs (Petersdorf et al. 1983).

The difficulties of detecting irreversible respiratory effects complicate the prevention of pulmonary disease. Pulmonary function can be evaluated with a variety of tests, including measurements of the vital capacity and of resting and forced expiratory volumes. However, certain conditions, including emphysema and fibrosis, are difficult to diagnose even with such tests. In addition, these same diseases often continue to progress even after the affected individual has recognized the problem and obtained medical assistance; also, respiratory effects may worsen even after exposure has ceased, which makes prevention even more vital. In addition to the threat posed to the general occupational population by respiratory toxins, certain subpopulations, such as persons with impaired lung function caused by asthma, bronchitis, emphysema, or pulmonary fibrosis, are at special risk from the adverse effects of exposure to these substances. Tobacco smoking can cause or aggravate all of the respiratory conditions discussed above and can interact additively or synergistically with respiratory toxins to increase their adverse effects on the pulmonary system. For example, tobacco smoking acts additively with coal dust to diminish pulmonary function. Because

tobacco smoke contains nitrogen oxides, cadmium, and ammonia, occupationally exposed workers who smoke have an additional source of exposure to these respiratory toxins.

Two general categories of lung injuries are relevant to the group of substances under consideration:

- Damage to cells lining the airways, which results in necrosis (localized areas of dead cells), increased permeability, and edema.
- Production of fibrosis, which may become massive and greatly reduce lung capacity.

Cellular damage resulting in edema and emphysema. A number of substances cause damage to cells lining the airways. This can result in increased permeability of cell membranes and subsequent edema, hemorrhage, and localized necrosis (areas of dead cells). Chronic inhalation of certain chemicals causes destruction of the alveolar septa and results in emphysema. Cellular damage may be either localized or diffuse, depending on the distribution of the toxicant in the lung.

Edema is the release of fluid into the lumen (open spaces of the airways) or alveoli. Serious edema can take several hours to develop so that, in some cases, life-threatening or even fatal exposures can take place without the individual's being aware at the time of exposure of the extent of the damage. Ozone, nitrogen dioxide, and paraquat all cause localized cellular damage leading to edema (Klaassen, Amdur, Doull et al. 1986/Ex. 1–99).

Fatalities from pulmonary edema have resulted from exposures to concentrations of nitrogen dioxide of about 200 ppm (Sax 1984). Paraquat is unusual in that it can cause delayed pulmonary damage following exposure, even when exposure occurs via routes other than inhalation (Klaassen, Amdur, Doull et al. 1986/Ex. 1–99).

Necrotic changes can reduce the functional surface area of the lung. One type of lesion often noted in persons exposed to respiratory toxins is benign granulomas, which are localized masses formed when the immune system attempts to sequester a foreign object. Depending on the extent of the damage, these masses may reduce the functional capacity of the lung. Exposure to selenium-doped bismuth telluride has been associated with the production of benign granulomas without fibrosis (Wagner, Madden, Zimber, and Stokinger 1974, as cited in ACGIH 1986/ Ex. 1–3, p. 59).

Emphysema is caused by a gradual destruction of the cells of the alveolar septa, which causes a loss of elasticity in the lung. A slight degree of emphysema is present in much of the adult population and does not cause any functional impairment. As the disease progresses, however, serious and life-threatening reductions in functional capacity can occur. Once the disease has advanced to the point of serious functional impairment, it is, for the most part, irreversible (Petersdorf et al. 1983). There is evidence that a number of the substances in this group cause emphysema, including sulfur tetrafluoride (ACGIH 1986/ Ex. 1–3), ozone, and nitrogen dioxide (Klaassen, Amdur, Doull et al. 1986/Ex. 1–99).

Fibrotic changes. Pulmonary fibrosis was one of the earliest recognized forms of occupational disease. Fibrosis should be distinguished from pneumoconiosis, although these terms are often used interchangeably. Pneumoconiosis is a more general term indicating the presence of a foreign substance in the lungs, as determined by radiographic (X-ray) analysis. This definition encompasses a variety of conditions and does not by itself necessarily indicate functional damage (Petersdorf et al. 1983). In contrast, fibrosis is a seriously debilitating disease. One type of fibrosis is interstitial fibrosis, which is a kind of pneumoconiosis characterized by deposition of fibrous tissue in the interstitial spaces between the alveolar membrane and the pulmonary capillary membrane. Interstitial fibrosis greatly reduces the diffusing capacity of the lung and thus causes oxygen deprivation in the body (Guyton 1981/Ex. 1–1002). Like emphysema, fibrosis is largely irreversible; it sometimes progresses even in the absence of further exposure (Petersdorf et al. 1983).

Silicosis is a form of interstitial fibrosis that is caused by exposure to respirable silica particles (Klaassen, Amdur, Doull et al. 1986/Ex. 1–99). Exposure to coal dust causes a pneumoconiosis with fibrosis that can be severely debilitating (Petersdorf et al. 1983). In addition, exposure to graphite, mica, and grain dust have all been associated with fibrosis in workers (ACGIH 1986/Ex. 1–3).

Dose-Response Relationships and Respiratory Effects. For most of the substances in this group, the proposed permissible exposure limits have been based on health surveys and case reports of occupationally exposed populations. In some cases, animal studies have provided the evidence of a substance's toxicity. As is the case for most of the substances for which OSHA is proposing new, reduced, or revised limits, the dose-response curve for respiratory irritants tends to be S-shaped. Table C6–2 presents dose-response data on the adverse pulmonary effects of representative chemicals in this group, the populations exposed, and the endpoints observed. The substances on Table C6–1, and describe the nature of the risks faced by worers exposed to them in construction, maritime, and agricultural workplaces.

TABLE C6–2.—SUMMARY OF DOSE-RESPONSE EVIDENCE FOR ADVERSE RESPIRATORY EFFECTS CAUSED BY SELECTED RESPIRATORY TOXINS

H.S. No./chemical name	CAS No.	Current PEL in construction and maritime	Dose-response data			
			Proposed PEL in construction, maritime, and agriculture	Dose/duration associated with observed effect	Species	Comments
1034 Bismuth telluride (Se-Doped).	1304–82–1		5 mg/m³ TWA	15 mg/m³ 1 year	Dogs, Rats, Rabbits	Granulomatous lesions in lungs seen after 6 months of exposure.
1096 Coal Dust, < 5% quartz.	None	2.4 mg/m³ TWA	2 mg/m³ TWA	4 mg/m³ 35 years	Humans	Calculated estimate of 10 percent probability of developing pneumoconiosis with fibrosis after 35 years of exposure to coal dust.
1097 Coal Dust, > 5% quartz.	None	10 mg/m³ % SiO_2+2	0.1 mg/m³ TWA			(Quartz content not identified.)
1190 Grain Dust (oat, wheat, barley) symptoms..	None		10 mg/m³ TWA	> 10 mg/m³	Humans	Chronic bronchitis, shortness of breath, reduced pulmonary function, increased incidence of respiratory symptoms.

TABLE C6-2.—SUMMARY OF DOSE-RESPONSE EVIDENCE FOR ADVERSE RESPIRATORY EFFECTS CAUSED BY SELECTED RESPIRATORY TOXINS—Continued

H.S. No./chemical name	CAS No.	Current PEL in construction and maritime	Dose-response data			
			Proposed PEL in construction, maritime, and agriculture	Dose/duration associated with observed effect	Species	Comments
				< 10 mg/m³	Humans	Increased incidence of respiratory symptoms.
					Humans	Fibrosis and mottling; pneumoconiosis.
1191 Graphite, natural, respirable.	7782-42-5	15 mppcf TWA	2.5 mg/m³ TWA	N/A	Humans	Anthracosilicosis, similar to that seen in coal miners.
1213 Indium & compounds	7440-74-6	0.1 mg/m³ TWA	0.1 mg/m³ TWA	24-97 mg/m³	Rats	Widespread alveolar edema following exposure to In_2O_3.
1276 Mica	12001-26-2	20 mppcf TWA	3 mg/m³ TWA	N/A	Humans	Signs and symptoms resembling silicosis and pneumoconiosis in 8 of 57 workers.
1289 Nitrogen dioxide	10102-44-0	5 ppm Ceiling	1 ppm STEL	N/A	Humans	Fatal pulmonary edema.
				0.4-2.7 ppm chronic	Humans	Change in pulmonary vital capacity.
1300 Oxygen difluoride	7783-41-7	0.05 ppm TWA	0.05 ppm Ceiling	0.5 ppm two 7-hr exposures	Laboratory Animals	Lethal to a wide variety of laboratory species, causing pulmonary edema and hemorrhage after several hours of exposure.
1301 Ozone	10028-15-6	0.1 ppm TWA	0.1 ppm TWA 0.3 ppm STEL	1.5 ppm 3 hrs/day	Humans	Significant reduction in pulmonary vital capacity.
				1 ppm 1 day	Mice	Damage to alveolar tissue.
1303 Paraquat, respirable dust.	4685-14-7	0.5 mg/m³ TWA, Skin	0.1 mg/m³ TWA Skin	N/A	Humans	69 accidental deaths from pulmonary injury reported through 1972.
1354 Silica, crystalline cristobalite.	14464-46-1	½ value for quartz	0.05 mg/m³ TWA	0.5 mg/m³ (as total dust) 2.5 years	Dogs	Cellular infiltration of lung and fibrotic nodules in pulmonary lymph nodes.
1355 Silica, crystalline quartz, respirable.	14808-60-7	10 mg/m³ % SiO_2+2	0.1 mg/m³ TWA	0.1 mg/m³-chronic	Humans	Accelerated loss of pulmonary function beyond effects of aging alone.
1356 Silica, crystalline tridymite.	15468-32-3	½ value for quartz	0.05 mg/m³ TWA	N/A	Rats	Most active form of free silica when administered by intratracheal injection.
1357 Silica, crystalline tripoli.	1317-95-9	10 mg/m³ % SiO_2+2	0.1 mg/m³-TWA	N/A	Lab. Animals	Progressive nodular fibrosis.
1375 Sulfur dioxide	7446-09-5	5 ppm TWA	2 ppm TWA 5 ppm STEL	1 ppm	Humans	Accelerated loss of pulmonary function.
1378 Sulfur tetrafluoride	7783-60-0		0.1 ppm Ceiling	4 ppm	Rats 4 hrs/day/ 10 days	Emphysema, marked clinical signs of respiratory impairment.
1409 Trimellitic anhydride	552-30-7		0.005 ppm TWA		Rats	Intra-alveolar hemorrhage. (No exposure duration indicated.)

* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.
N/A = Not available.

ALUMINUM (PYRO POWDERS)
CAS: 7429-90-5; Chemical Formula: Al
H.S. No. 1017

OSHA has no permissible exposure limit for aluminum pyro powders in the construction, maritime, and agriculture industries. The ACGIH has an 8-hour TLV®-TWA of 5 mg/m³; NIOSH has no REL for these powders. The Agency is proposing an 8-hour TWA PEL of 5 mg/m³ for aluminum pyro powders in the construction, maritime, and agriculture industries; NIOSH (Ex. 8-47, Table N1) concurred with this limit when the Agency recently established it in general industry. Promulgation of this limit will make OSHA's PEL for these substances consistent across all OSHA-regulated sectors.

Aluminum in its elemental form is a white, malleable, ductile metal. Powder and flake aluminum are flammable and can form explosive mixtures in air. Aluminum pyro powders are used in fireworks and aluminum paint (Proctor, Hughes, and Fischman 1988, p. 66).

In addition to respiratory effects, aluminum pyro powders can cause gastrointestinal effects and eye irritation. Exposure to aluminum compounds can also affect the absorption of other substances such as calcium and iron compounds (Spencer et al. 1969, 1977). Particles of aluminum can cause necrosis of the cornea when deposited in the eye; in both rabbit and human eyes, aluminum particles have been shown to cause some local necrotic imprints in the fundus at the site of the absorbed particles (Fontana 1938; Jess 1924; Sanin 1947; Knave 1969). Aluminum pyro powders are more acutely toxic than the metal dusts of aluminum (Stokinger 1981a/Ex. 1-1133). Several British studies have examined the effects of exposure to this finely flaked aluminum on workers employed in paint and pyrotechnics plants. Their findings revealed that pulmonary fibrosis may result from exposure to pyro powders, although epidemiologic evidence indicates that additives used to prevent oxidation and agglomeration may have contributed to the incidence and nature of the disease (Edling 1961/Ex. 1-733; Jordan 1961/Ex. 1-559; Mitchell, Manning, Molyneux, and Lane 1961/Ex. 1-564).

OSHA believes that an 8-hour TWA permissible exposure limit of 5 mg/m³ for aluminum pyro powders will prevent the significant risk of lung changes in workers exposed to such powders in construction, maritime, or agricultural operations. In the recent general-industry rulemaking, the Agency determined that these lung changes constitute material impairments of health. Accordingly, OSHA believes that the proposed PEL is necessary to reduce the risk of these health impairments in workers in these sectors. In addition, promulgation of this limit will make OSHA's PEL for aluminum pyro powders consistent across all regulated sectors.

ASPHALT FUMES
CAS: 8052-42-4; Chemical formula: None

H.S. No. 1028

There is no current OSHA PEL for asphalt fumes. In the Air Contaminants rulemaking, a PEL of 5 mg/m³ (8 hour TWA) for asphalt fumes measured as total particulate was proposed. Many comments were received by the Agency and the complexity of the issues at that time caused OSHA to delay rulemaking on this substance. The AFL/CIO, in the case of AFL/CIO vs. OSHA #89–7185 (11th Cir.) challenged OSHA's failure to issue an asphalt standard. OSHA stated to the Court that it intended to set an exposure limit for asphalt fumes for all sectors as part of the construction rulemaking.

In this rulemaking OSHA is also proposing a PEL of 5 mg/m³ total particulate for general industry, maritime, construction and agriculture. However, subsequent to the 1988 proposal, an extensive body of new scientific data has become available. This data and criticisms of it are presented and extensively analyzed below. Consequently, commenters should respond to the various studies and the regulatory alternatives which would be appropriate based on final conclusions as to these studies. The economic aspects of the various alternatives are presented in the Regulatory Impact Analysis.

Asphalt, also called bitumen, is a mixture of hydrocarbons that is produced by the evaporation of the lighter hydrocarbons from petroleum distillation and subsequent partial oxidation of the residue. Occupational exposure to asphalt fumes can occur during its manufacture or as a result of the secondary heating of asphalt in processes such as road building and repair, roofing and the coating of construction materials (Thayer et al, 1981).

OSHA is addressing the risk to those who are exposed to asphalt fumes in the course of their employment (e.g., roofers and pavers). OSHA has not studied the issue and does not have current information that exposures to the general population are sufficiently high to warrant concern. It is the fume released from the asphalt which is of concern, and it is the physical proximity of the workers to these fumes that makes occupational exposure hazardous. OSHA has requested submission of any additional exposure monitoring data including environmental data for airborne asphalt fume levels that occur some distance from the worksite. Further, OSHA is maintaining dialogue with the Environmental Protection Agency on this issue.

Among the issues raised in the earlier rulemaking regarding asphalt fumes were:

1. Whether asphalt fumes are similar to coal tar fumes;
2. Which is the appropriate method of measurement of asphalt fumes, the benzene-soluble fraction or total particulate;
3. Irritant and other respirator effects of asphalt fumes;
4. Whether asphalt fumes are carcinogenic;
5. What is the proper PEL for asphalt fumes; and,
6. Whether the PEL determined on the basis of the health data is technologically and economically feasible.

OSHA requests comment on all these issues and all other issues which may be relevant to occupational regulation of asphalt fume. In the following discussion, OSHA discusses the studies and presents the views of various organizations. Final decisions will be reached only after consideration of all comments.

On the first issue, OSHA's preliminary view is that there are significant differences between petroleum-derived asphalt fumes and coal tar pitch volatile; in 1983, the Agency published a **Federal Register** notice making clear that the term "coal tar pitch volatile" (CTPV) and OSHA's CTPV standard do not apply to petroleum asphalt. It should be noted here that in the 1983 rulemaking in which asphalt fume was specifically excluded from the standard for coal tar pitch volatile, 48 FR 2768 January 21, 1983, NIOSH supported the continued inclusion of asphalt in the regulation of CTPV, noting the differences between the two substances but recognizing the need for worker protection against high concentrations of PAHs. OSHA notes that coal tar pitch volatile is more irritating than asphalt fume, however the two substances look the same, have the same consistency, contain some common components such as polycyclic aromatic hydrocarbons (PAHs), and have often been used in the same applications. OSHA requests comment on all aspects of this issue.

As for the second point, OSHA's proposed PEL is measured as "total particulate." Another view is that the benzene-soluble fraction is the more specific and thus more appropriate means of measuring asphalt fume and includes this method of determining exposure in the current proposed PEL. The Asphalt Institute (AI) has pointed to several studies which indicate anomalies in measurements using that technique. It has been suggested that the anomalies are the result of the use of plastic filters and that use of glass fiber filters will solve this problem. OSHA requests comment on all aspects this issue.

It should be noted that there are a number of different types or fractions of asphalt. Some commenters have suggested that the type or fraction may be relevant in analyzing these studies.

Respiratory Effects. A very recent study (Norseth, Waage and Dale, 1991, Am J Ind Med, Vol. 20, No. 6, Acute Effects and Exposure to Organic Compounds in Road Maintenance Workers Exposed to Asphalt) attempted to quantify the levels at which a variety of types of irritant and other acute effects were reported as a result of exposure to asphalt fumes. The authors attempt to correlate the reporting of acute symptoms and exposure to organic compounds in a cohort of road maintenance workers. Subjective symptoms and exposure to organic compounds and asphalt fumes were recorded for 254 workers using warm asphalt for road repair and construction, and for a comparison group of 247 maintenance workers who did not work with asphalt. Mean exposure to asphalt fume was 0.358 mg/m³ (carbon disulfide-soluble fraction). No correlation between symptoms and total amount of volatile compounds was found; however, a significant positive correlation was demonstrated with both increasing concentration of asphalt fumes and increasing asphalt temperature. Abnormal fatigue, reduced appetite, laryngeal/ pharyngeal irritation and cough, and eye irritation were found more often in asphalt workers than in the reference group without asphalt exposure. No differences were found for headache, dizziness, nausea, abdominal pain, disturbed sleep, skin reactions, or a smell of sweetness. The authors conclude that asphalt temperature should be kept below 150 degrees C (302 degrees F), fume concentration below 0.4 mg/m³, and the use of harder asphalt types which also require higher temperatures should be avoided.

As this study is very recent it has not yet been subjected to extensive review and comment. But on its face, an argument could be made that an exposure limit of lower than 0.5 mg/m³ may be necessary to prevent acute health effects, OSHA requests comments on all aspects of the study and the appropriate regulatory response.

A few older reports of respiratory effects of asphalt fumes exist in the literature, although information on exposure levels and the possibility of

concomitant exposure with coal tar pitch volatiles is sketchy. Zeglio (1950, as cited in NIOSH, Criteria for a Recommended Standard * * * Occupational Exposure to Asphalt Fumes, 1977) published observations on 22 workers who insulated electrical cables and telegraph and telephone lines. The workers were exposed to fumes from tanks containing bitumen heated to 120 degrees (presumed to be Celsius). Although the concentration of the fumes was not measured, Zeglio stated that the atmosphere had an acrid odor that was irritating to the nose and throat and that stimulated coughing.

Workers exposed in the plant complained of coughing with expectoration, a burning sensation in the throat and chest, and frequent hoarseness. Headache and nasal mucous discharge were also frequently mentioned by the workers. Physical examination of these 22 workers revealed 10 cases of rhinitis, 13 cases of otopharyngitis, 4 cases of laryngitis, and 19 cases of bronchitis. Roentgenographic examination revealed an increased vascularization in the larger bronchial areas, particularly evident in the mediobasal zones in all the workers with bronchitis.

Baylor and Weaver (1968) conducted a health survey of asphalt workers from 7 asphalt companies (462 workers) and a control group from 25 oil refineries (379 workers). Physical examinations emphasized the skin and respiratory tract and included chest roentgenograms. Prevalence of respiratory disease other than cancer were reported in 31 of 360 asphalt workers (8.6%) and in 24 of 379 controls (6.3%). Comparison of these rates with National Center for Health Statistics data yields relative risks which are not significantly different. Baylor and Weaver concluded that "* * * petroleum asphalt cannot rationally be considered a hazardous substance." They did, however, point out that individual predisposition might cause some workers to be subject to health problems with contact with asphalt.

There are a number of studies which consider the issue of the carcinogenicity of asphalt in animals and which consider the prevalence of cancer or the cancer mortality among road paving workers. The following discusses those studies.

Carcinogenicity Data: Animal Studies. The following discusses the toxicological literature, both negative and positive studies, which consider the carcinogenic potential of asphalt fume in animals as evidenced by its ability to induce tumors in mice in skin painting assays. These studies include the following: Hueper and Payne (1960); Simmers (1965, 1966); Wallcove et al, (1972); Bingham et al (1980); Thayer et al, (1981); Emmett et al, (1981); Malaiyandi et al, (1982); IARC (1985); Belinky et al, (1986); Niemeier et al (1989); and Sivak et al, (1989).

Several studies have reported findings on the carcinogenicity of asphalt by skin painting. A number of studies have sought to correlate the ability of a complex mixture to produce skin cancer in rodents with its ability to initiate lung tumors in humans. For example, Nesnow (1989) found very high correlation between the human lung cancer risk estimates and mouse skin tumor initiation activity of three known carcinogens: coke oven emission, roofing tar emission, and cigarette smoke condensate.

In a preliminary study sponsored by NIOSH, Thayer et al (1981) reported positive tumorigenic activity when condensate from two different types of roofing asphalt volatile and condensate from two types of coal tar pitch fumes generated at both 232 °C and 316 °C were tested for tumorigenicity. In this study, groups of CD–1 and C3H/HeJ mice were treated with one of the four types of asphalt condensates or one of the four types of coal tar pitch condensates. Results indicated that both mouse strains developed skin tumors (papillomas and carcinomas) with the CD–1 mouse being less sensitive to the tumorigenic activity of asphalt fumes than the C3H/HeJ strain. When comparing the tumorigenic response to fumes generated at the two temperatures, the asphalt volatile generated at the higher temperature produced a greater tumorigenic response. Although Thayer et al. (1981) reported a statistically significant increase in the incidence of skin tumors, it is difficult to estimate quantitatively the cancer risk from these data because of the lower reliability of the estimate of q_1 (the slope of the dose-response curve) determined from data providing one dose level and a 100% tumorigenic response.

In a paper more fully describing the results of the NIOSH-sponsored study, Niemeier et al. (1988) noted that the carcinogenicity of the asphalt volatile observed in the mouse skin painting experiments previously mentioned in Thayer et al. (1981), could not be entirely accounted for by the concentration of known carcinogens, such as polycyclic aromatic hydrocarbons (PAH) or benzo(a)pyrene (BaP), which is the case for CTPV. Approximately ten-fold more asphalt fume was required to obtain the same biological effect as CTPV. The amounts of total PAH or benzo(a)pyrene (BaP) found in asphalt fume are 30- to 40-fold less than those amounts found to be effective in eliciting the same response with condensed coal tar pitch fumes. This observation led Niemeier et al. (1988) to hypothesize that the asphalt preparation contained other components which might augment the activity of the very low concentrations of measured PAH found in the asphalt study.

In an attempt to further characterize the components of the asphalt fumes responsible for the carcinogenic activity, Sivak et al. (1989) heated roofing asphalt to 316° C. The fumes generated were fractionated by preparative high performance liquid chromatography (HPLC) and analyzed by gas chromatography/mass spectroscopy (GC/MS). The five fractions collected (labeled A through E) were used in skin application bioassay in male C3H/HeJ and Sencar mice (30 animals per test group). Fractions were applied in proportion to their amount in neat asphalt fume.

Fraction C which contained alkylated phenylethanones and alkylated dihydrofuranones had the greatest tumor incidence. Fraction C produced tumors in 17 of 30 C3H mice while fraction B which contained olefins, alkylated aryl thiophenes and alkylated phenanthrenes produced tumors in 10 of 30 C3H mice. The other fractions exhibited no tumorigenic activity. Neither synergism, co-carcinogenicity, nor tumor-promoting activities was observed among the fractions at the concentrations tested.

In a 1988 submission to the Air Contaminants rulemaking docket H020, Exxon reviewed the literature and concluded: "To date, skin painting studies in mice have proven to be the most reliable predictors of the potential carcinogenicity of petroleum-derived materials since these tests reflect the integrated biological response to the presence of tumor initiators, promoters and co-carcinogens" (Exhibit 3–681).

The position of the Asphalt Institute, as stated in their "Report to OSHA and NIOSH, Status of Asphalt Industry Steering Committee Research Program On the Health Effects of Asphalt Fumes and Recommendation for a Worker Health Standard" (1990) is as follows: "The bioassay studies do confirm that some asphalts contain small amounts of carcinogenic species which, if volatilized or otherwise made bioavailable to test animals, may cause skin cancers. But the bioassays do not identify the active species. Nor do they

demonstrate that, under conditions of real world use of all, most, or even any asphalt products, those known constituents are present in fumes in sufficient quantities to yield tumorigenicity in a skin-painting bioassay".

Short-term Tests

The fractions generated in the NIOSH study were used in a modified Ames test of mutagenicity (in which the ability of a test substance to induce revertant in a gene of the TA98 strain of *Salmonella typhymurium* is determined). This measure of mutagenicity correlated well with the observed mouse skin carcinogenicity (r = 0.94). (Asphalt Institute submission.)

In a recent study, NIOSH reported that fractionated asphalt fume condensate inhibited inter-cellular communication. Most inhibitors of intracellular communication have been observed to have tumor promoting activity. It is hypothesized that the promoting agent serves to isolate an initiated cell from the growth-regulating signals (communications) of surrounding cells leading to the development of tumors.

In this study NIOSH researchers tested the same fume fractions used in the skin painting study (Sivak) in a metabolic cooperation assay. When wild type Chinese hamster lung fibroblasts are grown in the presence of 6-thioguanine (6-TG), they are killed by its phosphorylated metabolite. Resistant cells do not produce the toxic metabolite and therefore survive exposure to 6-TG. However, in a mixed cell culture containing both sensitive and resistant cell type, there is transfer of the 6-TG metabolite via gap functions from the sensitive to the resistant cells and both types are killed. In the V79 metabolic cooperation assay, co-cultivated 6-TG sensitive and 6-TG resistant cells were exposed to condensate of asphalt fume fractions prior to the addition of 6-TG to the medium. If the test substance inhibited intracellular communication, only those resistant cells which did not receive the toxic 6-TG metabolite from the sensitive cells via gap junctions would survive. The parent mixture of asphalt fume condensate fractions and all of the fractions, except fraction A, inhibited intercellular communication in a concentration dependent fashion (dose-response). Fraction A inhibited metabolic cooperation only at the maximum concentration tested. Fractions D and E, which contained alkylated ketones, alkylated napthols and phenols, were most potent fractions in this assay. The authors hypothesized that "If the similarity between cigarette smoke and asphalt fume condensates goes beyond inhibition of metabolic cooperation in V79 cells, the carcinogenicity of the asphalt fume condensate may be due in part to the presence of tumor promoters acting through inhibition of intracellular communication."

In a study by Monarca et al. (1987), extracts of bitumen fume demonstrated no mutagenic activity in the Ames test, although the benzo(a)pyrene [B(a)P] concentration predicted that the samples would be positive. This observation led these workers to hypothesize that there was inhibition of mutagenicity due to other sample components. This research group further reported results of biological monitoring of the workers exposed to the asphalt fumes analyzed in their earlier work (Pasquini et al., 1989). The worker exposures were between 0.6 and 0.8 mg/m^3 (total particulate) and were approximately 0.2 mg/m^3 measured as benzene soluble fraction. Urine samples from asphalt fume-exposed and non-exposed workers were collected and tested for mutagenicity in the Ames assay.

Microsomal enzyme activation was required for all observed mutagenic activity and was detected only with the TA 98 bacterial strain. Neither of the two additional potential indicators measured, thioether and D-glucaric acid, was significantly different in the exposed and non-exposed groups.

Nine of 11 (82%) non-smokers exposed to asphalt fume had mutagenic urine, while 5 of 18 (28%) of asphalt non-exposed, non-smokers were positive. The urine of all smokers, whether or not exposed to asphalt fume, was positive.

These researchers noted an apparent inconsistency in that environmental samples of asphalt fume were non-mutagenic in their assay, while many of those exposed to asphalt produced mutagenic urine. They offered several explanations: Escape of active components from the fume samples, difficulties inherent to Ames testing of complex mixtures, and, as mentioned earlier, the potential for inhibition of mutagenic activity by other components. They further stated:

> * * * urinary mutagenicity is considered more meaningful for evaluating human exposure to unknown complex mixtures than ambient monitoring, because it is a nonspecific indicator and can estimate the uptake of the mutagens in the body.

In other words, some metabolism that takes place in the body and converts the exposure to a mutagen is not duplicated in bacterial systems used for the Ames test. Therefore, a mutagenic effect will not be observed in the latter test system, particularly if the toxic chemical requires a multi-step metabolic process for it to become an active mutagen.

Several classes of carcinogens, including polycyclic aromatic hydrocarbons (PAH), are hypothesized to mediate their activity through their metabolic activation to reactive species that subsequently covalently bind to DNA of the cells of the target organ. Shocket et al. (1988a) studied the covalent binding of aromatic compounds to DNA in mouse skin following topical application of coal-tar, creosote or bitumen. The level of adducts (covalently linked species) observed in mouse skin 24 hours after treatment are as follows:

Agent	Dose/mouse (skin)	fmol adduct/μg DNA
Coal tar	30 mg	0.38
Coal tar	6 mg	0.14
Creosote	25 μl	0.40
Creosote	5 μl	0.19
Bitumen	15 mg	0.09
Bitumen	3 mg	0.00

Adducts were also detected in lung DNA of treated animals. As with coal-tar and creosote, higher levels of accumulation of DNA adducts were observed in skin than in lung following multiple applications.

Schocket et al. (1988b) also looked at the formation of DNA-adducts in human fetal and adult skin cell cultures following treatment with coal-tar, creosote, and bitumen (as a paint formulation containing tetrahydrofuran). These results are as follows:

Treatment	fmol adduct/μg DNA	
	Human adult skin	Human fetal skin
Control	0.097	0.063
Coal tar (30 μg)	0.354	0.355
Creosote (25 μl)	0.313	0.197
Bitumen (3 μg)	0.000	0.156
Bitumen (15 μg)	0.216	0.185

All fetal adduct levels, including those of the bitumen treated, contained statistically significant amounts of adducts. The adult cell cultures treated with 3 μg of bitumen paint solutions failed to produce any adducts. This leads to the point presented by the authors of the possible implications of the ability of fetal tissue to activate the carcinogenic activity of PAHs *in utero*. These data indicate that bitumen is a less potent inducer of DNA adducts in mouse skin cells, (the site of induction in the *in vivo* skin painting studies) than it

is in the human skin cells, relative to coal tar and creosote. Another point that Schocket et al. (1988b) mentions is that bitumens have low levels of activity when compared to the coal-tar and creosote, but that there appears to be potential to produce malignant tumors. The authors concluded that bitumen was similar to coal-tar and creosote in forming DNA adducts in the target cells and recommended that "it would seem prudent to regard preparations of bitumen as representing a hazard to man comparable to that of the other two agents" (Schocket et al. 1988b).

The ACGIH TLV for asphalt fume is a TWA of 5 mg/m³ measured as total particulate to reduce the possible risk of carcinogenicity (ACGIH, 1986). However, ACGIH did not specifically designate asphalt fumes a carcinogen. In 1977, NIOSH recommended a 15-minute ceiling value of 5 mg/m³ measured as total particulate based upon the prevention of irritation to the eyes and respiratory tract of workers, and to minimize the possibility of a carcinogenic response from occupational exposure to asphalt fumes (NIOSH, 1977).

In a 1987 publication, IARC concluded that there is sufficient evidence for the carcinogenicity of extracts of steam-refined bitumens and air-refined bitumens in experimental animals; that there is limited evidence for the carcinogenicity of undiluted steam-refined bitumens and for cracking-residue bitumens in experimental animals; that there is inadequate evidence for the carcinogenicity of undiluted air-refined bitumens in experimental animals; and that there is inadequate evidence the bitumens alone are carcinogenic to humans. (IARC 1987) IARC's assessment of asphalt, however, was made prior to the publication of the above mentioned epidemiologic and DNA-adduct studies.

In its Report to OSHA and NIOSH (1990), the AI states: "Quite apart from the unresolved questions about the representativeness of the fume condensates tested by NIOSH, asphalt fumes have been shown to be tumorigenic only in skin-painting studies. Inhalation bioassays have not shown similar effects. By their very nature, skin-painting studies do not generate the dose-response data needed to estimate carcinogenic potency. And still further biological uncertainties are introduced by extrapolating the results of dermal exposures in mice to inhalation exposures in workers". The AI concludes: "The data support a PEL of 5 mg/m³ for asphalt fumes to protect workers against eye and respiratory tract irritation, but do not support a finding of significant cancer risk".

Epidemiologic Studies

In a recent epidemiologic study of asphalt exposed workers, Hansen (1989) compared the mortality experience between a cohort of 679 mastic asphalt workers with that of all Danish men. These workers' jobs require them to fill buckets with asphalt (at approximately 250 °C), carry the bucket to the area of application where they tip out the asphalt and level it by drawing out the mixture with long wooden trowels. Much of this work is done by the worker while kneeling. The process is used both in flooring and paving operations with the flooring operations having the higher exposure levels.

The 679 male mastic asphalt workers were identified from several sources as having performed mastic asphalt work between 1959 and 1980. Subjects were followed through January 1985. Information on death and emigration was obtained through the Danish National Register. By the end of follow-up, 149 had died, 6 emigrated resulting in a total of 6,692 person-years of follow-up. Incident cancer cases were ascertained through the Danish Cancer Registry. Data on individual exposure history was generally not available. Age grouping was taken as proxy for time from first exposure, allowing an estimation of latency.

The SMR for all malignant neoplasms was 195 (CI, 153–244). Other cancer sites having significantly elevated mortality rates are as follows:

Site	SMR	95% confidence interval
Mouth	1,111	(135–4,014)
Esophagus	556	(144–2,039)
Rectum	318	(128–656)
Lung	344	(227–501)

Two samples taken to measure exposure levels to asphalt fume during road paving, in which approximately $^2/_3$ of the cohort was engaged, were 4.3 and 3.4 mg/m³ with a mean of 4 mg/m³. Thirty five samples taken during flooring operations, in which the remaining $^1/_3$ of the cohort worked, had an average exposure of 20 mg/m³. These estimates of exposure are based on 1976 measurements by the Danish National Institute of Health, and the author of this study has informed OSHA that it is likely an overestimate of exposure since the amount of mastic applied per worker increased during the study period, prior to the 1976 sampling. Dr. Hansen further informed OSHA that the workers receive approximately 90% compensation during the period of the year in which they cannot perform mastic asphalt work. After adjustment for the seasonal nature of the work, Hansen estimated that the average exposure of mastic workers in the cohort was equivalent to a continuous exposure of 5 mg/m³, the current Danish PEL.

Because coal tar pitch was added to the mastic asphalt mixture during World War II due to shortages of asphalt, it was necessary to determine the number of workers who were potentially exposed to both asphalt fumes and coal-tar. In order to do this, the cohort was further divided into three subcohorts. The three subcohorts divided the workers according to the time of birth as follows: Group I (N=194), born 1893–1919; Group II (N=129), born 1920–1929; and, Group III (N=356), born 1930–1960. Group I was the most likely to have exposure to coal-tar during World War II; Group II may have had some coal-tar exposure in World War II, and Group III could not have had wartime exposure. The lung cancer SMRs for all three groups (302, 392, and 857 respectively) were significantly elevated, further supporting the role of asphalt in this tumor excess. The results also suggested that the increased lung cancer risk manifested itself earlier (at younger ages) in the subsequent birth cohorts, but numbers of cancers in this youngest group were too small to reach statistical significance. Hansen also adjusted the respiratory and digestive cancer SMRs for estimated confounding effects of urbanization and smoking habit. This adjustment resulted in somewhat lower SMRs of 214 and 206 for respiratory and digestive cancers respectively, but still demonstrated statistically significant excess risk.

In this incidence study, urinary system cancer, which includes bladder cancer, known to be smoking-related, showed no statistically significant increase (SMR=133). In a subsequent mortality study of this cohort, Hansen (1990) reported that an SMR of 100 for cardiovascular disease deaths-a cause of death also known to be associated with smoking. Thus, two major causes of death related to cigarette smoking were not elevated among the cohort members. These observations suggest that cigarette smoking is unlikely to have accounted for the excess lung cancer observed in the study.

In a subsequent mortality study of this cohort, Hansen (1990) reported that a 1976 survey of mastic workers (110 members of the study cohort) indicated that 22% were non-smokers, 36%

consumed less than 14 grams of tobacco per day, and 43% consumed 15 or more grams per day, while a 1982 Danish population-based survey gave these percentages as 39, 24 and 30% respectively. She used the lung cancer death rates among non-smokers, medium smokers, and heavy smokers from the Doll and Peto study (1976) to estimate that these differences in smoking would bring about an 18% excess of lung cancer deaths and 21% excess of the bronchitis deaths in her cohort of workers. This adjustment of the SMR for lung cancer lowered the estimate from 290 to 246.

Similarly, because most of the cohort was from urban areas of Denmark and the comparison population contained both urban and rural groups, adjustment for urbanization was also undertaken. The correction which was based on Danish census data, lowered the estimate of the SMR for lung cancer from 290 to 264. Independent adjustment for both, a procedures which over-adjusts because smoking and urbanization are related, still resulted in a statistically significant SMR of 224.

Hansen (1989a) also undertook a separate epidemiologic study comparing mortality between 1,320 workers employed in the Danish asphalt industry with that of 43,024 workers from service trades, agriculture, forestry and specific light industries. She found a significantly increased mortality for all malignant neoplasms among asphalt workers aged 45 or more when 5 years of latency was taken into account (SMR 159, 95% confidence interval, 106–228) compared with other unskilled workers. There were increases for respiratory, bladder and digestive cancers, but these increases were not statistically significant. No attempt was made to assess exposure levels—the only criteria for a subject to be in either exposure category were his occupation and being employed on the date of the census, which is likely to have diluted any differences. For instance, the comparison group might have included asphalt-exposed workers who were not employed in asphalt work on that date. Similarly, the "exposed" group might have included workers with low cumulative asphalt exposures.

Epidemiologic studies by Hammond et al, (1976) Menck and Henderson (1976), and Milham (1982) reported increased standardized mortality ratios or proportionate mortality ratios for lung cancer among roofers and slaters. It is difficult to establish a clear association between asphalt exposure and lung cancer in these studies because most workers had combined exposures to asphalt and coal tar, and smoking histories of the workers were not known.

A study by Baylor and Weaver (1968) reported no significant differences in a cross-sectional survey on the health of 462 asphalt workers compared with the health of 379 refinery workers. These workers had asphalt exposures for at least 5 years and were then given a physical examination, which included a medical and occupational history. No significant differences were noted between the 2 groups of workers regarding: respiratory diseases, skin cancer, non-carcinogenic skin disease, hypertension, ulcer, heart disease, or other cancers. This is not an appropriate study for demonstrating whether or not there is an excess risk of cancer for a number of reasons. Without mortality data and longer follow-up, few conclusions regarding the risk of asphalt exposure can be drawn from these studies. Since refinery workers are also exposed to PAHs, etc., they are therefore not a true control group for asphalt fume exposures.

Based on preliminary reports of excess leukemia among highway maintenance workers in a small Minnesota community, Bender et al (1989) assembled a cohort of Minnesota highway maintenance workers and determined their cause-specific mortality and compared this rate with that of the entire Minnesota population. This study had a large number of subjects (4,849) and deaths (1,530), and lengthy and nearly complete follow-up (99.6%). The authors did not, however, present any exposure information. The results indicated a statistically significant SMR of 425 for leukemia mortality among those with 30 to 39 years of work and, as mentioned earlier, a significant SMR of 292 for urologic cancer among those having 40 to 49 years of latency. Lung cancer SMRs and other cancer SMRs were not significantly elevated.

In its "Briefing Paper On the Regulation of Asphalt Fumes" the Asphalt Institute described this study by Bender as "(T)he best study of U.S. asphalt workers," and stated that it found no increased mortality at any relevant cancer site. In a letter to OSHA staff Dr. Parker, a secondary author of the Minnesota Highway Maintenance Worker Study, stated that this study "does not support a contention, either pro or con, with regard to the potential carcinogenicity of asphalts. Although the primary workers we evaluated performed highway maintenance work, these workers were not primarily involved in road construction" (where they would be most likely to be exposed to asphalt fumes).

Parker et al (1989) reported the results of a study on the same cohort in which non-cancer causes of death were analyzed. The SMR for renal failure among long-term rural workers was significantly elevated (SMR=676) as was the SMR for transportation-related injuries (SMR=280). The lack of information on the exposures of this cohort render it of little value in determining the level of risk from exposure to asphalt and does not allow the determination of risk levels among more highly exposed subgroups.

Quantitative Risk Assessment

The best available human data for quantifying the lung cancer risk associated with paving workers is found in the Hansen study of Danish mastic asphalt workers (Hansen 1989b). This study has the best exposure data along with estimates of relative risk of cancer in humans to evaluate a dose-response relationship. If some cohorts of workers in this study did not have substantial exposure to coal tar pitch volatiles, (CTPV) a known carcinogen, then this study provides quantitative evidence of cancer risk from asphalt. The issue of CTPV exposure is controversial and discussed below.

Hansen derived the mean exposure level of asphalt fume in her study cohort, 5.4 mg/m^3, as follows: two thirds of the cohort was engaged in road paving operations, having a mean exposure level of 4 mg/m^3; one third was involved in flooring operations, having a mean exposure to asphalt fume of 20 mg/m^3; and, that exposure occurred only during seven months of the year. This leads to:

$[(^1/_3 \times 20\ mg/m^3) + (^2/_3 \times 4\ mg/m^3)] \times {}^7/_{12} = 5.4\ mg/m^3$.

OSHA assumed that exposure began at 22.5 years of age and continued at a constant level for 45 years. These assumptions are reasonable because Hansen stated in the report of this study that new mastic workers are always young, typically in their early twenties, and that the turnover in this job is moderate. Dose was estimated in units of milligrams per cubic meter-years (mg/m^3-yr) and the expected cancer incidence was calculated from age-, period-, and site-specific incidence rates for Danish men during the period 1958–1982.

A relative risk model was used to quantify the risk from occupational exposure to asphalt fume. The relative risk model is based on the assumption that the increase in the relative risk of

lung cancer death due to asphalt fume exposure is the product of an individual's background risk at time t, $h_o(t)$, and the risk attributable to asphalt fume exposure, $h_e(t)$. The risk attributed to asphalt fume exposure is given by

$$h_e(t)=\beta X,$$

where X represents cumulative dose up to time t. An individual's total risk of lung cancer death at time t, h(t), is given by

$$h(t)=h_o(t)+(h_o(t)h_e(t))$$
$$=h_o(t)+(h_o(t)\beta X).$$

This model can also be written as a linear model where

$$\frac{h(t)}{h_o(t)}=1+\beta X.$$

Here, 1 is the intercept (i.e. the relative risk assuming no exposure), and β is the slope of the dose-response line representing the change in relative risk per unit dose.

If we consider each individual to be an independent Bernoulli trial and sum over all individuals in the jth exposure group, we obtain the expected number of lung cancer deaths for the period of observation, $E(O_j)$, which is given by

$$E(O_j)=E_j+(E_j\beta X_j),$$

where E_j is the expected number of cases assuming no asphalt exposure, (i.e. background), and X_j is the median dose for exposure group j. Assuming that the observed number of deaths is distributed as a Poisson random variable with expectation given above, we obtain the likelihood of observed results

$$LIK=\prod_{j=1}[\exp-(E_j+(E_j\beta X_j))]$$
$$[E_j+(E_j\beta X_j)]/O_j$$

The maximum likelihood estimate of the unknown parameter β is obtained by maximizing the log likelihood with respect to β. The variance of β is given by the inverse of the observed information. We solve for the parameter β using a Newton-Raphson algorithm. Estimates of β and its variance derived from the Hansen data using the relative risk model are given in Table 1.

The relative risk model rests on the assumption that the ratio of the risk of lung cancer death for asphalt-exposed individuals to the risk of lung cancer death for individuals with no asphalt exposure depends only on dose and is constant across age groups. In other words, for any given cumulative dose, the risk of death for a twenty-five year old exposed individual relative to a non-exposed twenty-five year old individual is the same as the risk of death for a fifty year old exposed individual relative to a non-exposed individual of the same age.

OSHA recognizes the importance of this assumption in estimating true occupational risk. Thus, OSHA specifically requests comments concerning the significance of assumptions and their applicability for estimating risks from exposure to inhaled asphalt fume.

After fitting the model to the data, it is reasonable to ask whether or not the fit is good. The standard approach for measuring goodness-of-fit is to perform a Chi-Square test, in this case calculating the deviation of the number of lung cancers predicted for each exposure group from the number of lung cancers observed in each group. The number of lung cancers predicted for the jth exposure group, O_j, is given by

$$O_j=E_j+(E_j\beta X_j),$$

where E_j is the expected number of lung cancers for the jth exposure group, X_j is the median continuous dose expressed in mg/m^3-years for the jth exposure group , and β is the estimated parameter for the relative risk model (given in Table 1). The numbers of lung cancers predicted by the model for each exposure group are presented in Table 3. The goodness-of-fit Chi-Square is 14.0 on 18 degrees of freedom, therefore, OSHA concludes that the model provides a reasonable fit to the observed data.

Gail describes an approach for estimating the excess risk of cancer death due to constant exposure to environmental carcinogens in the presence of competing risks (From Cadmium docket #, Ex. 8–651). This method is easily adapted to estimate the excess risk of lung cancer death due to occupational exposure to asphalt.

Occupational dose was first converted to continuous dose on the assumption that exposure occurs 8 hours per day, 5 days per week and 48 weeks per year. The lung cancer death rates, which were given for five-year age intervals, were assumed to be constant throughout each interval.

Let d_i = the cumulative dose received at the midpoint of the ith age interval. Thus, for example, if an individual is exposed at a constant level X from age 20 on, then at age 24, d_{24} would equal 4.5X. From age 65 on, cumulative exposure would be 45X. Let $q_x(i)$ = the probability of death from all causes at age i, and let $q_L(i)$ = the probability of lung cancer death at age i.

Using the relative risk model and the MLE of β derived from this model above, the lifetime excess risk of lung cancer due to 45 years of occupational exposure to asphalt fume is given by

$$7\sum_{i=20}^{j}\beta d_i q_L(i)\exp[-$$

$$\sum_{j=20}^{j}\beta d_j q_L(j)+q_x(j).$$

Table 3 presents estimates of excess deaths derived from the Hansen data using the method described here. In addition, 95% upper confidence limits and 5% lower confidence limits were constructed for each of the MLEs. This was done by replacing β by $\beta\pm1.645$ SE(β) and using the formulas above.

Using data for the entire cohort, and assuming no substantial contribution to the lung cancer risk from CTPV, exposure to 0.2 mg/m^3 of asphalt fume for the occupational lifetime is associated with an estimated excess cancer death rate of one per 1,000 workers. This rate is based on the maximum likelihood estimate. The 95-percent upper bound estimate or risk corresponding to this dose is about one and a half excess lung cancer deaths per 1,000 workers. By comparison, the maximum likelihood estimate of risk for lifetime exposure to 0.5 mg/m^3 or 1.0 mg/m^3 are 2.5 and 5 excess deaths per 1,000 workers respectively. The estimate at a lifetime exposure of 5 mg/m^3 is 25 deaths per 1,000 workers.

A similar assessment of risk was performed using data representing only that portion of the cohort which was born between 1930 and 1960. Dr. Hansen believes that these workers did not have any exposure to CTPV, and the Asphalt Institute believes they had some

exposure. The resulting risk estimates were higher than those of the entire cohort of mastic workers. However, the numbers of deaths in this cohort were quite small and accordingly, the risk estimates have wide confidence intervals. Therefore the combined cohort may be more useful for estimates of risk.

In extensive submissions to OSHA, the Asphalt Institute (AI) presented its research program and the results of research to date. Included was a report by A. Kriech and J. Kurek in which they described the chemical analyses of core samples of Copenhagen roadways, purported to have been paved with mastic asphalt by the cohort of workers studied by Dr. Hansen. AI requested that the Danish Road Laboratory locate pavements and take core samples from roadways applied approximately every five years. In this report, core samples were identified by year of application and street name. AI concluded from the quantification of polynuclear aromatic hydrocarbons present in the core samples, that "* * * this study has produced persuasive evidence that the workers studied by Dr. Hansen were exposed to significant concentrations of coal tar fumes * * *"

Dr. Hansen, who performed the study, has informed OSHA that in interviews with mastic asphalt workers and other plant personnel, she was told that following World War II, coal tar use was avoided. For example, an engineer who formulated asphalt at one of the studied facilities related to Dr. Hansen that coal tar was not used at his company in either the lower (Pulver) layer or the mastic layer after 1949. He further stated that migration of coal tar from lower layer(s) into mastic asphalt would make the mastic asphalt less suitable by altering its properties making it soft when warm and brittle when cold.

Further, Dr. Hansen has informed OSHA that the core sampling and analyses will be repeated under controlled circumstances by an independent institution. OSHA anticipates that these data will become available to the Agency during this rulemaking. The evidence presented by Dr. Hansen is not certain because some of it relies heavily on human recollection.

The evidence for concomitant exposure presented by AI is also not certain. The accuracy of the records is undetermined. Information regarding the nature of any repairs of the sampled roadways subsequent to application of the mastic asphalt, which might have included coal tar, was not included in the report. The potential migration of the measured chemical components of the core samples was not discussed. In addition, it is unclear to what degree the PAH measurements made by AI discriminate between asphalt and coal tar. Moreover, it is not clear that the AI core samples were taken from the roads which were paved by the studies workers, who paved specifically with mastic asphalt.

If AI's suppositions are accurate and part of the increased risk of lung cancer in the Danish mastic asphalt worker cohort was due to concomitant exposure to coal tar pitch volatile (CTPV), the risk estimates due to asphalt exposure would be less certain. If Dr. Hansen's views are the better supported, then more confidence is given to the risk estimates.

OSHA is proposing a PEL for asphalt fumes of 5 mg/m³ total particulate as an 8-hr. TWA for general industry, construction, maritime and agriculture. That was the level which OSHA proposed for general industry in 1988, and is the ACGIH recommendation. NIOSH's original recommendation was 5 mg/m³ as a 15-min. STEL. One of OSHA's procedures for the Air Contaminants rulemaking was to make use of ACGIH and NIOSH recommendations for its proposals, but base its final decisions, of course, on all evidence, comments, and views.

Subsequent to OSHA's original proposal and the recommendations of NIOSH and ACGIH, new studies have become available. However, at this time only one commenter, the Asphalt Institute, has provided comment on these new studies (Docket office, Docket #H-020A). The Norseth study is so recent that OSHA has not received a single comment on it as yet.

In light of the number of significant new studies and the varying conclusions that can be drawn from them, arguments can be made to support PELs of 5 mg/m³, 1 mg/m³, 0.5 mg/m³ and 0.2 mg/m³. The selection of a PEL depends on one's conclusions as to the level at which irritation and other respiratory effects occur, the relevance and persuasiveness of the carcinogenicity evidence, and the relevance of the risk assessment. The Regulatory Impact Analysis presents exposure data and other information as to feasibility of all of these levels.

OSHA requests comments on all the evidence, studies and analyses, its proposal of 5 mg/m³ total particulate as an 8-hr. TWA, the other exposure limits mentioned, the appropriate method of measurement and all other relevant issues. Comment is also requested on whether different fractions of asphalt may have different health effects, and whether it may be appropriate to take this into consideration in final regulatory decisions.

TABLE C6-3.—OBSERVED AND EXPECTED CASES OF LUNG CANCER BY AGE AND SUBCOHORT AMONG DANISH MASTIC ASPHALT WORKER COHORT

Age (years)	X^1 Dose (mg/m³-yr)	Subcohort					
		I		II		III	
		O_i	E_i	O_i	E_i	O_i	E_i
30–34	54			0	0.00	0	0.01
–39	81			0	0.00	0	0.03
–44	108	0	0.00	0	0.02	1	0.08
–49	135	0	0.02	1	0.07	1	0.17
–54	162	0	0.14	2	0.35	1	0.11
–59	189	2	0.44	2	0.72		
–64	216	4	1.29	1	0.37		
–69	243	5	1.77				
–74	243	4	1.42				
–79	243	3	0.61				
–84	243	0	0.24				
–89	243	0	0.03				

E^1—expected lung cancer cases, assuming no asphalt exposure.
O^1—observed lung cancer cases.

TABLE C6–4.—ESTIMATES OF EXCESS LUNG CANCER DEATHS PER 1,000 WORKERS WITH 45 YEARS OCCUPATIONAL EXPOSURE TO ASPHALT FUME

Dose (mg/m^3)	Continuous dose (mg/m^3)	Number lung cancer deaths	5% lower and 95% upper confidence limit
0.1	0.02	0.5	(0.3, 0.7)
0.2	0.04	1.0	(0.1, 1.5)
0.5	0.11	2.5	(1.4, 3.7)
1.0	0.22	5.0	(2.7, 7.3)
2.0	0.44	10.0	(5.4, 14.6)
4.0	0.88	19.9	(10.8, 28.9)
5.0	1.10	24.8	(13.5, 35.9)
10.0	2.19	48.7	(26.7, 70.0)

TABLE C6–5.—PARAMETER ESTIMATES FROM THE RELATIVE RISK MODEL USING THE HANSEN DATA

Parameter	Subcohort I born 1893–1919	Subcohort II born 1920–1929	Subcohort II born 1930–1960	Combined cohort born 1893–1960
MLE	8.7056E–03	1.5313E–02	4.9589E–02	1.0807E–02
var β	0.9500E–05	0.7410E–04	0.1093E–02	0.9155E–05

Variance is estimated by the observed information.

TABLE C6–6.—ESTIMATES OF EXCESS LUNG CANCER DEATHS PER 1,000 WORKERS WITH 45 YEARS OF OCCUPATIONAL EXPOSURE TO ASPHALT FUME

Dose (mg/m^3)	Continuous dose (mg/m^3)	No. of lung cancer deaths	5% Lower and 95% upper confidence limits
0.1	0.02	0.5	(0.3, 0.7)
0.2	0.04	1.0	(0.1, 1.5)
0.5	0.11	2.5	(1.4, 3.7)
1.0	0.22	5.0	(2.7, 7.3)
2.0	0.44	10.0	(5.4, 14.6)
4.0	0.88	19.9	(10.8, 28.9)
5.0	1.10	24.8	(13.5, 35.9)
10.0	2.19	48.7	(26.7, 70.0)

BISMUTH TELLURIDE (Se–DOPED)

CAS: 1304–82–1; Chemical Formula: Bi_2Te_3

H.S. No. 1034

OSHA has no limit for doped bismuth telluride in the construction, maritime, or agriculture industry. The ACGIH has a TLV®–TWA of 5 mg/m^3 for bismuth telluride that has been doped with selenium sulfide. NIOSH has no REL for this substance. The Agency is proposing a PEL of 5 mg/m^3 as an 8-hour TWA in construction, maritime, and agricultural operations. NIOSH (Ex. 8–47, Table N1) concurred with this limit when the Agency recently established it in general industry.

Bismuth telluride takes the form of gray, hexagonal platelets; it is also available as ingots or single crystals. Bismuth telluride is used for semiconductors and in thermoelectric cooling and power generation applications (ACGIH 1986, p. 59).

Wagner, Madden, Zimber, and Stokinger (1974) conducted a one-year study in which rabbits, dogs, and rats were exposed for 6 hours/day, 5 days/week to doped bismuth telluride dust (containing 80.04 mol % Bi_2Te_3 and 0.20 mol % SnTe, plus a small stoichiometric excess of Te) of 1.04 um particle diameter at a mean concentration of 15 mg/m^3. Small, granulomatous lesions without fibrosis appeared in the lungs of dogs at 6 months. In dogs that were sacrificed four months after an 8-month exposure, the lesions had regressed, and the affected lymph nodes were without cellular reaction. Rabbits exhibited similar histologic effects, but with decreased numbers of pulmonary macrophages, no fibrous tissue proliferation, and no cellular or fibrous tissue reaction around the dust deposits in the lymph nodes. The rats showed fewer granulomas but some areas of epithelialization of the alveolar walls. As was true for the other species, the rats showed neither fibrosis nor cellular reaction in the lymph nodes, despite accumulation of the intermetallic dust (Wagner, Madden, Zimber, and Stokinger 1974).

In humans, exposure to bismuth telluride (whether doped or not is not specified) is reported to lead to eye, nose, and throat irritation (Hazardous Substance Fact Sheet 1985). Exposure to the doped telluride is known to cause garlic breath, a sign of exposure both to tellurium and selenium (Clayton and Clayton 1981, p. 1561).

OSHA is proposing an 8-hour PEL of 5 mg/m^3 TWA for Se-doped bismuth telluride in the construction, maritime, and agriculture industries to prevent the occurrence of pulmonary lesions in exposed workers. OSHA preliminarily concludes that this limit will substantially reduce the significant risk of these pulmonary effects, which constitute material impairments of health. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CARBON BLACK

CAS: 1333–86–4; Chemical Formula: C

H.S. No. 2027

Currently, OSHA has an 8-hour TWA limit of 3.5 mg/m^3 for carbon black in general industry and in the construction and maritime industries. There is no limit in agriculture. The ACGIH TLV®–TWA and NIOSH REL are both 3.5 mg/m^3; however, NIOSH also recommends that carbon black containing more than 0.1 percent polyaromatic hydrocarbons be handled as a potential human carcinogen. OSHA is proposing to extend its 3.5 mg/m^3 8-hour TWA PEL to agricultural workplaces; this action will make the PEL for carbon black consistent across all regulated sectors.

Commercial carbon black is a finely divided form of carbon that contains trace amounts of organic and inorganic impurities adsorbed on the surfaces of the particles. Oil furnace black, which typically contains less than 0.1 percent solvent-extractable impurities, is the most widely used of the carbon blacks in the United States.

Data on humans and animals suggest that exposure to carbon black at high levels is associated with declines in pulmonary function and cardiovascular stress. Oleru et al. (1983) reported a significant loss of pulmonary function among a group of 125 Nigerian carbon

black workers exposed to airborne concentrations as high as 34 mg/m³; radiograms taken on members of this population were normal. Another study, reported in an abstract (Crosbie et al. 1979), found significant annual declines in pulmonary function, as well as radiologic changes, in a group of 35 workers exposed to carbon black concentrations below 10 mg/m³. Nau et al. (1962) exposed hamsters, mice, guinea pigs, rabbits, and monkeys to 56.6 mg/m³ furnace black (carbon black) for prolonged periods of time. Electrocardiographic changes were noted in monkeys after 2500 hours of exposure, and marked right atrial and right ventricular strain were noted in these animals after 10,000 hours of exposure. The authors attributed these effects to the excessive accumulation of carbon black dust in the lung.

More recent studies conducted in U.S. carbon black plants, where OSHA's 3.5 mg/m³ PEL is in force, report no association between exposure and excess morbidity. In a cross-sectional study of pulmonary function in 913 U.S. carbon black workers, Robertson et al. (1988) found no significant declines in pulmonary function after correcting the data for age and smoking habits. Robertson and Ingalls (1989) also conducted a case-control study of all male carbon black production workers in the United States. In this study, a case was defined as a member of the study population who had filed a health insurance claim for a physician's diagnosis of malignant neoplasm, circulatory disease, or pulmonary disease. No associations were found in this study between cumulative exposure level of carbon black and diagnoses of these disorders.

OSHA is proposing an 8-hour TWA PEL of 3.5 mg/m³ for agricultural workplaces. The Agency preliminarily concludes that this limit is necessary to protect workers in this sector against the significant risk of pulmonary and cardiovascular disorders that have been associated with exposure to high concentrations of carbon black. OSHA considers these effects material impairments of health and believes that the proposed limit will substantially reduce this risk. Promulgation of this limit will also make OSHA's PEL for this substance consistent across all regulated sectors.

CHLORINE DIOXIDE

CAS: 10049–04–4; Chemical Formula: ClO_2

H.S. No. 1080

Currently, OSHA has an 8-hour TWA limit of 0.1 ppm for chlorine dioxide in the construction and maritime industries. There is no limit in agriculture. The ACGIH has the same time-weighted average TLV®, plus a 15-minute TLV®–STEL of 0.3 ppm. NIOSH has no REL for this substance. The Agency proposes to add a 15-minute STEL of 0.3 ppm to its current limit in construction and maritime and to extend both the 8-hour TWA and 15-minute PELs to agriculture. NIOSH concurred (Ex. 8–47, Table N1) with these limits when OSHA recently established them in general industry.

Chlorine dioxide is a red-yellow gas at ordinary temperatures. It is used as a biocide, to bleach textiles, paper pulp, flour, cellulose, leather, fats and oils, and in beeswax. Chlorine dioxide is also employed in purification, taste and odor control of water, as an oxidizing agent, and in the manufacture of chlorite salts (ACGIH 1986, p. 118; Hawley's 1987, p. 260). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In addition to respiratory effects, chlorine dioxide can cause eye irritation, gastrointestinal irritation, and reproductive effects in experimental animals. The oral LD_{50} in rats is 292 mg/kg. Exposure causes pulmonary effects and skin irritation in rabbits. Instilled into rabbit eyes, 100 mg of chlorine dioxide caused mild irritation (RTECS 1991). Rats exposed to 0.1-ppm concentrations of chlorine dioxide for 5 hours/day for 10 weeks showed no adverse effects from exposures (Dalhamn 1957/ Ex.1–307). Chlorine dioxide also has reproductive effects in rats and embryotoxic effects in their offspring (RTECS 1991).

Data on human exposures indicate that marked irritation occurs on inhalation of 5 ppm (no time specified), and that one death occurred after exposure to 19 ppm (Elkins 1959). Repeated exposures in humans have been linked to bronchitis and pronounced emphysema (Petry 1954/ Ex.1–1163). Clinical studies conducted by Gloemme and Lundgren (1957/Ex.1–323) revealed that the majority of workers who had been exposed for five years to average concentrations of chlorine dioxide below 0.1 ppm, in combination with about 1.0 ppm chlorine, experienced eye and respiratory tract irritation and slight bronchitis. Some gastrointestinal irritation was also observed in these workers. Gloemme and Lundgren (1957/ Ex.1–323) attributed all of these effects to elevated short-term exposures involving excursions above the 0.1 ppm level. Ferris, Burgess, and Worcester (1967/Ex.1–316) have shown that concentrations occasionally ranging as high as 0.25 ppm were associated with respiratory effects in workers concomitantly exposed to chlorine.

OSHA is retaining the 0.1-ppm 8-hour TWA PEL and is proposing to add a 15-minute STEL of 0.3 ppm for chlorine dioxide in construction and maritime; the Agency also is proposing to extend both limits to agricultural operations. OSHA preliminarily concludes that both of these limits are necessary to protect workers in these sectors from the significant risk of respiratory, skin, and eye irritation known to occur as a result of exposure to this substance. OSHA considers these adverse effects material impairments of health and believes that promulgation of these limits is necessary both to reduce these significant risks and to make OSHA's PELs for this substance consistent across all regulated sectors.

CHROMIUM (II) COMPOUNDS

CAS: 7440–47–3; Chemical Formula: Cr

H.S. No. 2083

OSHA has no limit for the chromium II (divalent) compounds in construction, maritime, or agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. The ACGIH has a TLV®–TWA of 0.5 mg/m³ for the divalent chromium compounds, and OSHA is proposing the same limit for these substances in workplaces in construction, maritime, and agriculture. This action will make OSHA's limit for the chromium II compounds consistent across all industry sectors.

Chromium is a metallic element that can have a valence of 2, 3, or 6; the divalent, or chromium II, compounds include such substances as chromous bromide, chromous carbonate, chromous chloride, chromous fluoride, and chromous sulfate. These compounds are used as reducing agents, catalysts, reagents, and oxygen scavengers, and as components of electroplating solutions (Hawley's 1987, p. 282).

The chromium compounds vary greatly in toxicity, but the chromium II compounds in general are believed to have a relatively low order of toxicity (ACGIH 1986, p. 139). The available toxicological literature often does not distinguish between the various compounds, and information on the divalent form is rare. There are no acute toxicity studies in animals for the divalent forms. These compounds, however, do appear to have sensitization potential when in contact with the skin. Some workers have demonstrated positive patch tests after exposure to divalent chromium

compounds in the workplace (Burrows 1984).

OSHA is proposing an 8-hour TWA limit of 0.5 mg/m³ for the chromium II compounds in construction, maritime, and agriculture. This is the PEL currently in effect in general industry, and the Agency believes this limit is necessary both to reduce a significant risk of sensitization among construction, maritime, and agricultural workers and to make the Agency's limit for these substances consistent across all regulated sectors. OSHA considers dermal sensitization a material health impairment and believes that the proposed PEL is necessary to substantially reduce this significant risk.

CHROMIUM III COMPOUNDS
CAS: 7440–47–3; Chemical Formula: Cr
H.S. No. 2039A

OSHA has no limit for the chromium III (trivalent) compounds in construction, maritime, or agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. The ACGIH has a TLV®–TWA of 0.5 mg/m³ for the trivalent chromium compounds, and OSHA is proposing the same limit for these substances in construction, maritime, and agriculture. This action will make OSHA's PEL for the chromium III compounds consistent across all industry sectors.

Chromium is a metallic element that can have a valence of 2, 3, or 6; the trivalent, or chromium III, compounds include such substances as chromic oxide, chromic sulfate, chromic chloride, chromic potassium sulfate, chromic carbide, and chromite ore (ACGIH 1986, p. 139; Clayton and Clayton 1981, p. 1591). Chromic compounds are used as textile mordants, paint pigments, and catalysts, in refractory bricks, and in gauge blocks, hot extrusion dyes, and as a spray-coating material (Clayton and Clayton 1981, pp. 1590–1591).

The oral LD_{50}'s for various chromic salts range from 600 to 2600 mg/kg (Smyth, Carpenter, Weil, Pozzani, Striegel, and Nycum 1969). Guinea pigs repeatedly exposed to chromic salts developed allergic contact dermatitis (National Research Council Canada 1976). Mice pretreated with a 12 mg/kg dose of chromium III compounds experienced a 250 percent increase in mortality when subsequently challenged with a 120 mg/kg dose of chromium III (Yoshikawa 1970). In-vitro studies show that exposure to chromic compounds reduced the viability of rabbit alveolar macrophages and increased cell lysis (Walters et al. 1975). Rats exposed for one year via their drinking water to 0 to 25 mg/liter chromium III showed no significant differences from controls in body weight gain, appearance, or blood or tissue pathology (MacKenzie, Byerrum, Decker, Hoppert, and Langham 1958). Rats exposed to chromium oxide pigments in the diet for 2 years showed no increase over controls in tumor rates (Ivankovic and Preussman 1975).

In humans, exposure to the dust of chromite ore has caused "exaggerated" lung markings on chest roentgenograms, and other groups of ore workers have developed pneumoconiotic changes consistent with histologic fibrosis (ACGIH 1986, p. 139; Mancuso and Hueper 1951). One case of asthma caused by exposure to a chromic compound (chromic sulfate) has been reported and confirmed in a plating worker (Novey et al. 1983). Eighteen percent of 85 chrome factory workers exposed to 4.5 to 9.2 mg/m³ Cr III compounds developed chronic bronchitis after 5 years of exposure, and almost 6 percent of these factory workers developed pneumoconiosis after 9 years of exposure (Mikov 1967, in HSDB 1990).

Based on these effects in humans and animals, OSHA is proposing an 8-hour TWA PEL of 0.5 mg/m³ for the chromic (Cr III) compounds of chromium; this limit would apply in construction, maritime, and agricultural workplaces. OSHA believes the proposed PEL is necessary to reduce the significant risk of chronic respiratory effects in workers in these sectors who are exposed to these compounds. The Agency considers chronic respiratory effects such as pneumoconiosis and chronic bronchitis material impairments of health. Promulgation of this PEL will also make OSHA's limit for the Cr III compounds consistent across all regulated sectors.

CHROMIUM, METAL
CAS: 7440–47–3; Chemical Formula: Cr
H.S. No. 1093

OSHA currently has an 8-hour TWA PEL of 1 mg/m³ for chromium metal in general industry, construction, and maritime. There is no limit in agriculture. NIOSH has no REL for this substance, and the ACGIH has established an 8-hour TLV®–TWA of 0.5 mg/m³ for chromium. The Agency is proposing a 1 mg/m³ 8-hour TWA limit for chromium in agricultural operations. NIOSH concurs (Ex. 8–47, Table N1) that this limit is appropriate. Promulgation of this limit will make OSHA's PEL for chromium consistent across all regulated sectors.

Chromium is a hard, brittle, semi-gray metal. Chromium is used for corrosion resistance and as an alloying and plating element on metal and plastic substrates. It is also used as a protective coating for automotive and equipment accessories, in nuclear and high-temperature research, and in inorganic pigments (Hawley's 1987, p. 280).

Chromium metal is relatively nontoxic. Some studies in experimental animals have shown that a few animals develop tumors after intravenous injection; however, the relevance of these results for occupational exposure is not clear. In the general industry rulemaking, OSHA found that the preliminary markings reported in the Mancuso and Hueper (1951/Ex.1–215) study did not present a risk of material impairment of health in exposed workers because these markings did not presage any decrement in pulmonary function or interfere with the functional capacity of exposed workers. A study by Princi et al. (1962) reflects the problem of confounding exposures in the case of chromium. In this study, ferrochrome alloy workers were exposed to several toxic contaminants simultaneously, including chromium salts, silica, iron oxide, and chromium metal. OSHA believes it likely that exposure to the other contaminants present, which included a high percentage of silica, accounts for the development of pulmonary disease in these workers. The ACGIH (1986/Ex.1–3, p. 139) stated, after reviewing the Mancuso and Hueper (1951/Ex.1–215) and the Princi et al. (1962, as cited in ACGIH 1986/Ex.1–3, p. 139) studies, that "[e]xposure to chromium metal does not give rise to pulmonary fibrosis or pneumoconiosis." Other authors (Proctor, Hughes, and Fischman 1988) concur in this assessment of the relative lack of toxicity of the metallic form of chromium.

Based on this evidence, OSHA is proposing an 8-hour TWA limit of 1.0 mg/m³ for chromium metal in the agriculture industry. The Agency believes that this limit is adequate to protect workers in agriculture from the significant risk of pulmonary effects potentially associated with exposure to the metallic form of chromium. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

COAL DUST, < 5% QUARTZ
COAL DUST, > 5% QUARTZ
CAS: None; Chemical Formula: None
H.S. Nos. 1096 and 1097

In the construction and maritime industries, OSHA's current limits for coal dust are expressed as a formula limit of $10mg/m^3/\%SiO_2+2$ (8-hour TWA) for coal dust containing a respirable quartz fraction greater than 5 percent and a 2.4-mg/m³ 8-hour TWA

limit for coal dust containing a respirable quartz fraction of less than 5 percent. There is no limit in agriculture. The ACGIH has a TLV®-TWA of 0.1 mg/m³ for the respirable quartz fraction of coal dust containing more than 5 percent quartz and of 2 mg/m³ for the respirable dust fraction of coal dust containing less than 5 percent quartz. NIOSH has no RELs for this substance. In agriculture, OSHA is proposing an 8-hour TWA limit of 0.1 mg/m³ for the respirable quartz fraction of coal dust containing more than 5 percent quartz and an 8-hour TWA limit of 2 mg/m³ for the respirable dust fraction of coal dust containing less than 5 percent quartz. The Agency is also proposing that the current limit for coal dust containing more than 5 percent respirable quartz in construction and maritime be expressed as mg/m³, which reflects the current approach to airborne particulate measurement; this action would establish a PEL of 0.1 mg/m³ for coal dust containing more than 5 percent respirable quartz. OSHA's proposed limit in these two sectors thus does not represent an actual change in the value of the limits for coal dust containing more than 5 percent respirable quartz; instead, it would do away with the Agency's cumbersome formula limit. In all sectors—construction, maritime, and agriculture—OSHA is proposing a PEL for coal dust containing less than 5 percent quartz of 2 mg/m³.

Coal is a dark brown to black solid formed from fossilized plant material. Coal is used as a fuel and in the manufacture of fertilizers, insecticides, disinfectants, synthetic rubber, and in the production of coke, coal gas, water gas, and coal tar compounds (ACGIH, 1986, p. 142.1 (87)).

The literature on occupational exposure to coal dust does not generally distinguish between dusts containing less than 5% respirable quartz and dusts containing a higher percentage of respirable quartz. The discussion below also does not make this distinction.

Animal data on coal dust effects are sparse. Rats inhaling 6600 μg/m³ coal dust for 86 weeks (6 hours daily) developed lymphomas, and those inhaling 14,900 μg/m³ on the same regimen developed tumors of the adrenal cortex (AIHAJ 1981).

Exposure to coal dust can cause respiratory effects such as pneumoconiosis, bronchitis, emphysema, and progressive massive fibrosis (PMF) in humans. Coal worker's pneumoconiosis (CWP) often occurs concomitantly with chronic bronchitis and emphysema. The small opacities associated with CWP are most frequently located in the upper lung zones. CWP is also a precursor to progressive massive fibrosis, which is associated with reduced ventilatory capacity, low arterial oxygen tension, pulmonary hypertension, and premature death (Morgan 1984). A study conducted from 1969 to 1971 showed that, in 9,076 U.S. miners, there was an overall 30 percent prevalence of CWP, with 2.5 percent experiencing progressive massive fibrosis (Morgan 1973). In some workers reporting symptoms of chronic bronchitis, even moderately low exposure to coal mine dust was associated with severe lung function impairment (Hurley and Soutar 1986). A recent study in Welsh and English miners demonstrates that lifetime exposure to coal mine dust is related to increased mortality due to pneumoconiosis, bronchitis, or emphysema (Miller and Jacobsen 1985).

NIOSH (Ex. 8–47, Table N2; Tr. p. 3–86) believes that the limit for quartz-bearing coal dust should be reduced to 0.05 mg/m³ as an 8-hour TWA on the basis of the potential carcinogenicity of respirable crystalline silica. OSHA is aware of some recent studies (NIOSH 1986b; Hurley and MacLaren 1987; IARC 1987) on the health effects of exposure to coal dust, and the Agency is monitoring this literature to assess the need for a reevaluation of this limit in the first PEL Update. However, OSHA's primary objective in the present rulemaking is to achieve consistency of limits across all sectors, and the adoption of the proposed coal dust limit in agriculture will achieve that goal. In addition, expressing these limits in terms of mg/m³ rather than the formula will simplify measurement in workplaces in construction and maritime.

Accordingly, OSHA is proposing an 8-hour TWA PEL of 0.1 mg/m³, measured as the respirable dust fraction, for coal dust having a respirable quartz fraction of more than 5 percent quartz, and an 8-hour TWA PEL of 2 mg/m³, measured as the respirable dust fraction, for coal dust having a respirable quartz fraction of less than 5 percent quartz; these limits are being proposed for workplaces in the construction, maritime, and agriculture industries. The Agency's previous formula limit for silica containing more than 5 percent quartz (respirable fraction) is equivalent to the 0.1-mg/m³ limit in terms of airborne concentration, and the proposed limit in construction and maritime is thus intended merely to simplify the units used to measure and express the limit. OSHA believes that this revision will simplify employee exposure monitoring. For coal dust having a respirable quartz fraction of less than 5 percent, OSHA is proposing to lower the 8-hour TWA limit from 2.4 mg/m³ to 2 mg/m³ in construction and maritime and to extend this limit to agriculture. Promulgation of these limits will make OSHA's PELs for coal dust consistent across all regulated sectors.

COTTON DUST

CAS: N.A.; Chemical Formula: Not applicable

H.S. No. 2041

In general industry, OSHA's permissible exposure limits (PELs) for cotton dust are: 200 μg/m³ in yarn manufacturing and cotton washing operations; 500 μg/m³ in textile mill waste house operations or in yarn manufacturing using "lower grade washed cotton;" and 750 μg/m³ in slashing and weaving operations. There is also a PEL of 1 mg/m³ that applies to cotton waste processing operations (see 29 CFR 1910.1000, Table Z–1–A). These exposure concentrations are averaged over an 8-hour period, as measured by a vertical elutriator or an equivalent instrument. Currently, the construction, agriculture, and maritime sectors have no PELs for cotton dust. The ACGIH TLV®-TWA for cotton dust is 0.2 mg/m³ for lint-free cotton dust with fibers less than 15 microns in length. The NIOSH REL is less than 200 μg/m³ (lint-free, cotton dust). OSHA is proposing to establish an 8-hour TWA PEL of 500 μg/m³ for cotton dust in the construction, agriculture, and maritime industries. Promulgation of this limit will make the PEL for cotton dust in construction, maritime, and agriculture consistent with the PEL for "all other operations" specified in OSHA's cotton dust standard. Also, the Advisory Committee on Construction Safety and Health recommended a 500 μg/m³ PEL for cotton dust in construction.

Cotton is commercially grown in at least 19 States and is a major crop in 14. Most of the crop is harvested and ginned in the fall and early winter; it is processed by domestic and foreign mills on a year-round basis. Hence, storage and transportation are major operations in the cotton industry. The cotton industry of the United States can be divided into several processes: harvesting; ginning; warehousing and compressing of cotton lint; classing and marketing of cotton lint; fabric manufacturing using cotton yarn; reclaiming and marketing of textile manufacturing waste; delintering of cottonseed; marketing and converting of linters; reclaiming and marketing of ginmotes; and batting, yarn, and felt manufacturing using waste cotton fibers and byproducts.

There are no data on the toxicity of cotton dust in animals. In humans, exposure to cotton dust causes byssinosis, a specific respiratory disease whose signs and symptoms are attributable to the action of cotton dust on the respiratory passages. The effects of byssinosis can be temporary or permanent, depending on the exposure and the individual, and can lead in time to chronic obstructive lung disease, primarily chronic bronchitis (Harris et al. 1972; Daum et al. 1974). Initially, the individual notices a tightness in the chest occurring on the first day of the work week. The tightness may be accompanied by a measurable decrease in breathing capacity, as shown by pulmonary function tests. Usually, the condition is mild and temporary at first, tending in time to progress to the stage of bronchitis by the end of the work week. This progression, which is characterized by constriction of the bronchial tubes of the lung, leads to a permanent narrowing of these airways. The individual develops a chronic cough with the production of phlegm and increasing shortness of breath. At this stage, the condition is readily detectable by pulmonary function measurements. Total disability and even death may follow. The description of these detailed symptoms appeared as early as 1908 in the work of Collis (1932).

As a result of the obviously subjective quality of the early symptoms of byssinosis, investigators have, in the past few years, subdivided byssinosis into different categories for purposes of diagnosis and treatment, namely: (a) the reversible symptomatic or physiological condition (sometimes referred to as a reactor state), and (b) the chronic irreversible lung disease.

Schilling (1955, 1963) distinguished the reversible symptoms of Grade one-half, Grade one, and Grade two byssinosis from permanent incapacity, which he labeled Grade three byssinosis. According to Schilling's classification, byssinosis is graded as follows:

(a) Grade one-half—occasional chest tightness on the first day of the work week.

(b) Grade one—chest tightness and/or breathlessness on Mondays only.

(c) Grade two—chest tightness and/or breathlessness on Mondays and other days.

(d) Grade three—grade two symptoms accompanied by evidence of permanent incapacity from diminished effort tolerance and/or reduced ventilatory capacity.

In their studies of textile mills in the United States, Merchant et al. (1973) reached the conclusion that exposure to 500 $\mu g/m^3$ cotton dust would result in a 25 percent prevalence of all grades of byssinosis. These investigators, using the vertical elutriator, found a strong linear association between the prevalence of byssinosis and the airborne concentration of lint-free dust (less than an aerodynamic equivalent diameter of 15 microns). In cotton preparation and yarn areas, exposure to untreated cotton was shown to produce a 3 percent prevalence of byssinosis (all grades) at 50 ug/m^3, a 7 percent prevalence at 100 ug/m^3, and a 13 percent prevalence at 200 ug/m^3. However, OSHA's experience under the cotton dust standard has demonstrated that the prevalence of byssinosis has been reduced substantially more by the cotton dust standard (see 50 FR 51120, Dec. 13, 1985) than these figures would suggest.

Byssinosis is the respiratory disease most commonly associated with exposure to cotton dust, but other diseases such as chronic bronchitis, mill fever, weavers' cough, and mattress makers' fever have also been associated with cotton dust exposure. These diseases and additional studies on byssinosis have been described and their association with cotton dust exposure has been documented extensively in previous **Federal Register** publications (41 FR 56500–56502; 42 FR 27352–27354; 48 FR 26964–26968; 50 FR 51120).

OSHA's cotton dust standard was based on data demonstrating significant excess risk of byssinosis and other respiratory symptoms in workers exposed to cotton dust in the early 1970s and before. Those data also showed that reductions in exposure substantially reduced risk. Both the Court of Appeals and Supreme Court upheld OSHA's analysis of the studies and OSHA's conclusion that a standard was needed to substantially reduce a significant risk of disease. Cotton dust is, of course, present in agriculture (cotton ginning) and marine transportation operations.

OSHA therefore believes that, in the absence of PELs, workers in agriculture, construction, and maritime are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed limit of 500 $\mu g/m^3$ as an 8-hour TWA is necessary to significantly reduce these risks of material health impairment among workers in the construction, maritime, and agriculture sectors. Promulgation of these limits will establish PELs for cotton dust in all OSHA-regulated sectors.

The vertical elutriator, though a better measuring device for correlating exposure with health effects, is not practical where area samples cannot be taken retrospectively, and these devices are also bulky. OSHA will permit employers to substitute a respirable dust personal sampler where it is not feasible to use a vertical elutriator. OSHA proposes that in those circumstances where a personal sampler is used, the exposure limit be 1 mg/m^3 respirable dust, personal sampler. Evidence in the 1978 rulemaking indicated that such a limit is roughly equivalent to 500 $\mu g/m^3$ as measured by a vertical elutriator.

ETHYL ACRYLATE

CAS: 140–88–5; Chemical Formula: $CH_2 = CHCOOC_2H_5$

H.S. No. 1161

OSHA currently has an 8-hour TWA limit of 25 ppm for ethyl acrylate, with a skin notation, in the construction and maritime industries. There is no limit in agriculture. NIOSH has no REL for this substance. The ACGIH has a TLV®–TWA of 5 ppm, a TLV®–STEL of 25 ppm, and a skin notation for ethyl acrylate. The Agency is proposing an 8-hour TWA PEL of 5 ppm and a 15-minute STEL of 25 ppm, with a skin notation, for ethyl acrylate in the construction, maritime, and agriculture industries. Promulgation of these limits will make the PELs for this substance consistent across all OSHA-regulated sectors.

Ethyl acrylate is a colorless liquid with an acrid odor. This substance is used in the manufacture of acrylic resins and in emulsions and solution polymers for the surface coating of textiles, paper, and leather. It is also used to produce acrylic fibers, adhesives, and binders and as a fragrance and flavoring agent (ACGIH 1986, p. 240).

Ethyl acrylate produces irritation of the skin, eyes, mucous membranes, and respiratory system (Dreisbach 1974/Ex. 1–896). The oral LD_{50} in rats is 800 mg/kg, and the 4-hour LC_{50} in the same species is 2180 ppm (RTECS 1991). In rabbits, the dermal LD_{50} is 1834 mg/kg (RTECS 1991). In contact with the eyes or skin of rabbits, ethyl acrylate produces mild irritation (RTECS 1991). Animal studies indicate that severe chronic effects may result from exposure to this substance. Rats exposed to levels of 70, 300, or 540 ppm of ethyl acrylate for up to 30 days showed accelerated mortality and pathologic changes in the lungs, liver, and kidneys. In those animals that developed pneumonia, renal and hepatic lesions were also seen. In a parallel study, rats, rabbits, and guinea pigs who were subjected to ethyl acrylate concentrations in excess of 75 ppm for 50 seven-hour inhalation periods exhibited pulmonary edema before death and had degenerative changes in the heart, liver, and kidneys

at autopsy (Treon, Sigmon, Wright, and Kitzmiller 1949/Ex. 1–769). Miller et al. (1980, as cited in ACGIH 1986/Ex. 1–3, p. 240) reported that rats and mice exposed to 75 or 225 ppm for 6 hours per day for 30 days developed nasal lesions and other degenerative inflammatory changes in the nasal structure. In other studies, rats and mice administered 100 or 200 mg/kg ethyl acrylate by gavage five times per week for 103 weeks developed inflammation and hyperplasia of the forestomach in addition to squamous cell carcinomas and papillomas in the same area (NTP 1983b). A recent study (Moore, Amtower, Doerr, Brock, and Dearfield 1988) has shown that ethyl acrylate is genotoxic in mammalian test systems.

In a study by Nemec and Bauer (1978, as cited in ACGIH 1986/Ex. 1–3, p. 240), human volunteers experienced drowsiness, headache, and nausea after prolonged inhalation exposures to this substance at 50 to 75 ppm. Opdyke (1975/Ex. 1–922) reported that the application of a 4-percent concentration of ethyl acrylate produced skin-sensitization reactions in 10 out of 24 volunteers. A recent study (Condesalazar, Guimaraens, and Romero 1988) has shown that occupational exposure to ethyl acrylate is associated with the development of allergic contact dermatitis in as many as 12 percent of exposed workers. Another recent study in chemical plant workers (Schwartz, Doty, Monroe, Frye, and Barker 1989) showed that exposure to ethyl acrylate in the work environment is associated with a decrement in olfactory ability, although the clinical significance of this finding is unclear.

In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA PEL of 5 ppm, a 15-minute STEL of 25 ppm, and a skin notation for ethyl acrylate. The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant risk of respiratory tract irritation, skin sensitization, and nausea and drowsiness associated with exposure to this substance. The Agency considers these adverse effects material impairments of health and believes that the proposed PELs are necessary to substantially reduce these risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

FERROVANADIUM DUST

CAS: 12604–58–9; Chemical Formula: FeV

H.S. No. 1177

OSHA currently has a limit of 1 mg/m³ as an 8-hour TWA for ferrovanadium dust in the construction and maritime industries. There is no limit in agriculture. The ACGIH has a TLV®–TWA limit of 1 mg/m³ with a TLV®–STEL of 3 mg/m³, and NIOSH has a 10-hour REL for this substance (measured as vanadium). The Agency is retaining its 8-hour TWA PEL of 1 mg/m³ in construction and maritime, proposing to add a STEL of 3 mg/m³ in these sectors, and proposing to extend both limits to agriculture. NIOSH concurred (Ex. 8–47, Table N1) with these limits when the Agency recently established them in general industry.

Ferrovanadium dust takes the form of dark, odorless, solid particles. It is generated in the preparation of steel that contains vanadium (ACGIH 1986, p. 269).

In addition to respiratory effects, ferrovanadium dust causes mild eye and nose irritation. Roshchin (1952/Ex. 1–1166) reported that no acute intoxication occurred in animals exposed to ferrovanadium dust at concentrations as high as 10,000 mg/m³; however, serious chronic pulmonary changes were observed after short-term exposures (one hour) on alternate days for two months to concentrations in the 1000- to 2000-mg/m³ range. These pulmonary changes consisted of chronic bronchitis and chronic lung inflammation (Roshchin 1952/Ex. 1–1166).

Workers exposed to unspecified concentrations of this dust developed mild irritation of the eyes and respiratory tract (Roberts 1965). –OSHA is retaining its 8-hour TWA PEL of 1 mg/m³ in construction and maritime, proposing to add a 3 mg/m³ STEL in these two sectors, and proposing to extend both limits to agriculture. The Agency preliminarily concludes that the combined TWA limit and STEL will substantially reduce the significant risk of eye and upper respiratory tract irritation and of chronic pulmonary damage shown to be associated with exposures to this substance. OSHA considers these adverse health effects to be material impairments of health and believes that the proposed PELs are necessary to substantially reduce this risk. In addition, promulgation of these limits will make OSHA's PELs for ferrovanadium dust consistent across all OSHA-regulated sectors.

FIBROUS GLASS, INCLUDING REFRACTORY CERAMIC FIBERS

CAS: None; Chemical Formula: None

H.S. No. 1178

Fibrous glass is used primarily for thermal and acoustical insulation of residential and commercial buildings. Refractory ceramic fibers (RCF) are primarily used for high temperature insulation applications, including blanket linings for industrial furnaces and vacuum-formed parts for specialty products with high-temperature tolerances. There are currently no specific OSHA limits governing occupational exposure to fibrous glass or RCF, although these substances are covered by the Agency's 15 mg/m³ (5 mg/m³ respirable) 8-hour TWA limit for all inert dusts and particulates. The ACGIH (1986/Ex. 1–3) has a 10 mg/m³ TLV®–TWA for fibrous glass. NIOSH (1977/Doc. H–043) recommended a 5 mg/m³ 8-hour TWA limit for total dust and a 3 fibers/cc limit for airborne fibers less than 3.5 μm in diameter and longer than 10 μm in length. OSHA is proposing an 8-hour TWA PEL of 1 f/cc for fibrous glass, including RCF, in general industry and in the construction, maritime, and agricultural industries.

Fibrous glass is used primarily for thermal and acoustical insulation of residential and commercial buildings. Fibrous glass is produced either as glass wool or glass filament (IARC 1988, Vol. 43). Glasswool is produced by drawing, centrifuging, or blowing molten glass and compromises cylindrical fibers of relatively short length (compared to filaments) (Boyd and Thomason 1980; McCrone 1980). Glass filaments are continuously drawn or extruded from molten glass. This class of materials includes longer, large-diameter filaments for textile and reinforcing applications as well as fine-diameter filaments (Mohr and Rowe 1978).

In the production of glass fibers, finely powdered sand is used as the major source of silica, and kaolin clay and synthetic aluminum oxides are the most common sources of aluminum. Boric oxide is introduced primarily from colemanite (a natural calcium borate), boric acid, and boric acid anhydride. Powdered dolomite [$CaMg(CO_3)_2$] or burnt dolomite (MgO•CaO) is used to introduce magnesium oxide (magnesia) and calcium oxide. Uncalcined and calcined limestone are used as magnesia-free sources of calcium oxide. Fluorspar (CaF_2) is used to introduce fluoride. Sodium sulfate is added to the glass mixture as a firing agent and to assist in dissolving residual grains of sand. Iron oxide (Fe_2O_3) may be added to assist the fiber-drawing process (Loewenstein 1983; Harben and Bates 1984).

Ceramic fibers comprise a wide range of amorphous or crystalline synthetic mineral fibers characterized by their refractory properties (i.e., stability at high temperatures) (IARC 1988, Vol. 43). Ceramic fibers are typically made of alumina, silica, and other metal oxides

or, less commonly, of nonoxide materials, such as silicon carbide (Arledter and Knowles 1964). Most ceramic fibers are composed of alumina and silica in an approximate 50/50 mixture. Monoxide ceramics, such as alumina and zirconia, are composed of at least 80 percent of one oxide, by definition; usually, they contain 90 percent or more of the base oxide, and specialty products may contain virtually 100 percent. Other ceramic fibers prepared for special applications may incorporate thoria, magnesia, beryllia, titania, hafnia, yttria, or potassium titinate. Nonoxide specialty ceramic fibers, such as silicon carbide, silicon nitride, and boron nitride, have also been produced (Arledter and Knowles 1964; Miller 1982; U.S. Environmental Protection Agency 1986).

Non-RCF Fibrous Glass Nonmalignant Respiratory Disease

Human Studies–

A number of epidemiological studies and case reports of workers exposed to fibrous glass have shown that these workers have an excess risk of nonmalignant respiratory disease (NMRD). A review of 691 physicians' reports of adverse effects caused by exposure to fibrous glass identified 66 reports of upper respiratory tract symptoms including rhinitis, sinusitis, pharyngitis, and laryngitis in workers exposed for only one or 1.5 years (Milby and Wolf 1969, as cited in NIOSH 1977/ Doc. H–043, p. 23). Irritation and inflammation of the nasopharyngeal region and upper respiratory tract have also been reported in a number of early studies (Champeix 1945, as cited in IARC 1988/Doc. H–043, p. 136; Trumper and Honigsberg 1946, as cited in NIOSH, 1977/ Doc. H–043, p. 29; Roche 1947, as cited in IARC 1988/Doc. H–043, p.136; Cirla 1948, as cited in IARC 1988/Doc. H–043, p. 136; Mungo 1960, as cited in IARC 1988/Doc. H–043, p. 136). Later reports of nasopharyngeal irritation include those of Müller et al. (1980, as cited in IARC 1988/Doc. H–043, p. 136) and Maggioni et al. (1980 as cited in IARC 1988/Doc.H–043, p. 136).

NIOSH (1990/Doc. H–043) conducted a Health Hazard Evaluation of a factory which manufactures fibrous glass for thermal and acoustical insulation. The evaluation was instigated by a request which stated that employees were experiencing nose bleeds, skin rashes, and respiratory problems. Potential exposures identified in the request were fibrous glass and formaldehyde. The full-shift personal exposure levels of formaldehyde ranged from 0.07 to 0.41 ppm, with a mean exposure of 0.11 ppm. Only two of the personal samples were greater than 0.1 ppm (0.14 and 0.41). The authors stated, however, that the validity of the 0.4 result was questionable when compared to all other personal and area samples collected for this worker. Respirable fibrous glass concentrations ranged from less than 0.01 to 0.07 f/cc. The workers were not following proper work practices to reduce or prevent skin irritation as described in the appropriate Material Safety Data Sheets. They were also not wearing appropriate work clothes (long-sleeve shirts, goggles, and gloves).

Approximately 50 percent of the workers completed a medical questionnaire. The following effects were reported: chronic cough (cough on most days for a period of 3 months or more), 61 percent; phlegm, 53 percent; some degree of shortness of breath, 61 percent; highest level of severity for shortness of breath (having to stop for breath when walking at own pace on level ground), 10 percent; chest tightness, 61 percent (many on all workdays); wheezing, 49 percent. Additional signs and symptoms reported to be present at work two or more times per week included eye irritation, 51 percent; nose irritation, 50 percent; cough, 42 percent; sneezing, 22 percent; sore throat, 10 percent; and nose bleeds, 4 percent.

Several studies report effects on the bronchi. Murphy (1961, as cited in NIOSH 1977/Doc. H–043, p. 28) reported bronchiectasis and multiple focal abscesses in the terminal bronchioles and peribronchial parenchyma, as well as slight pulmonary fibrosis in an electrical worker who dismantled fibrous glass-insulated appliances. Bayliss et al. (1976, as cited in NIOSH 1977/ Doc. H–043, Ex. 1–3, p. 37) studied a group of 1,448 fibrous glass production, packing, and maintenance workers who had been initially employed during the period January 1, 1940 through December 31, 1949 and who had been employed for at least 5 years by June 1, 1972 in the one major fibrous glass construction products manufacturing plant studied. The current mean fiber concentration was 0.08 f/cc. The range was 0.01 to 0.83. Although only limited historical dust measurements were available, it did appear that respirable fiber concentrations were also low about 10 years prior to the present industrial hygiene survey. A statistically significant excess risk of NMRD was found among these workers. In addition, bronchiectasis was found at autopsy in 6 of the 19 fatal cases (Bayliss, written report, 1977, as cited in NIOSH 1977/ Doc. H–043, p. 38).

Enterline and Henderson (1975/Doc. H–043) studied 416 retirees who had worked in six plants that manufactured fibrous glass insulation. A slight, non-statistically significant increase in deaths from NMRD was observed. Out of 35 workers who had retired early due to disability, chronic bronchitis was observed in three workers and expected in 0.5. The authors felt that the latter finding might be important but they pointed out that the numbers were small and that chronic bronchitis is not a well-defined disease entity in many instances.

An increased frequency of bronchitis that was directly related to duration of exposure to synthetic mineral fibers (SMF) has been observed in a group of 135,000 construction workers (Engholm and von Schmalensee 1982/Doc. H–043). The calculated standard ratios for workers in each smoking history category indicated a positive association between bronchitis and exposure to SMF. Among nonsmokers, 77 percent of all cases of bronchitis in workers with long exposure to SMF were attributable to the exposure. The corresponding percentage in present smokers was 28 percent. The authors concluded that there is a clear association between bronchitis and SMF exposure in the construction industry.

Tomasini et al. (1986, as cited in IARC 1988/Doc. H–043, p. 136) have reported on the occurrence of parenchymal involvement of lung tissue in four of seven workers with long-term exposure to fibrous glass in manufacturing. Three of these workers had pulmonary fibrosis and one had both fibrosis and parenchymal involvement. Three workers exposed to fibrous glass during manufacture for 9 to 17 years had respiratory distress and one showed slight pleural thickening on x-ray examination (Chiappini et al. 1981, as cited in IARC 1988/Doc. H–043, p. 136).

Enterline and co-workers (Enterline et al. 1983, Doc. H–043, Ex. 1–17; Enterline et al. 1986, as cited in Vu 1988/Doc. H–043, p. 76; Enterline et al. 1987/Doc. H–043; Enterline 1987/Doc. H–043) studied 16,661 white male workers employed in 17 synthetic mineral fibers plants and who had 1 or more years of experience in production or maintenance during the period 1945–1963. This cohort consisted of 14,815 workers in 11 fibrous glass plants (6, glass wool only; 3, glass filament only; 2, glass wool and glass filament) and 1,846 workers in 6 mineral wool plants. The SMR for nonmalignant respiratory disease (excluding influenza and pneumonia) for all synthetic mineral

fiber workers was statistically significant. In epidemiological studies, the SMR results from the comparison of the number of observed deaths in the study population with the number of expected deaths in a larger population with stable rates. Thus the SMR is equal to the number of observed divided by the number of expected deaths. By convention, this ratio is then multiplied by 100 to get the reported ratio. If this ratio is greater than 100, it means that more deaths are observed in the smaller population than would be expected based on rates in the larger (standard) population. If the ratio is less than 100, fewer deaths are observed than expected. The SMR was 132.3 ($p<0.01$) when U.S. rates were used for comparison and 134.1 ($p<0.01$) when local rates were used. When a difference that is observed between two groups is judged to be statistically significant (e.g., $p<0.01$), this means that differences as large as, or larger than, that observed would occur by chance alone less than (for this example) 1 percent of the time. For workers with greater than 20 years since first exposure in plants which manufactured both fibrous glass wool and filament, there was a statistically significant increase in NMRD (excluding influenza and pneumonia) (SMR 141.8, $p<0.01$, U.S. comparison). For workers in one of these plants, in which the cohort size was 5,651, the SMR was 137.5 ($p<0.01$, U.S. comparison). The mean average exposure concentration for all the fibrous glass plants was 0.039 f/cc and the maximum was 1.5 f/cc. In the plant just cited, the mean exposure concentration was 0.067 f/cc and the maximum was 1.5 f/cc. Among workers in this group who were "ever exposed" to small-diameter fibers, there was a slight nonsignificant increase in NMRD with time since first exposure.

Marsh et al. (1990/Doc. H–043) completed a follow-up to 1985 of the Enterline et al. cohort. The SMR for nonmalignant respiratory disease (excluding influenza and pneumonia) for all MMMF workers was 129.4 ($p<0.01$) when U.S. rates were used for comparison. A breakdown of NMRD rates by process showed no increase for fibrous glass workers when rates were compared to local rates. National rates were not used for comparison. Several studies have observed the occurrence on x-ray examination of opacities in the lungs of a limited number of workers in the fibrous glass manufacturing industry (Nasr et al. 1971, as cited in NIOSH 1977/Doc. H–043, p. 35; Valentin et al. 1977, as cited in IARC 1988/Doc. H–043, p. 135; Weill et al. 1983, 1984, as cited in IARC 1988/Doc. H–043, p. 135; Hill et al. 1973, 1984, as cited in IARC 1988/Doc. H–043, p. 135).

Animal Studies

In animal studies, exposure to non-RCF fibrous glass by several routes has been shown to cause respiratory effects. Schepers and Delahant (1955 as cited in NIOSH 1977/Doc. H–043, p. 41) continuously exposed guinea pigs and rats to glass wool (nominal diameter 6μm; concentration 5.0 to 5.2 mg/m³) by the inhalation route for 20 months and subsequently to "glass cotton" (maximum diameter 3μm; concentration 1.1 to 2.5 mg/m³) for 20 and 4 months, respectively. After 4 months of exposure, the guinea pigs had considerable epithelial hyperplasia (abnormally increased cell number) and cellular desquamation (cells sloughed off) in the smaller bronchioles and cellular infiltration of alveolar walls, with hyperplasia of parenchymal pulmonary lymph nodes (which indicates a reaction of the immune system). Atelectatic (areas of collapsed alveoli) were also present. At the 40th week of exposure, dust-reaction foci were detected that were related to the atelectatic areas. Lung abscesses were found in 10 percent of the rats. These effects indicate a toxic effect on the respiratory tract.

Botham and Holt (1971, as cited in NIOSH 1977/Doc. H–043, p. 43) reported the production of ferruginous bodies in guinea pigs within 2–5 days after inhalation for one 24-hour period of a "high" (not further specified) concentration of glass fibers (≈20 μm length; <1 μm diameter). Ferruginous bodies are protein-coated inclusions which form around fibrous mineral particles. When these bodies form around asbestos fibers they are often called "asbestos bodies." Ferruginous bodies are observed in humans exposed to asbestos. Alveolar lipoproteinosis, another indictor of lung toxicity, developed in rats and hamsters treated by inhalation for 90 days with 400 mg/m³ glass fibers <2 μm in length (Lee et al. 1979, as cited in IARC 1988/Doc. H–043, p. 130; Lee et al. 1981, as cited in Vu 1988/Doc. H–043, p. 52; Lee and Reinhardt 1984, as cited in IARC 1988/Doc. H–043, p. 130). The fibers had small aspect ratios (3:1) and only 7 percent were considered fibrous in shape. Very slight alveolar interstitial fibrosis occurred in a few old animals. Pulmonary inflammation has been reported in rats exposed by inhalation to fibrous glass (diameter 0.1–0.6 μm) for 6 months (Miller 1980, as cited in IARC 1988/Doc. H–043, p. 129).

Fibrosis is seen in experimental animals exposed to mineral fibers. For example, fibrosis is seen in response to asbestos exposure. In humans exposed to asbestos, diffuse interstitial fibrosis and fibrosis of the pleura are observed as components of the disease known as asbestosis. When it becomes massive, fibrosis causes the obliteration of the respiratory capacity of lung. A few of the oldest rats in a group exposed for 24 months to 100 mg/m³ of fibrous glass with an average diameter of 0.5 μm and an average length of 10 μm developed some foci of septal collagenous fibrosis, although most developed only a normal 'dust reaction' (Gross et al. 1970a, as cited in IARC 1988/Doc. H–043, p. 130). Johnson and Wagner (1980, as cited in Vu 1988/Doc. H–043, p. 53) treated rats by inhalation exposure with 10 mg/m³ of glass microfibers, resin-coated glass wool, or uncoated glass wool for 50 weeks. Focal fibrosis was observed. Very low levels of fibrosis were seen in rats and hamsters exposed by nose-only inhalation to fibrous glass ranging in diameter from 0.45 μm to 6.1 μm in concentrations up to 3000 f/cc for 2 years (Smith et al. 1984, 1987, as cited in IARC 1988/Doc. H–043, p. 131). Slight septal fibrosis was observed in rats exposed to both glass microfibers (diameter <1 μm) and thicker fibrous glass (diameter <1–3 μm) at concentrations of 5 mg/m³ for up to 2 years (Le Bouffant et al. 1987, as cited in IARC 1988/Doc. H–043, p. 131).

Baboons developed focal peribronchiolar fibrosis after inhalation exposure to fibrous glass with a median diameter of 0.5–1.0 μm and a length of 6 μm in a concentration of 1,122 f/cc (7.5 mg/m³) for up to 30 months (Goldstein et al., 1983, 1984, as cited in IARC 1988/Doc. H–043, p. 131 and Vu 1988/Doc. H–043, p.54). The lesions were similar to those produced by crocidolite asbestos. Cynomolgus monkeys and Fischer 344 rats exposed to fibrous glass dust for 18 and 21 months, respectively, at concentrations of 5 or 15 mg/m³ showed no fibrosis but did have pulmonary macrophage aggregates and granulomas (indicative of a foreign body reaction) containing fibrous glass. In addition, the rats had pleural plaques (Mitchell et al., 1986, as cited in Vu 1988/Doc. H–043, p. 53). Animals exposed to fibrous glass by intratracheal instillation or intrapleural or intraperitoneal injection have also exhibited fibrotic and other responses.

The intratracheal instillation of fibrous glass has been shown to produce an inflammatory response in rats and hamsters (Gross et al., 1970 a, b, as cited in I ARC 1988/Doc. H–043, p.131; Sykes et al., 1983, as cited in IARC 1988/Doc.

H–043, p. 129) and focal areas of pneumonitis (Vorwald et al. 1951, as cited in IARC 1988/Doc. H–043, p. 131) or focal atelectasis (Schepers and Delahant 1955, as cited in IARC 1988 Doc. H–043, p. 131) in guinea pigs. Wenzel et al. (1969, as cited in IARC 1988/Doc. H–043, p. 131) demonstrated the production of pulmonary fibrosis by the intratracheal instillation of fibrous glass in rats. Kuschner and Wright (1976, as cited in NIOSH 1977/Doc. H–043, p. 48; Wright and Kuschner 1977, as cited in IARC 1988/ Doc. H–043, p. 131) treated guinea pigs with glass fibers of various dimensions by intratracheal instillation. The diameters of the fibers varied from <0.3 μm to 1.0 μm. Lengths were either short (<10 μm) or long (>10 μm). All long fibers produced interstitial fibrosis, while short fibers did not. Syrian hamsters treated by intratracheal instillation with fibrous glass of various diameters or crocidolite asbestos had developed increased collagen deposition and pulmonary fibrosis when observed at 11 months after instillation. The degree of the effect was as follows: asbestos (0.24 μm diameter) > glass microfibers (0.2 μm diameter) > commercial glass fibers (2.3 μm). No fibrosis was seen in hamsters treated with fibers having diameters of 3–4 μm (Pickrell et al. 1983, as cited in Vu 1988/ Doc. H–043, p. 55).

Smith et al. (1986) treated rats by intratracheal instillation with glass fibers (mean diameter 0.45 μm), crocidolite asbestos, or saline once a week for five weeks. The incidence of marked lung fibrosis was 32 percent for those rats treated with glass fibers, 96 percent for those treated with asbestos, and 0 percent for the saline controls. Massive fibrosis, comparable to that seen in asbestos controls, has also been produced by glass fibers given to mice by intrapleural injection (Davis 1976, as cited in Vu 1988/Doc. H–043, p. 56). Mice were treated with fibers which were either 3.5 μm or 0.05 μm in diameter and either <20 μm or 100 μm in length. Fibers of either diameter that were >100 μm in length produced massive fibrosis. Fibers of either diameter that were <20 μm in length produced discrete granulomas with minimal fibrosis. Intraperitoneal injection of long glass fibers with mean diameters of 0.05 to 0.1 μm or 2.5 to 4 μm has also been shown to cause fibrosis in mice (Davis 1972, as cited in IARC 1988/Doc. H–043, p. 132). Peritoneal fibrosis was also produced in a dose-related manner in rats treated by intraperitoneal injection with glass fibers with an average diameter of 0.5 μm with 72 percent being <5 μm in length (Pott et al. 1974, as cited in Vu 1988/Doc. H–043, p. 56). Rats injected with glass fibers with a mean diameter of 0.45 μm also demonstrated a peritoneal fibrotic response (Smith et al. 1986, as cited in Vu 1988/Doc. H–043, p. 56).

Carcinogenicity

Human Studies

A number of epidemiological studies conducted on workers exposed to fibrous glass have shown that these workers have an excess risk of lung and other respiratory tract cancers. In some cases the excess risk has been small and the studies are subject to conflicting interpretations.

Morgan et al. (1984, as cited in IARC 1988/Doc. H–043, p. 138) studied 4,399 men who had worked for at least 10 years and were employed at some time during 1968–1977 at one or more fibrous glass plants owned by a single U.S. company. The SMR for respiratory cancer for the total cohort was 136. For men with 20 years or more of employment and who were first exposed 30 years or more ago, the SMR was 177. For lung cancer, the job categories of textile forming/wool forming and fabrication had SMRs of 181 and 132, respectively.

Moulin et al. (1986, as cited in IARC 1988/Doc. H–043, p. 141) studied 1,374 men who had worked in a French fibrous glass factory for at least 1 year and who were employed there at any time during 1975–1984. The Standardized Incidence Ratio for upper respiratory and alimentary tract cancers was 218. There was a nonsignificant increasing trend in incidence ratio with increasing duration of employment. Another study (Shannon et al. 1987, as cited in IARC 1988/Doc. H–043, p. 141) examined the mortality experience of 2,557 men who had worked 90 days or more and had been employed in 1955–1977 in a Canadian glass wool plant. The SMR for lung cancer was 199 ($p<0.05$). For those workers with 5 or more years of exposure and with 10 or more years since first exposure, the SMR for lung cancer was 182. Although historical exposure data were not available, samples taken since 1978 suggested that fiber concentrations were rarely greater than 0.2 fiber/cm^3, with mean levels in most areas less than 0.1 fiber/cm^3.

Two studies, one in the United States and one in Europe, have examined the mortality experience of very large groups of workers in fibrous glass plants. Enterline et al. (1987/Doc. H–043) studied 16,661 white male workers from 17 synthetic mineral fibers plants who had 1 or more years of experience in production or maintenance during the period 1945–1963. This cohort consisted of 14,815 workers in 11 fibrous glass plants (6, glass wool only; 3, glass filament only; 2, glass wool and glass filament) and 1,846 workers in 6 mineral wool plants. The SMR for all synthetic mineral fiber workers for respiratory system cancers was 115.6 ($p<0.01$) when U.S. rates were used for comparison and 109.1 when local rates were used. For deaths that occurred during the period 1978–1982, the SMRs were 129.7 (U.S.) ($p<0.01$) and 112.7 (local). For workers with fewer than 20 years since first exposure, the SMRs for respiratory system cancer were 93.8 (U.S.) and 99.6 (local). For workers with greater than 20 years since first exposure, the SMRs were 124.3 (U.S.) ($p<0.01$) and 112.3 (local). More specifically, for workers with more than 20 years since first exposure, SMRs for the category that included bronchus, trachea, and lung cancer were 125.4 (U.S.) ($p<0.01$) and 112.7 (local) ($p<0.05$).

A significant increase in respiratory cancer compared with U.S. rates (SMR 129.5, $p<0.01$) but not compared with local rates (SMR 110.6) was also observed in workers employed for more than 20 years in the two fibrous glass plants that manufactured both glass wool and glass filament. No increase was seen in workers employed for fewer than 20 years. The increase appeared to be accounted for primarily by the increased rate (SMR 128.3, U.S., $p<0.1$) seen in one plant that employed 5,651 workers. In fact, the experience of this plant drives the results of the overall study to some extent since, of the 301 deaths from respiratory cancer 20 years or more since first exposure, 147 occurred in workers from this plant. The authors of this study believe that the use of local rates for comparison is more appropriate because they tend to adjust for social, economic, ethnic, and cultural factors. Thus they concluded that respiratory cancer rates were not significantly increased in this cohort. However, other scientists believe that the use of national rates is more appropriate because local rates are affected by local conditions, including other employment in the area. The use of local rates, therefore, requires the demonstration that some area factor and not an employment factor is influencing local rates.

A further subdivision of "time since first exposure" revealed that SMRs for fibrous glass wool workers increased with time since first exposure. Also, respiratory cancer SMRs were significantly increased overall for workers in plants that manufactured

both glass wool and glass filament (SMR 121.1, $p<0.05$, U.S.; SMR 109.7, local) For workers in these plants who had 30 years of exposure since first exposure, SMRs were 137.8 (U.S.) ($p<0.01$) and 110.8 (local). Although stating that support for the notion that exposure to synthetic mineral fibers is related to respiratory cancer comes from patterns in mortality excess as they relate to probable fiber exposure, the authors point out that several features of the data are not consistent with a causal relationship between respiratory cancer and SMF exposure. These included a lack of relationship with duration of exposure and inconsistent relationships with time since first exposure. The authors also suggest that some other contaminant in the workplace may have caused the excess respiratory cancer. However, these studies lacked data on exposure to workplace contaminants other than fibers.

In the glass wool plants, fiber concentrations to which workers were exposed were estimated to range from 0.005 fiber/cm^3 in one plant to 0.293 fiber/cm^3 in a plant that produced small-diameter fibers. The mean exposure concentration was 0.039 f/cc. The highest individual average fiber exposure level estimated for any member of this cohort was 1.5 fiber/cm^3. For the plant with the large cohort (5,651) mentioned above, the mean exposure concentration was 0.067 f/cc. The maximum concentration in that plant was 1.5 f/cc.

In an attempt to adjust for cigarette smoking, a 'case-control within a cohort' study of glass wool workers was also included. Cumulative exposure to glass wool was not a significant variable, while age at exit, year of birth, and smoking were statistically significant ($p<.05$). However, the IARC Working Group believes that the results of this case-control study may have been affected by differences in the methods of information collection used to obtain information on smoking, since smoking histories for most cases were obtained from surrogate respondents, whereas those for the majority of the controls were obtained from the respondents themselves, an approach that introduces the possibility of bias (IARC, 1988/ Doc. H–043, p. 140)

These authors also made a separate analysis of 7,586 workers in four plants that produced small-diameter fibers (<3 μm). SMRs for respiratory cancer were 132.9 (U.S.) and 123.7 (local) for those exposed and 114.7 (U.S.) and 104.8 (local) for those never exposed. Although these overall increases were not statistically significant, the SMRs were significant for deaths occurring during the period 1978–1982, with 219.4 (U.S.) ($p<0.05$) and 197.7 (local) ($p<0.05$). SMRs also increased with time since first exposure. The authors concluded that these data were consistent with the notion that work in departments that produced small diameter fibers was associated with respiratory cancer.

Marsh et al. (1990/Doc. H–043) completed a follow-up (to the year 1985) of the Enterline et al. cohort. The SMR for respiratory system cancers for all SMF workers was 120.5 ($p<0.01$) when U.S. rates were used for comparison and 112.1 ($p<0.05$) when local rates were used. The SMRs for cancers of the bronchus, lung, and trachea were 121.5 (U.S.; $p<0.01$) and 112.5 (local; $p<0.05$). For workers exposed to fibrous glass only in the two plants that manufactured both glass wool and glass filament, the SMR was 115.6 ($p<0.05$) for those with more than 20 years since first employment, as compared with local rates. (The SMRs as compared with national rates were not provided.) A further subdivision of cases by time since first exposure suggested a trend for those workers exposed to fibrous glass in "both" and combined wool and "both" plants. Also, respiratory cancer SMRs were significantly increased overall for workers in plants that manufactured both glass wool and glass filament (SMR 114.0, $p<0.05$, local) and in combined wool and "both" plants (SMR 111.8, $p<0.05$, local). For workers in "both" plants who had 30 years since first exposure, the SMR was 119.9 ($p<0.05$, local).

Simonato et al. (1987/Doc. H–043, pp. 603–623) studied 24,609 workers producing rock wool/slag wool, glass wool, or glass filament in 13 European factories in seven countries. Four of these factories produced glass wool. Among glass wool workers, the SMRs for lung cancer were 127 ($p<0.05$) (compared with national rates) and 103 (local). Workers whose time since first exposure was greater than 30 years had an SMR of 173 ($p<0.05$). There was an increasing trend in SMRs for lung cancer with time since first exposure, but this trend was not statistically significant.

The average fiber concentration in these plants, on the basis of measurements taken in 1977–1980, was <0.1 fiber/ml (range, 0.01 to 1.00 fiber/ml). The highest concentrations were associated with the manufacture of special fine fiber earplugs. In the main production and secondary production groups, the concentrations ranged from 0.01 to 0.16 f/cc (Cherrie et al., 1986/Doc. H–043, Ex. 1–4, pp. 18–25). Although no historical exposure data exist, Cherrie et al. estimated that early technological phase production techniques probably produced airborne fiber levels similar to those in the late phase measured. The average plant median for fiber length ranged from 8 to 15 μm, and the corresponding median diameters ranged from 0.7 to 1 μm.

Animal Studies

Numerous studies have been done using several species of animals and with fibrous glass administered by several routes. The inhalation route of exposure has been used in five experiments with rats and one experiment each with hamsters and baboons. In all of these studies there was no statistically significant increase in the incidence of lung or pleural tumors. In most experiments with rats, however, a few respiratory tract tumors occurred. In all of these inhalation experiments, one of the intended positive controls, crocidolite asbestos, also produced no statistically significant increased incidence of lung tumors. It is thought that rodent inhalation tests may be less sensitive than tests by other routes for evaluating the carcinogenicity of fibers because the qualitative and quantitative aspects of fiber deposition and retention are substantially different from those in humans. Fibers relevant for human disease may never reach the target organ in sufficient quantities. Other routes of exposure, such as intratracheal instillation and intrapleural and intraperitoneal administration, bring fibers into direct contact with the same target tissues as would occur in humans (IARC 1989/Doc. H–043, p. 34).

In two experiments with rats and one experiment with hamsters, animals were treated with glass wool (mean fiber diameter <0.3 μm) by repeated intratracheal instillation. In one rat experiment, a statistically significant increase in the incidence of lung tumors was observed (Pott et al. 1987, as cited in IARC 1988/Doc. H–043, p. 91). In the other rat experiment, no respiratory tract tumors were observed; however, the tumor response to the positive control, crocidolite, was also low in this study (Smith et al. 1987, as cited in IARC 1988/Doc. H–043, p. 91). In the experiment with hamsters, however, both lung tumors and mesotheliomas were observed (Pott et al. 1984a, as cited in IARC 1988/Doc. H–043, p. 91).

There have been five studies with rats and one study with mice in which the route of administration was intrapleural implantation or injection. Pleural tumors were found in four of the rat studies.

The tumor incidence varied with the fiber size (Wagner et al. 1973, 1976, 1984, as cited in IARC 1988/Doc. H–043, p. 93; Monchaux et al. 1981, as cited in IARC 1988/Doc. H–043, p. 93; Stanton et al. 1977, 1981, as cited in IARC 1988/Doc. H–043, p.93). No tumors were observed in mice, but there was a relatively short observation time and a low response in positive controls (Davis 1976, as cited in IARC 1988/Doc. H–043, p. 92). Stanton et al. (1977, 1981, as cited in IARC 1988/Doc. H–043, p. 93) treated rats with a single implantation of one of 72 different types of synthetic and natural fibers (including asbestos). Nineteen of the fibers were fibrous glass. The investigators found that incidences of pleural mesothelioma ranged from 0/28 to 20/29 and correlated with fiber size rather than physicochemical properties. They observed that the most carcinogenic fibers were those <1.5 μm in diameter and >8 μm in length. Thus, these studies demonstrated that glass fibers with length and diameter distributions comparable to those of asbestos were as carcinogenic as asbestos by intrapleural implantation.

Injection of glass wool into the peritoneal cavity of rats produced mesotheliomas or sarcomas in all eight studies with rats (Pott et al. 1976, 1984b, 1987, as cited in IARC 1988/Doc. H–043, pp. 95–96; Muhle et al. 1987, as cited in IARC 1988/Doc. H–043, p. 96; Smith et al. 1987, as cited in IARC 1988/Doc. H–043, p. 97).

Refractory Ceramic Fibers

Nonmalignant Respiratory Disease

Human Studies

RCF production workers are the subject of an ongoing epidemiological study being conducted by the University of Cincinnati. In this longitudinal morbidity study, workers at five RCF manufacturing facilities are being evaluated. Interim results of this study (TIMA, 1990a/Doc. H–043) showed RCF exposure TWAs that ranged from <0.01 to 1.57 f/cc. Approximately 30 percent of the RCF exposed workers had between 10 and 20 years of exposure, and 4 percent had greater than 20 years of exposure. With regard to morbidity, the prevalence of respiratory symptoms was increased in production workers, but this increase was not statistically significant. The duration of RCF exposure was related to pleuritic chest pain. Pulmonary function was decreased, and this decrease was significantly associated with duration of exposure. Chest radiographs revealed that the prevalence of pleural plaques overall in RCF production workers was 2.4 percent, while the prevalence in non-production workers was 0 percent. Pleural plaques were seen in 18.2 percent of the workers with greater than 20 years' of RCF exposure in a production job. A recent study of asbestos workers demonstrated that those with pleural plaques had significantly higher death rates from lung cancer, mesothelioma, and asbestosis than those without pleural plaques. It did not appear, however, that the plaques became pleural mesothelioma, nor did lung cancer occur preferentially in the regions of the plaques (Selikoff et al., 1990, Doc. H–033e, Ex. 7–34).

Exposure data provided by the Thermal Insulation Manufacturers Association (TIMA) (1990, as cited in EPA, 1990/Doc. H–043) show airborne concentrations that range from 0.01 to 6.4 f/cc (average 0.62 f/cc) for manufacture, 0.02 to 56 f/cc (average <1 f/cc for most operations; 4–8 f/cc for vacuum-formed shapes manufacture and fabrication), and 0.01 to 24.72 f/cc (average 1.24 f/cc) for end use. Recent results of a study in progress at Johns Hopkins University directed by Morton Corn (Corn et al., 1990/Doc. H–043) show that workers in blanket fabrication are exposed to RCF concentrations in the range of 0.11 to 1.91 f/cc (mean 0.61 f/cc, maximum individual sample 8.38 f/cc) and in blanket installation in the range of 0.08 to 8.91 f/cc (mean 1.84 f/cc, maximum individual sample 22.9 f/cc). Those in bulk fiber fabrication are exposed to concentrations in the range of 0.05 to 1.83 f/cc (mean 0.57 f/cc, maximum individual sample 1.67 f/cc) and in bulk installation in the range 1.50 to 7.72 f/cc (mean 4.61 f/cc, maximum individual sample 12.14 f/cc). Those in vacuum-formed products fabrication are exposed to concentrations in the range of 0.08 to 34.0 f/cc (mean 3.58 f/cc, maximum individual sample 143 f/cc) and in installation to concentrations in the range 2.03 to 6.58 f/cc (mean 5.31 f/cc, maximum individual sample 11.8 f/cc).

Animal Studies

In animal studies, exposure to RCF by several routes has been shown to cause nonmalignant respiratory effects. Alveolar lipoproteinosis and significant levels of pulmonary fibrosis developed in rats exposed for one year to 10 mg/m³ respirable ceramic fiber dust with 90 percent of the fibers having a diameter of <3 μm (Davis et al. 1984, as cited in IARC 1988/Doc. H–043, pp. 130 and 131). In another study, rats and hamsters exposed by inhalation to 0.5–10 mg/m³ RCF developed low levels of pulmonary fibrosis (Smith et al. 1987, as cited in IARC 1988/Doc. H–043, p. 131). Hamsters, guinea pigs, and especially rats developed pulmonary fibrosis after a 3-month inhalation exposure to between 3000 and 40,000 f/cc of potassium octatintanate RCF, 3–15 μm in length (Lee et al. 1981; Lee and Reinhardt 1984, as cited in IARC 1988/Doc. H–043, p. 131). Gross et al. (1976b, as cited in IARC 1988/Doc. H–043, p. 131) found that intratracheal instillation of RCF in rats at a dose of 10.5 mg produced dust deposits that were surrounded by inflammatory cells.

Carcinogenicity

Human Studies

No epidemiological studies have been done to examine the risk of respiratory tract cancer or mesothelioma in workers exposed to RCF. However, as stated above, a very small percentage of RCF workers has a 20-year or longer time period since first exposure. Studies of asbestos workers have demonstrated that the latency period (the time between exposure to a substance and the onset of disease) for mesotheliomas can be as long as 45 years and is routinely between 20 and 40 years (Maltoni et al., 1991/Doc. H–033e, Ex. 124). Thus, it may be too early to be able to detect chronic diseases such as respiratory tract cancer and mesothelioma in RCF workers.

Animal Studies

Refractory ceramic fibers have also been shown to produce cancer in experimental animals treated by several routes of administration. Inhalation exposure to RCF has been shown to cause tumors in several studies. Lee et al. (1981, as cited in IARC 1988/Doc. H–043, p. 101) exposed rats and hamsters to 2900 f/cc potassium octatintanate fibers (19.1 percent <3 μm diameter) or 3100 f/cc amosite asbestos for 6 hours per day/5 days per week for 3 months. The animals were then observed for 21 months. At the end of the study, bronchoalveolar tumors were observed in 1/14 rats treated with potassium octatintanate, 3/11 treated with amosite asbestos, and 0/13 in untreated controls. One of the four potassium octatintanate-treated hamsters sacrificed at 18 months had a pleural mesothelioma. No other animals in any hamster treatment group had tumors.

Davis et al. (1984, as cited in IARC 1988/Doc. H–043, p. 101) exposed rats by inhalation to 10 mg/m³ respirable dust from fibrous ceramic aluminum silicate glass (90 percent of fibers <3 μm length, <0.3 μm diameter, aspect ratio $>3:1$) for 7 hours per day/5 days per week for 12 months. Malignant pulmonary

neoplasms were observed in 7/48 animals. There was also one benign adenoma. No pulmonary tumors were observed in 39 untreated controls. In another study, hamsters were exposed by nose-only inhalation to RCF dust at a concentration of 10.8 mg/m³ (35 percent respirable, geometric mean diameter 0.9 μm, geometric mean length 25 μm) for 6 hours per day/5 days per week for 2 years and observed for life. The exposure concentration was 200 f/cc, with 88 f/cc >10 μm in length and <1.0 μm in diameter. A spindle cell mesothelioma of the lung developed in one of the 58 treated hamsters (Smith et al. 1987, as cited in IARC 1988/Doc. H–043, p. 102).

The Thermal Insulation Manufacturers Association (TIMA, 1990/Doc. H–043) has reported the results of a 2-year study in which hamsters and rats were exposed to RCF by nose-only inhalation at a concentration of 250 f/cc (>15 μm in length) for 6 hours per day, 5 days per week. A unique aerosol generation procedure was used in which large concentrations of unbroken fibers with little fiber dust could be produced. This procedure allowed for much greater respirability of fibers than had been achieved in previous fiber inhalation studies. Rats were treated with four types of RCF: Kaolin, zirconia, high purity, and "after service." Hamsters were treated with kaolin only. The study also included positive control groups treated with chrysotile asbestos (approximately 5000 f/cc, <5 μm in length) and negative controls treated with filtered air. At the 18-month final sacrifice, 35 percent (36/102) of the hamsters had pleural mesotheliomas. Fibrosis was also present. In contrast, the positive control chrysotile asbestos-exposed hamsters had only fibrosis, with one hamster showing a non-malignant pleural mesothelial growth. No lesions were seen in negative controls. By the 24-month interim sacrifice for the rats, pathological studies had shown two benign lung tumors, seven lung carcinomas, and three mesotheliomas in RCF-exposed animals. Chrysotile asbestos controls had a thymic adenoma and one lung carcinoma.

Mesotheliomas have also been produced by the intrapleural administration of RCF to rats. In one study, mesotheliomas developed in 3 of 31 rats that received a single intrapleural injection of 20 mg ceramic aluminum silicate fibers (0.5–1.0 μm in diameter). One mesothelioma developed in a similar number of rats treated with nonfibrous aluminum oxide. Rats treated with chrysotile asbestos had tumor incidences of 23/36 and 21/32 (Wagner et al. 1973, as cited in IARC 1988/Doc. H–043, p. 103). Stanton et al. (1981, as cited in IARC 1988/Doc. H–043, p. 104) treated rats with a single intrathoracic implantation of one of 13 different types of ceramic fibers. The incidences of pleural sarcomas were correlated with the number of fibers <0.25 μm in diameter and >8 μm in length and ranged from 1/45 to 21/29 in exposed animals.

The intraperitoneal (i.p.) administration of RCF has been shown to produce tumors in three studies with rats and one study with hamsters. In one study, 9 percent of rats injected with 25 mg fibrous ceramic aluminum silicate glass (90 percent fibers <3 μm in length and <0.3 μm in diameter) developed peritoneal tumors (one mesothelioma and two tumors similar to fibrosarcomas). Five percent of a group of untreated controls used for an inhalation study developed malignant tumors of the peritoneum or digestive system (Davis et al. 1984, as cited in IARC 1988/Doc. H–043, p. 105). In another study, tumors of the abdominal cavity were found in 32/47 and 12/54 rats that had received five i.p injections of two types of ceramic wool (median length, 8.3 μm; diameter 0.91 μm and median length 6.9 μm; diameter 1.1 μm). Injections of titanium dioxide caused tumors in 5/53 rats, and saline controls had a tumor incidence of 2/102. Positive control groups receiving actinolite or chrysotile had tumor incidences of 20/36 and 31/36 rats, respectively (Pott et al. 1987, as cited in IARC 1988/Doc. H–043, p. 105). Smith et al. (1987, as cited in IARC 1988/Doc. H–043, p. 105) treated rats and hamsters with a single i.p. injection of 25 mg RCF (geometric mean length, 25 μm; geometric mean diameter 0.9 μm; 83 percent fibers >10 μm length and 86 percent <2.0 μm diameter. Mesotheliomas were found in 19/23 of the treated rat group, in 2/15 and 5/21 of the treated hamster groups, and in none of the saline-treated or untreated controls.

Discussion

Based on what it viewed as the lack of reported adverse health effects in epidemiologic studies, the ACGIH (1986/Doc. H–020, Ex. 1–3) considered fibrous glass dust to be essentially a nuisance dust and applied a 10 mg/m³ TLV®–TWA.

In contrast, NIOSH (in 1977/Doc. H–043) concluded that "available data are sufficient to demonstrate that fibrous glass does not act like an inert or nuisance dust because it can produce fibrosis in animals and respiratory tract irritation in humans." Relying on several investigations that showed a lack of adverse effects among workers exposed to mean respirable fiber concentrations that were generally less than 5 to 6 f/cc, NIOSH recommended that exposures be limited to 3 f/cc for fibers >3.5 μm and >10 μm in length.

Recently, however, citing animal and epidemiological studies published since the NIOSH 1977 Criteria Document, NIOSH (1988, Doc. H–020, Ex. 8–47) has commented that these studies indicate the potential carcinogenicity of fibrous glass. NIOSH (1988/Doc. H–020, Ex. 8–47) commented, "It is likely * * * that even the 3 f/cc standard will not provide the degree of protection that OSHA believes is necessary for worker health and that reduction of the PEL to 0.2 f/cc * * * will be necessary to protect workers from the development of lung cancer." NIOSH (1988/Doc. H–020, Ex. 8–47, Table N6B) also believes that a full 6(b) rulemaking is needed for fibrous glass. NIOSH based its comments on the conclusion reached by Doll (1987/Doc. H–043, Ex. 1–11) to the effect that "* * * it is likely that man-made mineral fiber may have about the same carcinogenic potential as asbestos fibers of the same dimension, and that levels of 0.2 f/cc or less in industry are unlikely to produce a measurable risk after 20 years of exposure."

The Thermal Insulation Manufacturers Association (TIMA (1990b/Doc. H–043) has commented that it supports the application of NIOSH's REL of 3 f/cc for fibrous glass to all synthetic mineral fibers but does not believe that fibrous glass poses a human cancer hazard. Recently, however, TIMA (1991) has recommended that OSHA establish a PEL of 1 f/cc (8-hour TWA, NIOSH 7400B method) for fiber glass. According to TIMA, this recommendation is based on grounds of prudence and not significant risk. TIMA additionally recommended that fiber glass be classified as an irritant. Manville (1990/Doc. H–043), referring to fibrous glass, has stated that "Where industrial hygiene measurement is available on a continuous basis, a Manville workplace exposure guideline of 1 f/cc is suggested as a point above which respiratory protection should be used." Manville also believes that fibrous glass does not pose a human cancer hazard. The Carborundum Company (Doc. H–043) has a Recommended Exposure Guideline of 2.0 f/cc for worker exposure to RCF. Respiratory protection is recommended at and above this level. The refractory ceramic fiber subcommittee of TIMA has recently recommended an exposure

guideline of 1 f/cc, with workplace controls as appropriate (Given 1991/ Doc. H–043). E.I. du Pont de Nemours and Company has an Acceptable Exposure Level of 0.5 f/cc for RCF and 2.0 for fibrous glass (personal communication from Frieda Fisher-Tyler, 1991).

The Building and Construction Trades Department of the American Federation of Labor-Congress of Industrial Organizations (A.F.L.-C.I.O., 1991) has recommended that OSHA establish a PEL of 1.0 f/cc as a TWA for the respirable fibers of fiber glass. The authors stated that the recommendation was consistent with the current state of their understanding of health risks, including the suggestive but not conclusive indication that these fibers may possess the ability to produce respiratory system cancer in exposed workers. A PEL of 0.1 f/cc was recommended for respirable refractory ceramic fibers based on the fact that inhalation studies with experimental animals have demonstrated that the carcinogenic potential of these fibers is similar to that of asbestos.

The International Agency for Research on Cancer (IARC 1988/Doc. H–043), in evaluating the carcinogenicity of fibrous glass and RCF, has concluded that glass wool and RCF are "possibly carcinogenic to humans (Group 2B)."

Current exposure limits (8-hour TWA) for all synthetic mineral fibers (unless specified) in several other countries that have recently studied the issue are as follows: Australia, 0.5 f/cc; Denmark, 2.0 f/cc; Great Britain, 1.0 f/cc; The Netherlands, 5.0 f/cc for glass wool and glass microfibers and 1.0 f/cc for refractory ceramic fibers; New Zealand, 1.0 f/cc; Norway, 1.0 f/cc; Poland, 2.0 f/cc, Sweden, 1.0 f/cc.

OSHA believes that a fiber-based standard is necessary. Industry representatives (Doc. H–020, Exs. 3–743, 8–56, 132, 175; Doc H–043) have argued strongly for a fiber-based standard, and NIOSH has recommended fiber-based counting procedures since 1977. TIMA points out that, for example, a 5 mg/m^3 PEL for fibrous glass dust would permit a concentration of 4,000 f/cc for fibers 10 microns long and 0.25 microns in diameter (Doc. H–020, Ex. 3–743, p. 8).

In summary, OSHA has evaluated the available evidence on the health effects of fibrous glass, including refractory ceramic fibers. It has presented the strengths and weaknesses of the studies and discussed the possibility of confounding factors. Some of the human studies do not have contemporaneous exposure data; this means that, although current exposures are often quite low, it may be the case that earlier exposures were higher. OSHA's discussion also presents the views of other organizations.

A statistically significant increase in mortality from nonmalignant respiratory disease has been observed in several studies of fibrous glass workers, including Bayliss et al. (1976) and Enterline et al. (1986, 1987). An ongoing morbidity study of RCF workers has demonstrated an increase in the prevalence of respiratory symptoms, pleuritic chest pain, and pleural plaques and a decrease in pulmonary function that was significantly associated with duration of exposures. In each of these studies, workers were exposed to fiber concentrations in the range of OSHA's proposed PEL.

OSHA believes, on the basis both of animal and human evidence, that a link between respiratory disease and exposure to fibrous glass exists. These respiratory effects constitute a material impairment of health. Based on this evidence, OSHA is proposing a 1 f/cc 8-hour TWA limit for the respirable fibers of fibrous glass, including refractory ceramic fibers. OSHA preliminarily concludes that this limit will substantially reduce the significant risk of nonmalignant respiratory disease that exists in the absence of a limit for workers in all sectors.

OSHA has presented the evidence of possible carcinogenicity of fibrous glass, including refractory ceramic fibers. There is also a substantial body of ongoing research. At this time OSHA believes it is premature for the Agency to reach a final decision on an exposure limit based on carcinogenicity. However, the proposed limit will also clearly increase the protection of workers from this effect as well.

OSHA is proposing the use of NIOSH counting method number 7400B and soliciting information on alternative counting methods.

OSHA believes this proposal is appropriate based on its review of the studies and their strengths and weaknesses, the recommendations of expert organizations, and the various ongoing studies that may provide important new data.

GRAIN DUST (OATS, WHEAT, AND BARLEY)

CAS: None; Chemical Formula: None
H.S. No.: 1190

A decision by the Occupational Safety and Health Review Commission (*Secretary of Labor* v. *Krause Milling Company*, OSHRC Docket No. 78–2307, April 22, 1986) has held that there currently is no OSHA PEL for grain dust in the construction and maritime industries. There is also no limit in agriculture. The ACGIH has a 10 mg/m^3 TLV®–TWA for this substance. There is no NIOSH REL; however, NIOSH concurs (Ex 8–47, Table N1) with the limit OSHA is proposing in construction, maritime, and agriculture, which is a 10 mg/m^3 8-hour TWA PEL for grain dust generated from wheat, oats, and barley. Promulgation of this limit will make the PEL for grain dust consistent across all OSHA-regulated sectors.

Grain dust is a complex mixture of husk particles, cellulose hairs and spikes, starch granules, spores of fungi, insect debris, pollens, rat hair, and approximately 5 percent mineral particles. The mean particle size of the airborne dusts may be less than 5 μm. Grain is exported, milled into flour, malted by the brewing industry, distributed as a feedstuff for animals or returned to the land as seed (Rom 1983, p. 221).

The acute grain dust toxicity data in animals are sparse. However, in-vitro studies have demonstrated that grain dust can stimulate lymphocyte proliferation and the production of interleukin-1 by rat alveolar macrophages (Ye, Lewis, Sorenson, and Olenchock 1988). Another study (Alam, Lewis, and Olenchock 1988) has shown that grain dust can stimulate lymphocytes, mast cells, and basophils, i.e., those cells involved in producing inflammatory responses, in cultured guinea pig spleen and lung tissue.

In humans, exposure to grain dust causes both acute and chronic effects. The adverse effects of inhaling grain dust have been known for at least two-and-one-half centuries, dating back to Rammazini who, in 1713, described the respiratory hazards associated with exposure to cereal grain dust. More recently, several epidemiological studies conducted over the past few decades have demonstrated that exposure to grain dust causes "grain fever," wheezing, chest tightness, productive cough, eye and nasal irritation, and symptoms of chronic respiratory disease. Grain dust may also induce asthmatic reactions via an allergic mechanism, particularly in individuals who are predisposed to developing allergies (i.e., atopic individuals).

Epidemiological studies have shown that as many as 25 percent of grain elevator workers experience grain fever (Broder et al. 1979; Chan-Yeung et al. 1980; doPico et al. 1980; Tse et al. 1973). Increased rates of cough, shortness of breath, and wheezing were reported in 23 percent of English grain farmers (N=65) (Darke et al. 1976). Seventy-six percent of dock workers handling grain

were found to have chest tightness or grain fever (Lockcroft et al. 1983).

The basis for OSHA's proposed 10-mg/m³ limit in the construction, maritime, and agriculture sectors is a NIOSH-sponsored study of grain workers by Rankin et al. (*Study of the Prevalence of Chronic, Non-Specific Lung Disease and Related Health Problems in the Grain Handling Industry*, DHHS (NIOSH) Pub. No. 86-117, 1986). This study evaluated the health status of 310 grain handlers in Wisconsin and Minnesota. The grain handlers were selected from employees at eight elevator companies, from state grain inspection agencies, and from longshoring companies. Health status was determined by questionnaire and by physical examination, which included an assessment of pulmonary function, immunologic evaluation, blood and urine chemistries, and chest roentgenograms. The comparison group that served as controls consisted of 239 city workers who spent the majority of their workdays outside.

From the questionnaires, Rankin et al. (1986) found that the grain handlers had a higher prevalence of respiratory symptoms than did the city workers. The prevalence of respiratory symptoms was highly significant (Rankin et al. 1986, Table 13), and was independent of smoking status. The symptoms reported by grain handlers represented both acute and chronic airways reactions (occupational asthma and chronic bronchitis). Wheezing and/or chest tightness generally started within two hours of beginning the work shift. Episodes of grain fever occurred infrequently; this was attributed by the workers to improved working conditions over the previous three years. However, acute recurrent conjunctivitis and rhinitis were reported to occur among most grain workers.

Lung function tests showed that exposure to grain dust had a highly significant adverse effect on pulmonary function (Rankin et al. 1986, Table 30). There was, however, no correlation between reduced pulmonary function and job category, length of employment, or place of work. The lung function decrement observed among grain handlers was not related to smoking history alone; grain handlers who were smokers or ex-smokers showed significant declines in pulmonary function when compared to city workers who were smokers or ex-smokers.

Grain workers who reported symptoms had lower values of ventilatory function than did workers without symptoms. The prevalence of chronic bronchitis symptoms with measured airway obstruction was higher in grain workers than in controls, regardless of smoking history. Chronic bronchitis with airway obstruction was also related to length of employment. Rankin et al. (1986) concluded that these findings "suggest that chronic grain dust exposure may result in chronic obstructive pulmonary disease" (p. 26).

Rankin et al.'s (1986) study also included a work-shift study in which 248 grain workers and 192 city workers were sampled for grain dust exposure during a work shift. Symptoms occurring during the shift were recorded and pulmonary function readings were taken before and after the shift. Only 14 percent of grain workers were exposed to an 8-hour TWA level exceeding 5 mg/m³ total grain dust; 7 percent were exposed above 10 mg/m³. Rankin et al. (1986) reported that grain workers showed a significant excess of cough and expectoration during work shifts in which dust concentrations were below 5 mg/m³. At dust levels between 10 and 15 mg/m³, there was a significantly increased prevalence of wheezing and dyspnea during the shift among grain workers as compared with controls (Rankin et al. 1986, Table II-156). Workers with pre-existing airway obstruction experienced significant pre- to post-shift declines in ventilatory function at dust levels below 10-mg/m³. However, the changes observed in pre- to post-shift pulmonary function did not correlate with the presence of symptoms during the shift.

Rankin et al. (1986) also conducted a short-term (three-year) follow-up study of lung function among grain workers. Their results showed no greater declines in FEV or FVC over the three-year period than could be accounted for by age alone. However, there was a significant decline in other measures of lung function (MMF, V_{max50}, V_{max75}) among both smoking and nonsmoking grain workers. The authors concluded that, although a grain-dust-related decline in these measures was observed, the long-term effects of smoking on lung function were probably greater than those caused by grain dust.

The findings of the Rankin et al. (1986) study are consistent with those of other recent published studies (NIOSH 1988; Tse 1990; Hurst and Dosman 1990; Richerson 1990; Revsbeck and Andersen 1989) of grain workers employed in establishments in various sectors. For example, Dr. Roy Buchan, Chief of the Occupational Health and Safety Section, College of Veterinary Medicine and Biomedical Sciences at Colorado State University, performed a study of the general health of 31 grain handlers (submitted as part of Ex. 3-751). A total of 204 personal TWA dust samples were taken, of which only six exceeded 10 mg/m³. Dr. Buchan found that neither age of facility, smoking history, nor past exposure to grain dust had any significant effect on symptom responses. There was a statistically significant association between grain dust exposure levels and symptom responses. The reported symptoms included nasal and throat irritation, chest discomfort, and phlegm production. Dr. Buchan concluded that, "although the association was mathematically weak but statistically significant, it would rationally be expected that symptom severity would become more pronounced as dust concentrations increase, since dust exposures in this investigation were surprisingly low (mean=0.7 mg/m³ TWA)." In a larger study of 390 Canadian grain workers, Cotton, Graham, Li et al. (1983, submitted as part of Ex. 3-751) also reported a significant excess incidence of respiratory symptoms among grain workers despite total dust concentrations generally below 10 mg/m³.

Although these studies show a pattern of increased prevalence of respiratory symptoms among grain handlers exposed below 10 mg/m³, the association between low-level exposure to grain dust and the development of chronic pulmonary disease remains open to interpretation. Several studies, including those of Rankin et al. (1986), Chan-Yeung, Giclas, and Henson (1980/Ex. 1-474), and Broder, Corey, Davies et al. (1985, as cited in Ex. 3-751) have generally not found decrements in pulmonary function associated with long-term exposure to grain dust. In addition, chest roentgenograms have found no evidence of lung scarring or fibrosis (Rankin et al. 1986) among grain handlers. However, symptoms of chronic bronchitis have frequently been noted among grain handlers, including those who have never smoked (Rankin et al. 1986; Cotton, Graham, Li et al. 1983). According to Cotton et al. (1983, as cited in Ex. 3-751, p. 139), "The significance of the increase in chronic bronchitis and cough in workers and wheezing in nonsmoking workers in terms of eventual respiratory disability remains uncertain but the nuisance and discomfort of these symptoms for workers must also be considered."

In the prior rulemaking in general industry, some commenters (Exs. 8-55, 180, and 185) took the position that grain dust is a nuisance dust. OSHA does not concur with this view. In the studies described above, as well as in others in the record, grain workers in all regulated sectors have consistently reported an

excess prevalence of respiratory symptoms, including chronic bronchitis, at low levels of exposure to grain dust. OSHA believes that these symptoms, even in the absence of definitive evidence of irreversible lung damage, constitute material impairment of health and interfere with the well-being of workers. In the prior rulemaking, this was attested to at the informal hearing by Deborah Berkowitz, Director of Safety and Health for the Food and Allied Trades Department, AFL–CIO:

> I want to make it clear that study after study documents a very real acute hazard to grain workers. Living with chronic bronchitis is not a hazard that should go unchecked. In fact, study after study point to the possibility of very real long-term damage from chronic cumulative effects of exposure to grain dust. But even without the possibility of long-term disability, acute hazards clearly pose significant risk[s] to workers (Tr. pp. 6–306 to 6–307).

Thus OSHA preliminarily concludes that employees in construction, maritime, and agriculture are at significant risk of developing respiratory symptoms, including chronic bronchitis, as a result of their exposure to grain dust. It is clear that such symptoms occur at grain dust levels exceeding OSHA's current limit in construction and maritime for dusts and particulates (15 mg/m³ TWA). Increases in respiratory symptoms have also been reported to occur among grain workers exposed generally to concentrations of less than 10 mg/m³, although symptoms are diminished in intensity at these lower levels. At this time, it is difficult to identify the precise threshold at which adverse respiratory effects are likely to occur.

Based on this evidence in workers, OSHA is proposing an 8-hour TWA limit of 10 mg/m³ in construction, maritime, and agricultural operations for grain dust, measured as total dust. Grain dusts other than oats, wheat, and barley will continue to be covered under OSHA's generic "particulates not otherwise regulated" PEL of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction). Promulgation of this limit for grain dust will make OSHA's PEL for this substance consistent across all regulated sectors.

GRAPHITE, NATURAL

CAS: 7782–42–5; Chemical Formula: None

H.S. No. 1191

The current OSHA limit for natural graphite (total dust) in the construction and maritime industries is 15 million particles per cubic foot (mppcf), which is equivalent to 2.5 mg/m³ as respirable dust (assuming that respirable mass is one-half total particle mass). There is no limit for natural graphite in agriculture. The ACGIH has an 8-hour TLV®–TWA of 2.5 mg/m³ for graphite (respirable dust). NIOSH has no REL for this substance. In construction and maritime, OSHA is retaining the 8-hour TWA PEL of 2.5 mg/m³ for respirable natural graphite dust containing less than 1 percent quartz but changing the units in which the limit is expressed from mppcf to mg/m³. OSHA is also proposing an 8-hour TWA PEL of 2.5 mg/m³ in agriculture. NIOSH concurred (Ex. 8–47, Table N1) with this limit when OSHA recently established it in general industry.

Natural graphite, also called plumbago and mineral carbon, is a relatively soft, greasy-feeling, steel gray to black solid with a metallic sheen. In nature, graphite is usually associated with impurities, such as silica, mica, granite, etc. Graphite is used in "lead" pencils, refractory crucibles, foundry facings, stove polishes, pigments, lubricants, cements, matches, and explosives (Hawley's 1987, p. 576; Merck 1983, p. 652; ACGIH 1986, p. 290(89)).

There are few data on the toxicity of graphite in experimental animals. Administered to rats by intratracheal injection, graphite with a "low ash content" caused fibrosis of the lung; however, the amount of silica or other impurities in the graphite was not determined (Ray, King, and Harrison 1951/Ex. 1–46). A recent study (Thomson, Bergmann, Burnett, Carpin, and Crouse 1988) shows that exposure to natural graphite, which contains silicate minerals, and to synthetic graphite, which contains less than 1 percent silica, both elicit physiologic changes in the lungs of rats exposed to 100 mg/m³ of each substance for 4 hours/day for 4 days.

Early reports established that graphite deposited in the lungs of occupationally exposed workers caused pneumoconiosis (Koopman 1924/Ex. 1–131). Subsequent research described the condition produced by exposure to graphite as anthracosilicosis, a pulmonary condition similar to that seen in coal miners, based on radiographic and histologic examinations in exposed individuals (Harding and Oliver 1949/Ex. 1–71). Radiologic changes were also observed among graphite mine and production workers exposed to graphite containing from 3.6 to 10 percent silica (Pendergrass, Vorwald, Mishkin et al. 1967/Ex. 1–77). A recent study of the incidence of graphite-induced pneumoconiosis in Sri Lankan graphite miners shows that the incidence was substantially lower in workers from a mine equipped with dust controls (3.4 percent) compared with that in workers from a mine without such controls (18.3 percent) (Uragoda 1989). A 1983 review article (Hanoa 1983) concludes that, because information reported in the literature is often unclear both about the composition of the natural graphite and the extent of occupational exposure, it is difficult to distinguish the extent of pure graphite's contribution to the development of graphite pneumoconiosis. The author concluded, on the basis of a review of 18 epidemiologic studies of graphite-exposed workers, that the possibility that pure graphite causes pneumoconiosis cannot be ruled out, although most of these studies suggest that pneumoconiosis is a mixed-dust reaction (Hanoa 1983).

OSHA is proposing to revise its former limit of 15 mppcf to the equivalent limit of 2.5 mg/m³ for the respirable fraction of graphite containing less than 1 percent quartz for the construction and maritime industries and to extend this limit to agriculture. In construction and maritime, this change represents a change only in the units used to express or measure the limit, not a change in the value of the limit. OSHA is revising its limit to simplify the monitoring of employee exposures because the use of impingers and microscopic analyses are not required to measure exposures that are expressed in mg/m³ rather than in mppcf. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

INDIUM AND COMPOUNDS

CAS: 7440–74–6; Chemical Formula: In

H.S. No. 1213

OSHA's exposure limit for indium and compounds in the construction and maritime industries is 0.1 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.1 mg/m³ for these substances; NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. The Agency is retaining its limit for construction and maritime operations and is proposing to extend this 0.1 mg/m³ 8-hour TWA limit to agricultural operations. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Indium metal is silver-white, shiny, and ductile. Indium is used in bearings for automobiles and aircraft, in electronic devices, low melting alloys, and reactor control rods. Indium compounds are not widely used; however, indium antimonide, indium arsenide, and indium phosphide are used in semiconductor devices.

There are few acute toxicity data for indium and its compounds. The lowest lethal dose of indium in mice by subcutaneous injection is 10 mg/kg (RTECS 1990). The oral LD_{50} for indium in rats is 4200 mg/kg (ITI 1986, p. 620). Rats inhaling the sesquioxide form of indium at airborne concentrations ranging from 24 to 97 mg/m³ daily for a total of 224 hours developed widespread alveolar edema; these histologic lesions did not change over a 12-week post-exposure period (Leach, Scott, Armstrong et al. 1961). Animals exposed to indium show reduced alveolar clearance and may develop chronic respiratory insufficiency and recurrent acute pneumonitis before death (Jones 1960). Animals chronically poisoned with indium show weight loss, blood changes, signs of pulmonary edema, and, at autopsy, damage to the liver and kidneys (McCord, Meeks, Harrold, and Henssner 1942). Several studies have shown indium to be teratogenic in experimental animals (Farm 1972; Cralley and Cralley 1985).

In humans, exposure to indium and its compounds causes eye, mucous membrane, and skin irritation (Hazardous Substance Fact Sheet 1986). No other information on indium's toxicity in humans is available.

Based on this evidence in humans and animals, OSHA believes that exposed workers in construction, maritime, and agriculture are at significant risk of developing sensory irritation and chronic lung function impairment, both adverse health effects associated with exposure to indium. To substantially reduce the risk of these material health impairments, the Agency is retaining its 8-hour TWA limit of 0.1 mg/m³ for indium and its compounds in the construction and maritime industries and is proposing to extend this limit to agriculture. Promulgation of this limit will make OSHA's PEL for this substance consistent across all OSHA-regulated sectors.

IRON OXIDE (DUST AND FUME)
CAS: 1309–37–1; Chemical Formula: Fe_2O_3
H.S. No. 1215

In the construction and maritime industries, OSHA has an 8-hour TWA limit of 10 mg/m³ for iron oxide fume. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 5 mg/m³, measured as iron, total particulate. NIOSH has no REL for this substance. OSHA is retaining its 8-hour TWA PEL of 10 mg/m³ for the construction and maritime industries and is proposing to extend this limit to agriculture. NIOSH concurred (Ex. 8–47, Table N1) that this limit was appropriate when OSHA recently established it in general industry.

The fume of iron oxide is red-brown in color. Iron oxide is used as a pigment for rubber, paints, paper, linoleum, ceramics, and glass; in paint for iron work and ship hulls; as a polishing agent for glass, precious metals, and diamonds; as a catalyst; in colloidal solutions; and as a stain for polysaccharides (Merck 1983, p. 579).

The intraperitoneal LD_{50}s in rats and mice are 5500 mg/kg and 5400 mg/kg, respectively (RTECS 1990). Animals exposed to iron oxide or to iron oxide mixed with less than 5 percent silica by inhalation or by intratracheal injection did not develop pulmonary fibrosis (Naeslund 1940/Ex. 1–650; Harding, Grout, Durkan et al. 1950, as cited in ACGIH 1986/Ex. 1–3, p. 325). A recent study (Wright, Harrison, Wiggs, and Churg 1988) confirms these findings; iron oxide (10 mg) administered to rats by intratracheal injection had not caused any morphological or pulmonary function changes in these animals 30 days later. Hamsters injected once per week for 15 weeks by the intratracheal route of administration with 3 mg ferric oxide dust survived for 1 to 2 years and did not develop tumors (Saffiotti et al. 1968). In an evaluation of iron oxide's carcinogenic potential, the International Agency for Research on Cancer concluded that the evidence for the carcinogenicity of ferric oxide suggested that this substance was not carcinogenic in animals (IARC 1987, Suppl. 7, p. 217).

The evidence of iron oxide's respiratory toxicity in humans is conflicting. Drinker, Warren, and Page (1935/Ex. 1–315) concluded that exposures to iron oxide fume should be maintained below 10 mg/m³, and a U.S. Department of Labor study (1941, as cited in ACGIH 1986/Ex. 1–3, p. 325) found that exposures below 30 mg/m³ were without adverse effect. There are several studies, on the other hand, that report chest X-ray abnormalities in iron oxide-exposed miners, welders, silver polishers, electrolytic iron oxide workers, foundry workers, and boiler scalers (Doig and McLaughlin 1936/Ex. 1–626; Stewart and Faulds 1934/Ex. 1–764; Doig and McLaughlin 1948/Ex. 1–627; McLaughlin, Grout, Barrie, and Harding 1945/Ex. 1–642; Davidson 1951, as cited in McLaughlin 1951/Ex. 1–727; Pendergrass and Leopold 1945/Ex. 1–653; Dunner and Hermon 1944/Ex. 1–731). Some of these workers developed disabling pneumoconiosis; however, the exposures of many of these workers were mixed and in some cases included exposure to varying amounts of silica.

McLaughlin (1951/Ex. 1–727), whose opinion on the subject is widely accepted, believes that the presence of iron oxide dust or fume in the lung causes a pigmentation (termed siderosis) that is responsible for the changes seen in exposed individuals' chest X-rays. Siderosis is believed not to progress to fibrosis, and 6 to 10 years of exposure to about 15 mg/m³ iron oxide dust is required before this condition develops (Fawcett 1943/Ex. 1–736; Fleischer, Nelson, and Drinker 1945/Ex. 1–1051; Hamlin and Weber 1950/Ex. 1–698). In a group of 25 welders exposed primarily to iron oxide fume at 0.65 to 47 mg/m³ concentrations for 3 to 32 years, eight had shadows on their X-rays typical of siderosis, but none of these workers had pulmonary function deficits (Kleinfeld, Messite, Kooyman, and Shapiro 1969).

Dr. Stuart M. Brooks (NIOSH 1986b, p. 425) notes that "[m]ore sophisticated physiologic testing, including measurement of the lung's mechanical properties, is required to better document lung function changes that may occur following inhalation of iron-containing dusts. *In vitro* studies or animal experimentation might be helpful in determining dose-response relationships, understanding lung clearance mechanisms for iron, and elucidating any fibrogenic properties of various ferrous compounds."

Some studies have shown that workers with exposures to iron oxide and such other substances as silica, radon gas, diesel exhaust, corn oils, and the thermal decomposition products of synthetic resins (Faulds 1957/Ex. 1–635; Dreyfus 1936/Ex. 1–897; Bidstrup 1959/Ex. 1–1030; Boyd, Doll, Faulds, and Leiper 1970/Ex. 1–716; Braun, Guillerm, Pierson, and Sadoul 1960/Ex. 1–1141; Monlibert and Roubille 1960/Ex. 1–647; Jorgensen 1973/Ex. 1–1023; Müller and Erhardt 1956/Ex. 1–648; Koskela, Hernberg, Karava et al. 1976/Ex. 1–744; Gibson, Martin, and Lockington 1977/Ex. 1–1053) have a greater risk of developing lung cancer. However, OSHA agrees with the International Agency for Research on Cancer that "Some studies of metal workers exposed to ferric oxide dusts have shown an increased incidence of lung cancer, but the influence of factors in the workplace other than ferric oxide, i.e., soots, silica, and asbestos in foundry work, cannot be discounted. In other studies of metal and chemical workers exposed to ferric oxide, the incidence of lung cancer has generally not been increased" (IARC 1987, Suppl. 7, p. 217). A recent review of the epidemiological and experimental evidence for the carcinogenicity of iron oxide reaches the same conclusion (Lauwerys 1989) but notes that iron-

containing dust may act as a cocarcinogen.

Based on this evidence, OSHA is retaining the Agency's PEL for iron oxide dust and fume of 10 mg/m³, measured as total particulate, in construction and maritime, and is proposing this limit in agriculture. The Agency preliminarily concludes, based on the evidence currently available, that this limit will protect workers in these sectors from developing siderosis, a benign pneumoconiosis that occurs after many years of exposure to iron oxide dust or fume. OSHA considers this condition a material health impairment and believes that this PEL is necessary to reduce the risk of this effect. In addition, promulgation of this limit will make OSHA's PEL for iron oxide consistent across all regulated sectors.

METHYLENE BIS-(4-CYCLOHEXYLISOCYANATE)

CAS: 5124–30–1; Chemical Formula: $C_{15}H_{22}N_2O_2$

H.S. No. 1272

OSHA has no limit for methylene bis (4-cyclohexylisocyanate) in the construction, maritime, or agriculture industries. The ACGIH has a TLV® ceiling limit of 0.01 ppm for this alicyclic diisocyanate compound. NIOSH has no REL for this substance. OSHA is proposing a ceiling limit of 0.01 ppm for this substance in construction, maritime, and agricultural operations. NIOSH (Ex. 8–47, Table N1) concurred with this limit when the Agency recently established it in general industry.

Methylene bis (4-cyclohexylisocyanate) is a liquid belonging to the family of alicyclic diisocyanates. It is used primarily to produce urethane products (ACGIH 1986, p. 392.5(86)).

Methylene bis (4-cyclohexylisocyanate) is a skin, eye, and lung irritant. The oral LD_{50} in rats is 9.9 g/kg (RTECS 1990). By inhalation, exposure to a 20 ppm concentration for 5 hours caused death to 50 percent of exposed rats (RTECS 1990). A 5-percent solution applied to the skin of guinea pigs produced strong erythema and edema, and rabbits treated with 0.1 mg showed severe skin reactions (Younger Laboratories 1965, as cited in ACGIH 1986/Ex. 1–3, p. 392). Rats inhaling a lethal concentration of 20 ppm for five hours exhibited marked respiratory irritation, tremors, and convulsions during exposure, and their lungs revealed severe congestion and edema after death (E.I. du Pont de Nemours and Co. Inc. 1976, as cited in ACGIH 1986/Ex. 1–3, p. 392). Repeated inhalation exposure to 0.4 ppm produced initial weight loss in rats; exposure to 1.2 ppm caused respiratory irritation and decreased growth (E.I. du Pont de Nemours and Co. Inc. 1978, as cited in ACGIH 1986/Ex. 1–3, p. 392). Guinea pigs exposed to 0.12 ppm and mice exposed to 0.65 ppm did not exhibit dermal sensitivity (Stadler and Karol 1984/Ex. 1–612). Unlike toluene diisocyanate, which is a sensory irritant, methylene bis (4-cyclohexylisocyanate) depresses respiration by producing pulmonary irritation; for example, an exposed mouse showed a 50-percent decrease in respiration rate, along with lung irritation, when exposed to 3.7 ppm of this substance (Weyel and Schaffer 1985/Ex. 1–581).

Methylene bis (4-cyclohexylisocyanate) causes sensory irritation of the eyes, nose, and throat, and contact with the eyes can lead to permanent damage (Hazardous Substance Fact Sheet 1986). Workers exposed to this compound have developed skin sensitization, but pulmonary sensitization is less common (Emmett 1976/Ex. 1–552; Israeli, Smirnov, and Sculsky et al. 1981/Ex. 1–701).

OSHA is proposing a ceiling limit of 0.01 ppm for methylene bis (4-cyclohexylisocyanate) in the construction, maritime, and agriculture industries. The Agency believes that this limit will protect workers in these sectors against the significant risk of eye, skin, and pulmonary irritation potentially associated with occupational exposures to this substance at the levels permitted by the absence of an OSHA limit. The Agency considers these irritant effects caused by exposure to methylene bis (4-cyclohexylisocyanate) to be material impairments of health and believes that the proposed limit will substantially reduce the risk of these impairments. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

MICA

CAS: 12001–26–2; Chemical Formula: $K_2Al_4(Al_2Si_6O_{20})(OH)_4$

H.S. No. 1276

In the construction and maritime industries, OSHA has an 8-hour TWA PEL of 20 mppcf for mica containing less than 1 percent crystalline silica; this limit is equivalent to a PEL of 3 mg/m³ when expressed in mg/m³. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 3 mg/m³ for the respirable dust of mica containing less than 1 percent quartz. NIOSH has no REL for this substance. In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA limit of 3 mg/m³ for the respirable dust of mica containing less than 1 percent quartz. NIOSH concurred (Ex. 8–47, Table N1) with this limit when the Agency recently established it in general industry.

Mica is a colorless, odorless, nonflammable, nonfibrous, water-insoluble silicate occurring in plate form and containing less than 1 percent quartz; it includes nine different species. Mica is used in electrical equipment, vacuum tubes, incandescent lamps, dusting agents, lubricants, in windows of high-temperature equipment, exterior paints, cosmetics, roofing, rubber, wallpaper and wall board joint cement, and in oil-well drilling muds (Hawley's 1987, p. 785).

Early animal experiments in guinea pigs involving the intraperitoneal injection of mica showed that this substance was inert by this route of administration (Miller and Sayres 1941). Two workers exposed to muscovite (mica) dust at unspecified concentrations developed hepatic and pulmonary granulomas (Pimenthel and Menezes 1978). Five of six workers exposed to mica dust at concentrations greater than 25 mppcf (about 4 mg/m³) for more than 10 years developed pneumoconiosis (Dreessen et al. 1940). The symptoms most frequently reported by these workers were chronic cough, dyspnea, weakness, and weight loss (Dreessen et al. 1940). Some authors (Parkes 1974) question whether mica alone (without silica) can cause pneumoconiosis. However, Vestal et al. (1943) determined that more than 10 percent of workers exposed to pure mica had developed this condition.

OSHA believes that the evidence strongly suggests that mica is a pneumoconiotic agent and therefore that workers exposed to this substance in their work are at significant risk of experiencing this material health impairment. Accordingly, OSHA is proposing an 8-hour TWA limit of 3 mg/m³ for respirable mica dust containing less than 1 percent quartz in the agriculture industry and is retaining this limit in construction and maritime. This limit corresponds to the existing 20-mppcf PEL and is in keeping with the Agency's decision to delete mppcf values in favor of respirable dust values expressed in mg/m³. The Agency has decided to express this and other similar limits as mg/m³ to facilitate employee exposure monitoring. Promulgation of this limit also will make OSHA's PEL for this substance consistent across all regulated sectors.

MINERAL WOOL FIBER

CAS: None. Chemical Formula: None

H.S. No. 1277

OSHA currently has no limit in any industry sector specifically for mineral wool fiber, but this substance is covered by the Agency's 15-mg/m³ 8-hour TWA limit for all inert dusts and particulates. The ACGIH (1986/Doc. H–020, Ex. 1–3, p. 414) recommends an 8-hour TWA limit of 10 mg/m³ for mineral wool fiber as total dust containing less than 1 percent quartz. OSHA is proposing an 8-hour TWA PEL of 1 fiber per cubic centimeter (f/cc) of air for the respirable fibers of mineral wool in general industry, construction, maritime, and agriculture.

There are two types of mineral wool, known as rockwool and slagwool. These fibers are produced by blowing, centrifuging, or drawing molten rock or slag. Rockwool is typically made from igneous rocks such as diabase, basalt, and olivine, and carbonate rocks containing 40–60% calcium and magnesium carbonates. Slagwool is made from the fused agglomerate by-products of certain metal smelting processes.

In animal studies, exposure to mineral wool fibers by the inhalation route has been shown to cause respiratory effects. Focal fibrosis was observed at 4 months post-exposure in rats exposed to 10 mg/m³ of rockwool or chrysotile asbestos for 50 weeks (Johnson and Wagner 1980, as cited in Vu 1988/Doc. H–043, p. 86). The asbestos-exposed rats were affected to a greater degree. Unexposed rats did not develop fibrosis. Rats and hamsters exposed by inhalation to rockwool (Wagner et al. 1984, as cited in Vu 1988/Doc. H–043, p.86; Le Bouffant et al. 1987, as cited in IARC 1988/Doc. H–043, p. 131) or slagwool (Smith et al. 1987, as cited in IARC 1988/Doc. H–043, p. 131) at concentrations of 0.5–10 mg/m³ developed low levels of pulmonary fibrosis.

Several epidemiological studies of workers exposed to mineral wool have shown that these workers have an excess risk of nonmalignant respiratory disease (NMRD). Weill et al. (1983, as cited in IARC 1988/Doc. H–043, p. 135; 1984, as cited in IARC 1988/Doc. H–043, p. 135) and Hill et al. (1984, as cited in IARC 1988/Doc. H–043, p. 135) have reported the occurrence of lung opacities in a limited number of workers in the mineral wool or synthetic mineral fibers (SMF) manufacturing industry. An increased frequency of bronchitis that was directly related to duration of exposure to SMF has been observed in a group of 135,000 construction workers (Engholm and von Schmalensee 1982/Doc. H–043). The calculated standard rate ratios in each smoking category indicated a positive association between bronchitis and exposure to SMF. Among nonsmokers, 77 percent of cases of bronchitis in workers with long exposure were attributable to the exposure. The corresponding fraction in present smokers was 28 percent. The authors concluded that there is a clear association between bronchitis and handling of SMF in the construction industry. Enterline and co-workers (Enterline et al. 1983/Doc. H–043, Ex.1–17; 1986/Doc. H–043, Ex. 1–1; 1987, Doc. H–043; Enterline 1987, as cited in Vu 1988/Doc. H–043, p. 93) studied 16,661 white male workers in 17 synthetic mineral fibers plants, with 1 or more years of experience in production or maintenance during the period 1945–1963. This cohort consisted of 14,815 workers in 11 fibrous glass plants and 1846 workers in 6 mineral wool plants. The SMR for all synthetic mineral fiber workers for NMRD (excluding influenza and pneumonia) was statistically significantly increased. The SMR was 132.3 ($p < 0.01$) when U.S. rates were used for comparison and 134.1 ($p < 0.01$) when local rates were used. The authors found a nonstatistically significant excess mortality from NMRD among mineral wool workers in the 6 plants studied when their rates were compared with local or national rates. In one plant, however, there was a statistically significant excess risk of NMRD for those workers who had first been exposed 20 or more years ago (SMR 219.9, $p < 0.05$) and for plant workers overall (SMR 221.0, $p < 0.05$) but not for those who had first been exposed less than 20 years ago. The average exposure concentration was 0.353 f/cc for all plants and 0.427 f/cc for the plant with the significant excess risk. Marsh et al. (1990/Doc. H–043) completed a follow-up to 1985 of the Enterline et al. cohort. The SMR for NMRD (excluding influenza and pneumonia) for all SMF workers was 129.4 ($p < 0.01$) when local rates were used for comparison; U.S. rates were not used for comparison. In one mineral wool plant (a different plant than that observed to have increased risk in the earlier study), there was a significantly increased risk of NMRD for those workers who had been exposed 20 or more years ago (SMR 189.0, $p < 0.05$).

Two major epidemiological studies conducted in workers exposed to mineral wool have shown that these workers have an excess risk of lung and other respiratory tract cancers. Enterline et al. (1987/Doc. H–043) examined the 1946–1982 mortality experience of 16,661 SMF workers employed one year or more during 1945–1963 at one or more of 17 U.S. manufacturing plants. Compared with local death rates, there was a statistically significant excess in all malignant neoplasms and in the incidence of lung cancer 20 years or more after first employment. For respiratory cancer, this excess was greatest for mineral wool workers and small-diameter-fiber workers. In the subcohort of 1846 white male workers from six U.S. slagwool or rock-/slagwool plants, the SMRs for respiratory cancer were 148 (based on national comparison) and 134 (local) ($p < 0.01$ and $p < 0.05$). The SMRs for workers with fewer than 20 years since first exposure were 156 (national) and 143 (local) and 146 (national) ($p < 0.05$) and 131 (local) for those with 20 years or more since first exposure. Workers employed most recently had the highest SMRs. For example, in workers who started work during 1950–1959, the SMRs were 216 (national) and 198 (local) (both $p < 0.01$). A smoking survey showed that SMF workers have cigarette-smoking habits similar to those of all U.S. white males. When the effects of smoking were controlled for, the relationship between fiber exposure and respiratory cancer continued to be statistically significant for mineral wool workers. Mean fiber exposure levels in the mineral wool plants were estimated to range from 0.195 to 0.427 f/cc. The highest individual average fiber exposure level was estimated to be 1.4 f/cc. When the study was updated to include a follow-up to 1985, a statistically significant increase in mortality for respiratory cancer was still evident (Marsh et al. 1990/Doc. H–043). Simonato et al. (1987/Doc. H–043) studied 24,609 workers producing rock/slagwool, glasswool, or glass filament in 13 European factories in seven countries. Mineral wool workers numbered 10,115 and worked in seven factories in four countries. The study had an observation period of 30 years from first employment. A slight excess of mortality was seen in these workers overall, and this increase was primarily due to an overall excess rate of cancer. The excess mortality due to lung cancer was significant, particularly in rock/slag wool workers employed during the early production years. The highest SMRs (250 national; 303 local) were seen in the group that had been followed for more than 20 years. For all seven of the mineral wool workers, lung cancer risk increased with time since first exposure. In addition, there was a statistically significant excess in the incidence of cancers of the buccal cavity and pharynx (SIR, 181) (Simonato et al. 1986/Doc. H–043, Ex. 1–4) and a statistically significant increasing trend in bladder cancer mortality with time since first

exposure (Simonato et al. 1987/Doc. H–043). The combined occupational group mean fiber concentrations in these plants, on the basis of measurements taken in 1977–1980, ranged from 0.01 to 0.67 f/cc. The highest concentrations were observed in certain specialist secondary production groups. The mean fiber concentrations in the main production and secondary production groups ranged from 0.05 to 0.12. The average plant median for fiber length ranged from 10–20 μm. The median diameters ranged from 1.2 to 2 μm (Cherrie et al. 1986/Doc. H–043, Ex. 1–4). Robinson and co-workers (1982, as cited in IARC 1988/Doc. H–043, p. 145) reported an increase in mortality from cancer of the digestive tract in a U.S. mineral wool plant. SMRs increased with time since first exposure and with duration of exposure. An increase in stomach cancer mortality with time since first exposure to mineral wool in a plant in Germany was reported by Claude and Frentzel-Beyme (1986, as cited in IARC 1988/Doc. H–043, p. 145).

Mineral wool has also been shown to cause cancer in rats treated by several routes. In a group of 48 rats exposed by inhalation to 10 mg/m³ rockwool (71 percent of fibers between >5 μm and <20 μm in length, 58 percent < 1 μm in diameter) for 7 hours per day for 5 days/week for 12 months, two developed lung adenomas (one tending toward malignant) in the 500–1000 days after the start of exposure. In a group of 48 positive control rats treated with chrysotile (16 percent > 20 μm in length, 29 percent > 0.5 μm in diameter), 11 developed adenocarcinomas and 1 an adenoma (also tending toward malignant). There were no tumors in untreated controls (Wagner et al. 1984, as cited in IARC 1988/Doc. H–043, p. 99). Three of 48 rats treated by intrapleural injection with rockwool coated with resin (70 percent < 5 μm in length, 52 percent < 0.6 μm in diameter) developed mesotheliomas. Two of 48 rats treated with rockwool from which the resin had been removed (70 percent < 5 μm in length, 58 percent < 0.6 μm in diameter) developed mesotheliomas, and 6 of 48 animals treated with chrysotile developed mesotheliomas. There were no tumors in slagwool- or saline-treated rats (Wagner et al. 1984, as cited in IARC 1988/Doc. H–043, p. 100). Pott et al. (1987, as cited in IARC 1988/Doc. H–043, p. 100) treated rats with either fine (median length 14 μm, median diameter 1.5 μm) or coarse (median length 26 μm, median diameter 2.5 μm) slagwool by intraperitoneal (i.p.) injection. With the finer fibers, 2/99 rats developed sarcomas, mesotheliomas, or carcinomas. Six of 99 animals treated with the coarser fibers developed tumors. There were no tumors in controls.

In rats treated by the i.p. (intraperitoneal) route with rockwool (median length 20 μm, median diameter 1.8 μm), tumors of the abdominal cavity developed in 32/53 animals. Saline-treated controls developed tumors in 2/102 rats. Injection of actinolite or chrysotile induced tumors in 20/36 and 31/36 animals, respectively (Pott et al. 1987, as cited in IARC 1988/Doc. H–043, p. 100). Rats injected three times with 75 mg of rockwool (median length 23 μm, median diameter 1.9 μm) or once with 10 mg of a fine fraction (median length 4.1 μm, median diameter 0.64 μm) rockwool developed tumors of the abdominal cavity in 45/63 and 6/45 cases, respectively. The incidence in the saline-treated control group was 3/54 (Pott et al. 1987, as cited in IARC 1988/Doc. H–043, p. 100).

Based on the lack of reported adverse health effects in epidemiologic studies, the ACGIH (1986/Doc. H–020, Ex. 1–3, p. 414) considered mineral wool to be essentially a nuisance dust and applied a 10 mg/m³ TLV®–TWA. NIOSH does not have a REL for mineral wool; however, NIOSH (Doc. H–020, Ex. 8–47) cited recent animal and epidemiological studies indicating the possible carcinogenicity of mineral wool and pointed to statements by Doll (1987/Doc. H–043, Ex. 1–11) that "* * * it is likely that man-made mineral fiber may have about the same carcinogenic potential as asbestos fibers of the same dimension, and that levels of 0.2 f/cc or less in industry are unlikely to produce a measurable risk after 20 years of exposure." NIOSH (Doc. H–020, Ex. 8–47) concluded (citing Doll) that to provide the degree of protection that OSHA believes is necessary for worker health, the PEL should be 0.2 f/cc. NIOSH (Doc. H–020, Ex. 8–47, Table N6B) also believes that a full 6(b) rulemaking is needed for mineral wool. The Thermal Insulation Manufacturers Association (Doc. H–020, Ex. 3–743, p. 9) commented in the earlier rulemaking that OSHA should adopt NIOSH's fibrous glass REL of 3 f/cc for mineral wool. Manville (Doc. H–020, Ex. 8–56) stated that "in March, 1988, Manville recommended to its customers a Workplace Exposure Guideline of 1.0 f/cc for mineral wool" as part of its "commitment to strive for, and realize the lowest achievable exposure to any given substance * * *." E.I. du Pont de Nemours & Company has an Acceptable Exposure Level of 1.0 f/cc for mineral wool fibers (personal communication from Freida Fisher-Tyler, 1991).

The International Agency for Research on Cancer (IARC 1988/Doc. H–043), in evaluating the carcinogenicity of mineral wool, has concluded that mineral wool is "possibly carcinogenic to humans (Group 2B)."

OSHA believes, on the basis both of animal and human evidence, that a link between respiratory disease and exposure to mineral wool exists. Therefore, OSHA is proposing that a 1 f/cc 8-hour TWA for the respirable fibers of mineral wool (fibers less than 3.5 micrometers in diameter and greater than 10 micrometers in length) be established as the PEL for mineral wool to protect workers in general industry, construction, maritime, and agriculture from the significant risk of nonmalignant (and possibly malignant) respiratory disease. Since NIOSH has no counting method for mineral wool, OSHA is soliciting comments on potential counting methods. OSHA will continue to evaluate the scientific evidence regarding the risk of mineral wool-induced nonmalignant and malignant respiratory disease and will, at the time of the final rule, establish a new limit for this substance if the Agency determines that this limit will substantially reduce significant risk. If future information and priorities indicate the need for a more restrictive standard for mineral wool, OSHA will initiate individual-substance rulemaking for this substance.

NICKEL (SOLUBLE COMPOUNDS)
CAS: 7440–02–0; Chemical Formula: Varies
H.S. No. 1283

OSHA's PEL in the construction and maritime industries for soluble nickel compounds (measured as Ni) is 1 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.1 mg/m³ for these substances. NIOSH recommends that exposure to any form of inorganic nickel be maintained at or below 0.015 mg/m³. OSHA is proposing to revise its PEL in construction and maritime to 0.1 mg/m³ as an 8-hour TWA and to extend this limit to agriculture. This is the limit recently established for the soluble nickel compounds in general industry.

A variety of toxic effects results from exposure to soluble nickel compounds. Soluble nickel salts cause contact dermatitis in sensitized individuals and eye irritation (ACGIH 1986/Ex. 1–3, p. 422). Cases of asthmatic lung disease have been reported among nickel-plating workers (EPA 1986a/Ex. 1–1132). OSHA's proposal to lower the PEL for soluble nickel compounds to 0.1 mg/m³

is based primarily on evidence that exposure to soluble nickel at low levels and for relatively short durations causes pathological changes in the lungs of experimental animals. In addition, OSHA has reviewed several animal and human studies designed to investigate the carcinogenic potential of soluble nickel compounds. Three soluble nickel compounds have been tested for their carcinogenic potential: nickel chloride, nickel sulfate, and nickel acetate. Some sparingly soluble compounds, nickel carbonate and nickel hydroxide, have also been studied. The results of these animal studies suggest that some soluble nickel compounds are potentially carcinogenic; however, the data are derived predominantly from injection studies, and the results are often conflicting. Results from occupational studies on soluble nickel compounds are also conflicting and are confounded by the presence of several types of nickel compounds in the facilities studied. OSHA's preliminary findings on the toxicological evidence for the soluble nickel compounds are presented below.

Bingham and Zerwas et al. (1972/Ex. 1–204) exposed rats by inhalation to 0.1 mg/m^3 nickel chloride for 12 hours per day for two weeks. Animals showed evidence of pulmonary irritation and damage in the form of marked mucus secretion, hyperplasia, and accumulations of alveolar macrophages. Fluid obtained by lung lavage appeared very cloudy and viscous due to the presence of free alveolar cells. Rats and guinea pigs exposed daily to 1.0 mg/m^3 nickel chloride (measured as Ni) for six months showed increased lung weight, which is an indication of pulmonary damage and hyperplasia (Clary 1977, as cited in ACGIH 1986/Ex. 1–3, p. 422); exposed rats also developed signs of interstitial fibrotic lesions. Rabbits inhaling 0.3 mg/m^3 nickel chloride (measured as Ni) aerosol for 30 days showed a doubling in alveolar cell number and in the volume of alveolar epithelial cells, as well as nodular accumulation of macrophages and laminated structures (Johansson, Curstedt, Robertson, and Camner 1983/Ex. 1–273). These studies clearly show that exposure at or below 1.0 mg/m^3, even for durations considerably less than a working lifetime, is associated with increased cell turnover and pathological changes in the lung. These pathological changes, in particular the appearance of the fibrotic lesions observed in animals exposed to low levels of soluble nickel salts, indicate that lung damage has occurred and suggest that significant decrements in lung function may result from prolonged exposure to these low levels. Furthermore, the appearance of hyperplasia is indicative of abnormal cell growth and suggests the presence of pre-cancerous lesions.

Nickel chloride has been reported to be mutagenic in *Salmonella typhimurium* and *Cornebacterium* but negative in *E. coli* (EPA 1986a/Ex. 1–1132). The positive studies are not considered conclusive, however, because the *S. typhimurium* report is an abstract lacking detailed data and *Cornebacterium* is not the usual species used in these tests. Amacher and Paillet (1980/Ex. 1–286) reported that nickel chloride was mutagenic in mouse lymphoma cells and demonstrated a dose-response relationship for this endpoint.

Some *in vitro* studies using soluble nickel compounds report finding chromosomal aberrations (EPA 1986a/Ex. 1–1132). These studies do not, however, demonstrate a dose-response relationship or statistical significance, which weakens their findings. Several *in vivo* studies have failed to detect chromosomal aberrations (EPA 1986a/Ex. 1–1132). However, several *in vitro* studies on nickel sulfate and nickel chloride have reported findings of sister chromatid exchanges (EPA1986a/Ex. 1–1132).

Some animal studies involving soluble nickel compounds suggest that these compounds are carcinogenic in animals. Strain A mice receiving intraperitoneal injections of nickel acetate had an increased rate of lung adenomas and adenocarcinomas that was statistically significant in the high-dose group (Stoner, Shimkin, Troxell et al. 1976/Ex. 1–203). The animals were injected three times per week for eight weeks at doses of 72, 180, or 360 mg/kg.

EPA (1986a/Ex. 1–1132) reported a study in which rats were given monthly intramuscular injections of 35 mg/kg nickel acetate for four to six months (Haro, Furst, and Falk 1968/Ex. 1–1022). Twenty-two percent of the treated rats developed sarcomas. Payne (1964/Ex. 1–200) observed tumor responses in rats after intramuscular implantation of 7 mg nickel acetate, nickel sulfate, nickel chloride, or nickel carbonate. Implant-site sarcomas developed in one of 35 rats exposed to nickel acetate, one of 35 rats exposed to nickel sulfate, none of 35 rats exposed to nickel chloride, and four of 35 rats exposed to nickel carbonate.

Results of other studies on nickel sulfate have been negative. Three studies used intramuscular injection in rats and reported that no tumors developed in the treated group (Gilman 1982/Ex. 1–205; Gilman 1986, as cited in EPA 1986/Ex. 1–1132; Kasprzak, Gabryel, and Jaraczewska 1983/Ex. 1–201). An ingestion study also reported no tumors among treated rats or dogs (Ambrose, Larson, Borzelleca et al. 1976/Ex. 1–211).

Gilman (1986, as cited in EPA 1986a/Ex. 1–1132) administered 5 mg nickel hydroxide to rats by intramuscular injection in each thigh. Sarcomas developed at 19 out of 40 injection sites. Kasprzak, Gabryel, and Jaraczewska (1983/Ex. 1–201) gave rats intramuscular injections of nickel hydroxide in gel, crystalline, or colloidal form. Five out of 19 animals receiving the gel developed sarcomas (two with metastasis to the lung), three out of 20 receiving the crystalline form developed sarcomas (one with metastasis to the lung), and none of 13 rats receiving the colloid developed tumors.

In the prior rulemaking, some commenters (Exs. 3–915, 167, 3–668) discussed the limitations of the animal data. These commenters noted that soluble nickel compounds have produced tumors in animals only by injection and that the results among studies are conflicting. OSHA recognizes many of these limitations of the data. Although the animal data are "too limited to support any definitive judgment regarding * * * [the] carcinogenic potential [of soluble nickel compounds]," EPA noted:

> the observation of pulmonary tumors in strain A mice from the administration of nickel acetate by intra-peritoneal injections and the ability of nickel acetate to transform mammalian cells in culture and to inhibit RNA and DNA synthesis provides limited evidence for the carcinogenicity of nickel acetate and supports a concern for the carcinogenic potential of other soluble nickel compounds (EPA 1986a/Ex. 1–1132, p. 8–229).

OSHA agrees with EPA's assessment that, although some studies are suggestive of a carcinogenic effect and an ability of soluble nickel to transform cells, overall the animal data are too equivocal at this time to support any firm conclusions that soluble nickel compounds do or do not cause cancer in experimental animals.

In addition to the animal evidence described above, OSHA reviewed studies conducted on workers exposed to soluble nickel compounds. Electrolysis workers at a refinery in Kristiansand, Norway, experienced a higher lung cancer risk than employees from the same facility who worked in three other job categories, including roasting and smelting workers (Magnus, Andersen, and Hogetveit 1982/Ex. 1–241). Electrolysis workers were exposed to an aerosol composed predominantly

of nickel sulfate, which was estimated to contain soluble nickel at a concentration of 0.2 mg/m³ (EPA 1986a/Ex. 1–1132); these workers also had higher plasma and urine levels of nickel than did roasting and smelting workers, who were predominantly exposed to insoluble nickel subsulfides and oxides. However, exposure to nickel subsulfide and oxides may have occurred in the electrolysis building, and the electrolysis workers may also have worked in other process departments (Grandjean, Andersen, and Nielsen 1988/Ex. 1–207). Roasting and smelting workers were exposed to an estimated average of 0.5 mg/m³ (as Ni) of roasting dust.

The standardized mortality ratios (SMRs) for lung cancer were 550 for electrolysis workers, 390 for other process workers, and 360 for roasting and smelting workers. The pattern of SMRs for nasal cancer, which is a rare form of cancer in humans, was different among these groups: 2600 for electrolysis workers, 2000 for other process workers, and 4000 for roasting and smelting workers. The results seem consistent with studies that show that roasting and smelting workers have the highest concentrations of nickel in the nasal mucosa, presumably because of the relatively larger particles resulting from roasting. Conversely, electrolysis workers, who showed a larger lung cancer risk than roasting and smelting workers, have higher plasma and urine levels of nickel, suggesting that nickel aerosolized by this process penetrates to the deep lung (EPA 1986a/Ex. 1–1132).

The primary impetus to revise the PEL for soluble nickel is to protect workers in construction and maritime, and in agriculture, from soluble nickel's respiratory effects; the other objective is to make the limit for these compounds consistent across all sectors. OSHA preliminarily concludes that the available studies clearly demonstrate that exposure to soluble nickel compounds at the current construction and maritime PEL of 1 mg/m³ presents a significant risk of lung irritation accompanied by pathological changes that may presage cancer. OSHA has preliminarily determined that these effects constitute material impairments of health and functional capacity. OSHA also preliminarily concludes that the proposed rule's reduction in the PEL will substantially reduce these significant risks. Accordingly, OSHA is proposing an 8-hour TWA PEL of 0.1 mg/m³ (measured as Ni) for soluble nickel compounds in the construction, maritime, and agriculture industries. Promulgation of this limit will make the PEL for these substances consistent across all OSHA-regulated sectors.

NITROGEN DIOXIDE

CAS: 10102–44–0; Chemical Formula: NO_2

H.S. No. 1289

The limit for nitrogen dioxide in the construction and maritime industries is a 5 ppm ceiling. There is no limit in agriculture. The ACGIH has a 3 ppm 8-hour TLV®–TWA and a 5 ppm TLV®–STEL for this substance. NIOSH has a REL of 1 ppm as a 15-minute ceiling. The Agency is proposing a 1 ppm STEL for nitrogen dioxide in the construction, maritime, and agriculture industries. NIOSH (Ex. 8–47, Table N1) agreed with the selection of this PEL when the Agency recently established it for general industry.

Nitrogen dioxide is a reddish-brown gas at room temperature that condenses to a brown liquid and freezes to a colorless solid. Nitrogen dioxide has found use as a catalyst in oxidation reactions, an inhibitor to prevent polymerization of acrylates during distillation, a nitrating agent, an oxidizing agent, a rocket fuel and a flour bleaching agent and in the manufacture of liquid explosives (Braker and Mossman 1980, p. 531). Nitrogen dioxide also occurs as a component of diesel exhaust.

OSHA's current 5-ppm ceiling limit in construction and maritime is based primarily on the animal studies of Gray, MacNamee, and Goldberg (1952/Ex. 1–154), Gray, Goldberg, and Patton (1954/Ex. 1–165), and Wagner, Duncan, Wright, and Stokinger (1965/Ex. 1–102). Gray, MacNamee, and Goldberg (1952/Ex. 1–154), and Gray, Goldberg and Patton (1954/Ex. 1–165) demonstrated lung injury among rats exposed for 8 or more weeks to an 8-ppm concentration of a mixture of NO_2 and nitric acid, but these authors did not see such lesions in rats exposed for six months to 4-ppm concentrations of this mixture. Wagner, Duncan, Wright, and Stokinger (1965/Ex. 1–102) reported transient and mild acute effects but no adverse chronic effects in rats exposed to 1 ppm, 5 ppm, or 25 ppm pure NO_2 for 18 months. The ACGIH's recommendation (from which OSHA's limit in construction and maritime derives) that the 5-ppm TLV® be defined as a ceiling rather than as an 8-hour TWA was based on reports that NO_2 accelerated lung tumor development among lung-tumor-susceptible mice; in the late 1960s, the ACGIH believed that a TLV®–ceiling value would minimize the risk of accelerating lung tumor development.

The current ACGIH TLV®s for NO_2 are a 3-ppm 8-hour TWA and a 5-ppm 15-minute STEL, and these limits are based on human studies that indicate that normal respiratory function may be compromised at exposures below the current OSHA limit of 5 ppm NO_2. In particular, Kosmider, Ludyga, Misiewicz et al. (1972/Ex. 1–224) reported a slight reduction in vital capacity and maximum respiratory volume in 70 men exposed to 0.4- to 2.7-ppm concentrations of the oxides of nitrogen 6 to 8 hours daily for 4 to 6 years. These authors also reported an unspecified number of cases of chronic bronchitis among men in this group. Another study by Vigdortschik, Ancheeva, Matussevistch et al. (1937/Ex. 1–49) reported possible cases of chronic bronchitis and emphysema among 127 workers generally exposed below 2.8 ppm NO_2; these workers were also believed to be exposed to sulfuric acid mist at levels sufficient to cause dental erosion.

The NIOSH REL for NO_2 of 1 ppm as a 15-minute STEL is based on the two human studies discussed above, as well as some human studies involving short-term exposure. Abe (1967/Ex. 1–98) found a 40-percent decrease in effective lung capacity among healthy adult males 30 minutes after a 10-minute exposure to 4- to 5-ppm NO_2. Expiratory and inspiratory maximum viscous resistance also increased by 92 percent after exposure. NIOSH (1976j/Ex. 1–265) concluded that Abe's results "document a definite and undesirable effect" at exposures approaching or below the current OSHA limit.

A series of major studies by von Nieding also indicates the need for a 1 ppm STEL. A significant decrease in carbon monoxide diffusing capacity was observed by von Nieding, Krekeler, Fuchs et al. (1973/Ex. 1–770) in healthy adults exposed to 5 ppm for 15 minutes. Two studies (von Nieding, Wagner, Krekeler et al. (1971/Ex. 1–1204) and von Nieding and Krekeler (1971/Ex. 1–1175)) report significant increases in airway resistance among 88 chronic bronchitis patients after a 15-minute exposure to a concentration of NO_2 as low as 1.5 ppm.

NIOSH (1976j/Ex. 1–265) concluded that the specific concentration of NO_2 required to produce pulmonary changes in normal, healthy adults is unknown, but "is likely to be about the same or perhaps a slightly higher concentration than the one inducing pulmonary changes in humans with existing chronic bronchitis" (1.5 ppm). Therefore, NIOSH recommended a 1-ppm 15-minute short-term limit for nitrogen dioxide. To provide additional support for a short-term rather than a TWA limit, NIOSH cited several animal studies that

indicate that the toxic effects associated with exposure are primarily determined by peak, and not average, NO_2 concentrations. NIOSH (Ex. 150) also notes that, in 1984, the World Health Organization, after an independent review of cross-sectional occupational health surveys, recommended a short-term occupational exposure limit of 1.8 mg/m^3 (0.9 ppm) for NO_2 and 8-hour TWA limit of 0.9 mg/m^3 (0.45 ppm).

The EPA limit for NO_2 is 0.053 ppm averaged over 1 year. EPA considers 2 ppm for 1 hour to be a significant harm level that requires an emergency response. EPA's 1982 staff paper concludes that the 1971 von Nieding et al. (Ex. 1–1204) study "provides convincing evidence that chronic bronchitics exposed to NO_2 concentrations of 1.6 ppm or greater for approximately 3 minutes experience increases in airway resistance" (Ex. 3–2e, p. 18). A number of other studies were also cited by EPA (Ex. 3–2f) in which healthy adults were exposed to NO_2 concentrations in the range of 0.5 to 2.5 ppm. Folinsbee, Horvath, Bedi, and Delehunt (1978, as cited in Ex. 3–2e) reported no significant physiological changes in healthy adults exercising for up to one hour during a two-hour exposure to 0.6 ppm NO_2. Suzuki and Ishikawa (1965, as cited in Ex. 3–2e) reported a 50-percent increase in inspiratory flow resistance in healthy adults 10 minutes after a 10-minute exposure to an NO_2 concentration between 0.7 and 2 ppm. Small changes in pulmonary function and a slight increase in the prevalence of respiratory symptoms occurred among healthy adults exposed to 1 ppm NO_2 for 2 hours (Hackney, Thiede, Linn et al. 1978, as cited in Ex. 3–2e). Beil and Ulmer (1976, as cited in Ex. 3–2e) reported a statistically significant increase in airway resistance among healthy adults following exposure to 2.5 ppm NO_2 for two hours, but not following exposure to 1 ppm. Based on their review of these data, EPA concluded:

> [T]he lowest level of NO_2 exposure that credible studies have associated with measurable impairment of pulmonary function appears to be in the range of 1.0–1.6 ppm * * * (Ex. 3–2e, p. 18).

OSHA has also reviewed the most recent analysis of NO_2 toxicity conducted by the National Academy of Science's (NAS) Committee on Toxicology for the Department of Defense (Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants, Vol. 4, pp. 83–96, National Academy Press 1985). In this review, the NAS concluded that exposures to NO_2 at levels between 0.5 and 1.5 ppm have demonstrated "little or no persistent change in pulmonary function" (NAS 1985, p. 89). The NAS Committee on Toxicology recommended short-term public emergency guidance levels (SPEGLs) for NO_2 of 1 ppm, averaged over a 60-minute period, and 0.12 ppm as an 8-hour average.

In the prior rulemaking, several industry commenters objected to OSHA's proposal to establish a PEL at the level of NIOSH's REL for NO_2; these commenters believed that the ACGIH TLV®s of 3 ppm TWA and 5 ppm STEL are sufficiently protective. These commenters pointed to the Linn and Hackney (1984, 1985) papers (subsequently published), which indicated that healthy adults and asthmatics studied did not have pulmonary effects while exercising and being exposed to 4 ppm NO_2.

The AFL–CIO supported OSHA's proposed limits (Ex. 64) in the earlier rulemaking. The AFL–CIO believes that the same studies demonstrate the need for a 1 ppm STEL.

OSHA preliminarily concludes that a 1 ppm STEL is necessary to prevent significant risk of material health impairment. Many of the cohort studies indicate substantial reductions in lung function, a material impairment of health, as well as exacerbation of other health effects, at levels only slightly above 2 ppm. The von Nieding study indicates substantial reductions in lung function in chronic bronchitics exposed for 15 minutes to 1.5 ppm. A substantial percentage of the work force suffers from chronic bronchitis. Beil and Ulmer show reductions in lung function in healthy adults at exposures of 2.5 ppm. Suzuki and Ishikawa (1965) and Rokaw et al. (1968) also show pulmonary function declines at concentrations below 5 ppm. There are also studies showing more minor reductions in lung function in healthy adults at lower levels.

The Linn and Hackney studies present results that conflict to some extent with the findings of the studies described above, i.e., Linn and Hackney show no decline in pulmonary function on exposure to 4 ppm NO_2. However, because so many studies of high quality demonstrate substantial lung function declines at low NO_2 exposure levels, OSHA preliminarily concludes that the weight of the evidence already suggests that substantial declines occur on exposure to NO_2 levels as low as 1.5 ppm. In consequence, the Agency believes that the 1 ppm STEL is necessary to prevent significant risk of material health impairment. As discussed above, this view is clearly supported by NIOSH and the World Health Organization. OSHA's preliminary finding is also consistent with the conclusions reached by EPA and the National Academy of Science.

Thus, OSHA preliminarily concludes that the former 5-ppm ceiling limit for NO_2 in construction and maritime is not sufficient to protect employees from experiencing increased airway resistance. OSHA also preliminarily concludes that the risk of increased airway resistance in construction, maritime, and agriculture will be substantially reduced by promulgation of the proposed 1-ppm short-term limit for NO_2; a short-term limit is clearly indicated for NO_2 because all of the studies cited above demonstrate that increased airway resistance is associated with exposure to NO_2 for durations of between 3 minutes and 2 hours. OSHA considers the increased airway resistance caused by exposure to NO_2 to be a material impairment of health. Therefore, to reduce the significant risk associated with short-term exposure to NO_2, the Agency is proposing a 1-ppm limit, averaged over a 15-minute period, for nitrogen dioxide in the construction, maritime, and agricultural industries. Promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

OXYGEN DIFLUORIDE

CAS: 7783–41–7; Chemical Formula: OF_2

H.S. No. 1300

The PEL for oxygen difluoride in the construction and maritime industries is 0.05 ppm as an 8-hour TWA. The ACGIH has established a limit of 0.05 ppm as a ceiling value. OSHA is proposing a permissible exposure limit of 0.05 ppm as a ceiling for oxygen difluoride in the construction, maritime, and agricultural industries. NIOSH has no REL but concurred (Ex. 8–47, Table N1) with the selection of this limit when the Agency recently established it in general industry.

Oxygen difluoride is an unstable, colorless gas with a foul odor. Oxygen difluoride is used as an oxidizer for rocket propellants (Genium MSDS 1988, No. 228).

Oxygen difluoride is a substance having extremely high acute toxicity; it is an acute irritant and causes fatal pulmonary edema and hemorrhage in animals exposed to 0.5 ppm for a few hours (ACGIH 1986/Ex. 1–3). A single exposure to 0.1 ppm also had an effect on the lungs; animals acutely exposed to oxygen difluoride have also exhibited gross changes in the kidneys and internal genitalia (LaBelle, Metcalf,

Suter, and Smith 1945, as cited in ACGIH 1986/Ex. 1–3, p. 452; Lester and Adams 1965/Ex. 1–963).

Because of the extreme acute toxicity of this compound and the effects seen at 0.1 ppm, OSHA believes that the current 8-hour TWA PEL of 0.05 ppm in construction and maritime is not sufficiently protective of workers; this limit would permit brief periods of high exposure that have been associated with severe lung damage in animals. Furthermore, workers in agriculture are currently unprotected from oxygen difluoride's effects. The Agency has preliminarily determined that these respiratory effects constitute material impairments of health. Therefore, to reduce the significant risk of acute lung damage associated with brief excursion exposures to oxygen difluoride, OSHA is proposing a ceiling limit of 0.05 ppm for this substance in the construction, maritime, and agricultural industries. Promulgation of this limit will also make OSHA's PEL for this substance consistent across all regulated sectors.

OZONE

CAS: 10028–15–6; Chemical Formula: O_3
H.S. No. 1301

The current OSHA PEL for ozone in construction and maritime operations is 0.1 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 0.1 ppm and a 0.3 ppm TLV®–STEL for ozone. NIOSH has no REL. In construction and maritime, OSHA is retaining its permissible exposure limit of 0.1 ppm TWA and proposing to add a 0.3 ppm STEL; the Agency is also proposing to extend these limits to agriculture. Promulgation of these limits will make the PELs for ozone consistent across all OSHA-regulated sectors.

Ozone is a liquid or an explosive gas. Ozone is used as a disinfectant for air and water because of its oxidizing power. It is also used for bleaching waxes, textiles, oils, and in organic syntheses (Merck 1983, p. 6850).

OSHA is retaining the 8-hour TWA limit of 0.1 ppm in construction and maritime, proposing a 15-minute STEL of 0.3 ppm in these two sectors, and is also proposing to extend both limits to agriculture, based on observations that significant declines in pulmonary function can result from repeated intermittent exposures or even from a single short-term exposure (Bils 1970/ Ex. 1–58; Jaffe 1967/Ex. 1–101; Griswold, Chambers, and Motley 1957/Ex. 1–128). OSHA believes that, in the absence of a STEL, employees in these sectors will continue to be at significant risk of material impairment in pulmonary functional capacity associated with short-term exposures that could occur if exposures are controlled only by an 8-hour TWA. Thus the Agency believes that it is necessary to supplement an 8-hour TWA PEL of 0.1 ppm with a 15-minute STEL of 0.3 ppm to substantially reduce this risk. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

PARAQUAT

CAS: 4685–14–7; Chemical Formula:
H.S. No. 1303

OSHA's current limit for paraquat (respirable fraction) in the construction and maritime industries is 0.5 mg/m³ as an 8-hour TWA, with a skin notation. There is no limit in agriculture. The ACGIH has established a TLV® of 0.1 mg/m³ as an 8-hour TWA. NIOSH has no REL for paraquat. In construction, maritime, and agriculture, the Agency is proposing an 8-hour TWA permissible exposure limit of 0.1 mg/m³ for this substance, with a skin notation. NIOSH concurred (Ex. 8–47, Table N1) with this limit when the Agency recently established it in general industry.

Paraquat refers to a group of compounds that are odorless, yellow solids. The principal compounds are: 1,1'-dimethyl-4,4'-bipyridinium; 1,1'-dimethyl-4,4'-bipyridinium bis (methyl sulfate); and 1,1'-dimethyl-4,4'-bipyridinium dichloride. Paraquat is used as an insecticide, either alone or in combination with long-acting herbicides in no-till farming. Paraquat also finds use in industrial and noncrop weed control; in deciduous orchards; and on cotton, soybeans, potatoes, and seed crops (ACGIH 1986, p. 456; Clayton and Clayton 1981, p. 2751). When used in pesticidal applications, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The toxicity of paraquat depends on the compound's cationic moiety. Acute oral toxicity is reported as 30 mg/kg ion (as cation) for guinea pigs and 127 mg/kg ion for female rats, while the dermal LD_{50} in rabbits is 240 mg/kg ion (Clark 1964, as cited in ACGIH 1986/Ex. 1–3, p. 456; Clark, McElligott, and Hurst 1966/ Ex. 1–503; McElligott 1965, as cited in ACGIH 1986/Ex. 1–3, p. 456). The oral LD_{50} in monkeys is 50 mg/kg (Clayton and Clayton 1981, p. 2754). Paraquat can penetrate broken skin after it has broken down the skin's usual barriers (Swan 1969/ Ex. 1–576; Clark, McElligott, and Hurst 1966/Ex. 1–503). By inhalation or intratracheal injection, paraquat is very toxic because of its irritant properties (Gage 1968/Ex. 1–508). Rats exposed once for six hours to a concentration of 1 mg/m³ died if the aerosol contained particles with diameters of 3 to 5 microns (Gage 1968/ Ex. 1–508). Rats exposed 6 hours/day for 3 weeks to the same aerosol at 0.4 mg/m³ exhibited signs of pulmonary irritation; no effects were observed on the same exposure regimen when the concentration was reduced to 0.1 mg/m³ (Gage 1968/ Ex. 1–508).

When particles in the aerosol are not of respirable size, paraquat's toxicity is greatly reduced. The 4-hour LC_{50} for rats is 6400 mg/kg, and dogs, rats, and guinea pigs tolerated 3 weeks of daily exposures to 100 mg/m³ without apparent pulmonary effect (although nosebleeds were observed) (Palazzolo 1965, as cited in ACGIH 1986/Ex. 1–3, p. 456).

Dietary administration of paraquat doses ranging from 300 to 700 ppm for 90 days caused dose-related effects ranging from pulmonary edema to intra-alveolar hemorrhage and death in experimental animals (Kimbrough and Gaines 1970/ Ex. 1–560). Paraquat's teratogenic potency in mice is low (Bus and Gibson 1975/ Ex. 1–539), although 100 ppm administered in the drinking water of pregnant rats increased postnatal mortality significantly (Bus and Gibson 1975/Ex. 1–539).

In humans, 69 accidental deaths and 81 suicides were attributed to the effects of paraquat exposure in the period before 1972 (Chipman Chemicals 1972, as cited in ACGIH 1986/Ex. 1–3, p. 456). Bouletreau, Ducluzeau, Bui-Xuan et al. (1977/Ex. 1–538) reported 31 cases of renal insufficiency, and a spray applicator was killed when he absorbed a lethal dose of inadequately diluted paraquat through the skin (Jaros 1978/ Ex. 1–513). Workers using a 0.05- to 1-percent solution of paraquat developed skin and mucous membrane irritation but experienced no symptoms of systemic poisoning (Howard 1978/Ex. 1–512). Fugita, Suzuki, and Ochiai (1976, as cited in ACGIH 1986/Ex. 1–3, p. 456) reported five cases of reversible keratoconjunctivitis, with corneal injury, after one month of exposure to paraquat.

OSHA is proposing an 8-hour TWA limit of 0.1 mg/m³ for paraquat, with a skin notation, for the construction, maritime, and agriculture industries. The Agency preliminarily concludes that this limit will protect workers in these sectors from the significant risk of skin, eye, and pulmonary irritation observed in animals exposed to paraquat aerosols of respirable size. The Agency considers the irritant effects of paraquat to be material impairments of health. The skin notation for this substance is necessary because of paraquat's capacity to

penetrate the skin. Promulgation of this limit will also make the PEL for paraquat consistent across all OSHA-regulated sectors.

SILICA, AMORPHOUS-
DIATOMACEOUS EARTH
CAS: 68855-54-9; Chemical Formula: SiO_2
H.S. No. 1352

OSHA's permissible exposure limit for amorphous silica, diatomaceous earth in construction and maritime is 20 million particles per cubic foot (mppcf). There is no PEL in agriculture. The ACGIH has a TLV®-TWA of 10 mg/m³ for this substance as an 8-hour limit, measured as total dust; NIOSH has no REL. OSHA is retaining the 8-hour TWA limit of 6 mg/m³ (which is equivalent to 20 mppcf) for amorphous silica, diatomaceous earth in construction and maritime and is proposing this limit in agriculture. This is the limit recently established for this substance in general industry.

Amorphous silica is a non-crystalline, colorless to gray, odorless powder composed of the skeletons of prehistoric plants known as diatoms. Amorphous silica is used in the manufacture of glass, in refractories, abrasives, ceramics and enamels, for decolorizing and purifying oils, and in scouring and grinding compounds (Merck 1983, p. 122; NIOSH/OSHA Occupational Health Guideline 1981, p. 1).

The amorphous forms of silica are believed to be fibrogenic under some circumstances but are generally considered less fibrogenic than the crystalline forms (IARC 1987, Vol. 42, p. 39). The literature contains some reports demonstrating the fibrogenicity of the amorphous forms of silica and others showing no fibrotic effects. For example, the intratracheal instillation of diatomaceous earth dust in animals of several species has caused fibrosis (Gardner 1942, as cited in ACGIH 1986/Ex. 1-3, p. 520) and silicosis (Kovalevich 1957, as cited in ACGIH 1986/Ex. 1-3, p. 520). Another study (Tebbens and Beard 1957/Ex. 1-531) exposed guinea pigs to diatomaceous earth at an average concentration of 60 mg/m³ for 37 to 50 weeks and found both accumulations of dust-laden macrophages and alveolar epithelialization but no fibrosis at autopsy. Rabbits inhaling 40 mg/ml amorphous silica dust for 1100 days showed a diffuse tissue reaction without fibrosis, but intratracheal instillation of amorphous silica particles caused pulmonary lesions in rats within 6 months of the beginning of exposure (IARC 1987, Vol. 42, p. 39).

Cooper and Cralley (1958/Ex. 1-1145) reported linear-nodular changes in the lungs of workers exposed only to amorphous (noncrystalline) silica at unspecified concentrations for 5 years or more. Several other studies (Vigliani and Mottura 1948/Ex. 1-534; Gardner 1942, as cited in ACGIH 1986/Ex. 1-3, p. 520) have reported that diatomite workers develop mild silicosis after such exposure or, in some cases, show no signs of serious lung pathology. For example, Kovalevich (1957, as cited in ACGIH 1986/Ex. 1-3, p. 520) reported silicosis in diatomite workers, while Gardner (1942, as cited in ACGIH 1986/Ex. 1-3, p. 520) found no evidence of lung pathology in exposed workers.

OSHA preliminarily concludes that the evidence suggests that amorphous silica (diatomaceous earth) is a potential respiratory toxin in many exposed workers. OSHA therefore believes that workers in agriculture are at significant risk of experiencing these adverse health effects, and that establishing the proposed 8-hour TWA limit of 6 mg/m³ for amorphous silica is necessary to reduce the significant risk that agricultural workers will experience this material health impairment. OSHA is also changing the units used to express the PEL (but not the value of the PEL) in construction and maritime. In the previous rulemaking, NIOSH (Ex. 8-47, Table 1) concurred that this limit was appropriate for this substance and that expressing the limit in mg/m³ than in mppcf would facilitate sampling and analysis. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

SILICA, AMORPHOUS, PRECIPITATED OR GEL
CAS: None; Chemical Formula: SiO_2
H.S. No. 1353

OSHA has no limit in the construction, maritime, or agriculture industries for amorphous silica in precipitated or gel form. The ACGIH's TLV®-TWA for this substance is 10 mg/m³ as an 8-hour TWA limit, measured as total dust. NIOSH has no REL but concurs (Ex. 8-47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 10 mg/m³ (total dust) for amorphous silica in precipitated or gel form in construction, maritime, and agriculture; this limit is identical to the one recently established for these forms of silica in general industry.

Both the precipitated and gel forms of amorphous silica are non-crystalline, colorless or white, finely divided powders that are odorless. Precipitated silica is precipitated from aqueous solution, and silica gel is a coherent network of particles of colloidal silica. These forms of silica are used as fillers in paint, rubber, and paper, as grease thickeners, as diluents for insecticides, and as carrying agents for catalysts (ACGIH 1986, p. 521; NIOSH/OSHA Occupational Health Guidelines 1985, p. 1).

Studies in laboratory animals have shown no fibrosis after intratracheal or intraperitoneal injection of precipitated silica or silica gel (Klosterkotter 1954/Ex. 1-1156; Klosterkotter 1958/Ex. 1-1039). Schepers and colleagues reported in 1957 that rats exposed for one year and guinea pigs and rabbits exposed for two years to a concentration of 126 mg/m³ of amorphous silica in precipitated form displayed no pulmonary fibrosis; the effects of exposure were limited to macrophage accumulation and mild proliferation of reticulin fibers (Schepers, Durkan, Delahant et al. 1957/Ex. 1-755). An unpublished NIOSH study (Groth, Kommineni, Stettler et al. 1979) showed that rats, guinea pigs, and monkeys developed accumulations of macrophages in the lungs after exposure to precipitated silica; in addition, the presence of collagen was seen in a "very few" monkeys. By comparison, collagen was not seen in any animal exposed to silica gel but was seen in significant amounts in monkeys exposed to fumed silica (a crystalline variety of silica).

In a study of human exposures to amorphous silica in precipitated form, no ill effects were reported in 165 workers exposed to an unspecified concentration of this substance for an average of 8.6 years (Wilson, Stevens, Lovejoy et al. 1981/Ex. 1-1177). However, the literature suggests that exposure to all forms of silica dust may produce fibrosis, although exposure to the amorphous forms (such as precipitated or gel) is likely to be less fibrogenic than exposure to the crystalline forms (IARC 1987, Vol. 42, p. 85).

Based on this evidence, OSHA preliminarily concludes that exposure to amorphous silica (in precipitated or gel form) has the potential to cause respiratory effects in exposed workers. OSHA therefore believes that, in the absence of a limit, workers in construction, maritime, and agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that a limit of 10 mg/m³ for amorphous silica in precipitated or gel form is necessary in construction, maritime, and agriculture to substantially reduce this risk of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for these substances consistent across all regulated sectors.

SILICA, CRYSTALLINE—CRISTOBALITE

CAS: 14464–46–1; Chemical Formula: SiO_2

H.S. No. 1354

The OSHA PEL for cristobalite in the construction and maritime industries is based on the formula $250/\%SiO_2+5$ and is measured as millions of particles per cubic foot (mppcf). There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 0.05 mg/m³, measured as respirable silica dust, and the NIOSH REL for this substance is 50 μg/m³ as a 10-hour TWA. OSHA is proposing an 8-hour TWA permissible exposure limit of 0.05 mg/m³ for cristobalite, measured as respirable silica dust, for the construction, maritime, and agriculture industries. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Cristobalite, one of the three major crystalline polymorphs of silicon dioxide, is transparent, tasteless, and stable at high temperatures. It occurs naturally in volcanic rock and is used as a filtering and insulating medium and in silicaceous refractory materials for furnace linings and silica bricks (Clayton and Clayton 1981, p. 3020). Cristobalite is also used in the manufacture of water glass, abrasives, ceramics, and enamels (ACGIH 1986, p. 522).

There is widespread agreement in the literature that silica in crystalline form is highly fibrogenic in exposed animals and humans (Merchant 1986, p. 217). The fibrotic disease caused by the inhalation and deposition of silica is called silicosis; silicosis occurs both in acute and chronic form and is often associated with tuberculosis (Merchant 1985, p. 217). Although the human and toxicological literature often fails to distinguish among the three primary crystalline forms of silica (cristobalite, tridymite, and tripoli), a study by Gardner (1938, as cited in ACGIH 1986/Ex. 1–3, p. 522) that was subsequently confirmed by King, Mohanty, Harrison, and Nagelschmidt (1953/Ex. 1–85) showed that experimental animals injected with cristobalite showed a more severe response than that produced by pure quartz and that the resulting fibrosis that followed was diffuse rather than nodular. OSHA's toxicological writeup for the crystalline forms of silica appears under the entry for "Silica, Crystalline Quartz, Respirable," below; readers are referred to that section for additional details on the toxicology of crystalline silica.

OSHA is proposing to express the limit for cristobalite in mg/m³ rather than as a formula to simplify employee exposure monitoring in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that, in the absence of a limit, exposure to cristobalite presents a significant risk of silicosis and possible carcinogenicity to agricultural workers. OSHA believes that the promulgation of this proposed limit will substantially reduce this risk. In addition, promulgation of 0.05 mg/m³ for cristobalite will make the PEL for this substance consistent across all regulated sectors.

SILICA, CRYSTALLINE QUARTZ, RESPIRABLE

CAS: 14808–60–7; Chemical Formula: None

H.S. No. 1355

The current OSHA limit for silica-containing dusts in construction and maritime (there is no limit in agriculture) is a limit expressed in millions of particles per cubic foot (mppcf) and derived from the following formula:

$$\frac{250^*}{\%SiO_2+5}$$

* The percentage of crystalline silica in the formula is the amount determined from airborne samples, except in those cases in which other methods have been shown to be applicable.

At one time, the ACGIH also expressed its quartz limit in terms of this formula. However, the current ACGIH TLV®–TWA is 0.1 mg/m³, measured as respirable quartz dust. NIOSH has a REL for this substance of 0.05 mg/m³ as a 10-hour TWA. OSHA is proposing an 8-hour TWA permissible exposure limit of 0.1 mg/m³, measured as respirable quartz, for workplaces in the construction, maritime, and agriculture industries. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Quartz is a colorless, odorless, noncombustible solid. It is used in electronic components; radio and television components; wave filters; as a barrel-finishing abrasive; a piezoelectric control in filters; and in oscillators. Exposure can occur in sand blasting, pottery manufacture, hard rock mining, and in the spraying of vitreous enamels (Genium MSDS 1990, No. 71), as well as in any operation involving the handling, cutting, or shaping of stone. Silica exposures often occur in dusty out-of-doors environments, and workers in construction and agriculture are exposed to silica in areas where the soil is silicaceous.

Occupational exposure to respirable silica has been known for many years to produce silicosis, a fibrotic disease of the lungs characterized by the formation of silica-containing nodules (Merchant 1986). Silicosis may occur in both acute and chronic forms (Merchant 1986). The acute form usually occurs after "very high" (not further characterized) exposures lasting for 1 to 3 years; in this form of the disease, intraalveolar deposits, similar to those seen in alveolar proteinosis, develop in exposed individuals (Merchant 1986). The chronic forms of silicosis are more common and are usually seen after 20 to 45 years of exposure to dusts containing 30 percent or less silica. The lesions of chronic silicosis are nodular in form and are generally concentrated in the upper lobes of the lungs. In simple silicosis, nodules are 5 mm or less in diameter and tend not to cause substantial pulmonary deficits (Merchant 1986). As the disease progresses, however, these lesions coalesce to form progressive massive fibrosis; at this stage, the disease is called complicated silicosis, and the pulmonary function of the victim is seriously compromised (Merchant 1986).

In the prior rulemaking, several commenters on the PEL for silica focused on two issues: (1) The adequacy of the proposed 0.1-mg/m³ respirable quartz limit in reducing the risk of silicosis; and (2) recent evidence describing the potential carcinogenicity of silica dust.

With regard to the first issue, available data do not appear to be adequate to determine a no-effect level for crystalline quartz. For example, Dr. John Peters, the author of the chapter on silica in the 1986 NIOSH reference on respiratory diseases, concluded as follows:

> All of the studies described in this section provide evidence for adverse pulmonary effects at levels of exposure above 10 mppcf or 0.1 mg/m³. Some showed that foundry workers exposed to the equivalent of 0.05 mg/m³ of quartz developed silicosis while those with less exposure did not * * *. All the Vermont findings were seen with an average exposure of around 0.05 mg/m³ of quartz. It is possible, however, that since this was the average exposure, individuals whose exposure exceeded this level accounted for the noted effects. (The "no effect" level was probably below 0.05 mg/m³, but the available data did not allow accurate determinations.) (Peters, J.M., "Silicosis." In: Occupational Respiratory Diseases, p. 229, J.S. Merchant, ed. DHHS (NIOSH) Pub. No. 86–102, NIOSH 1986b).

OSHA agrees with Dr. Peters that the available data are not adequate for the purpose of establishing a NOEL for this substance and is therefore proposing at this time a 0.1 mg/m³ PEL that will ensure consistency in the limit across all

OSHA-regulated sectors. As discussed below, OSHA expects to consider silica's potential toxicity in detail in the first update proceeding, and the appropriateness of the PEL will be evaluated at that time.

The second major issue raised by participants in the prior air contaminants rulemaking concerned silica's carcinogenicity. There is considerable evidence that silica is carcinogenic both in animals and humans. For example, silica has caused tumors of many kinds at various sites (site-of-contact tumors and tumors of the lungs, liver, gastrointestinal tract, kidneys, and bronchi) in three species of animal (rats, mice, and hamsters) and by six routes of exposure (inhalation, intraperitoneal, intrapleural, intravenous, intratracheal, and implanted under the skin) (RTECS 1991; IARC 1987, Vol. 42; IARC 1987, Suppl. 7).

The International Agency for Research on Cancer characterizes the evidence for the carcinogenicity of silica in animals as sufficient and summarizes its findings as follows:

> Various forms and preparations of crystalline silica produced adenocarcinomas and squamous-cell carcinomas of the lung in rats after inhalation or repeated intratracheal instillation. Thoracic and abdominal malignant lymphomas developed in rats after single intrapleural and intraperitoneal injections of suspensions of several types of quartz. Malignant lymphomas developed after intrapleural injection of cristobalite and tridymite. No tumorigenic response was observed in hamsters after repeated intratracheal instillation of quartz dusts or in a mouse-lung adenoma assay with one sample of quartz.

In addition to these findings in animals, IARC summarized the epidemiologic studies of workers exposed silica in a variety of occupational settings as follows:

> A number of studies have shown that persons diagnosed as having silicosis after occupational exposure to dust containing crystalline silica have an increased risk for dying from lung cancer. This increase has been seen among miners, quarry workers, foundry workers, ceramic workers, granite workers and stone cutters.
>
> Workers in the granite industry have shown increased risks for lung cancer in some studies; the excesses were of the order of 10–30% and were not usually statistically significant. An extended follow-up of Finnish granite workers showed 22 lung cancer cases, with 17.1 expected. When allowing for a latency of 15 years, 21 cases were observed, whereas nine were expected ($p<0.01$; Poisson distribution). Smoking habits were similar to those of the active Finnish male population, and exposures to radon and asbestos were considered unlikely to have occurred. A recent joint Nordic register linkage study, combining lung cancer mortality and incidence data from the cancer registries with census-based records on previous occupation of 20–64-year-old males, showed an elevated risk of lung cancer among stone cutters in Finland and Denmark, but not in Sweden or Norway. Excess risk was also seen for Finnish males in excavation work, whereas no such risk was evident in the other countries.
>
> Three epidemiological studies of workers in the ceramics, glass and refractory brick industries, using different designs, have shown a roughly two-fold increase in mortality from lung cancer. Only one case-referent study took smoking into account. The Nordic register study also found an excess of lung cancer for Danish glass-workers, but workers in the ceramics industry did not have an elevated risk in any of the other countries. A U.S. cohort study of pottery workers exposed to silica and talc showed a nonsignificant standardized mortality ratio of 137 for workers exposed to high levels of silica dust with no talc exposure.
>
> Several studies of metal miners have shown mortality rates from lung cancer some 20–50% higher than expected. In the Nordic register study, relative risks from 1.0 (Norwegian metal miners) to 5.0 (Finnish nonferrous ore miners) were seen. The largest group was Swedish iron ore miners; their relative risk was 3.2 (95% confidence interval, 2.9–3.5), based on 124 observed cases. However, in repeated cohort studies of workers in a gold mine, no excess lung cancer risk was seen. The contribution of radon has not in general been assessed.
>
> Coal miners appear not to be at increased risk of lung cancer.
>
> Epidemiological studies of both exposed populations and silicotics give indications of the carcinogenicity of a working environment contaminated with crystalline silica, particularly in combination with other exposures. In most industries studied, such an effect cannot be separated from those of other concomitant carcinogenic exposures, but in the granite and stone industry the exposure to silica is fairly pure. Few studies provide data on smoking. It is not clear whether the mechanisms of a possible carcinogenic effect of crystalline silica requires a fibrotic process.

Based on a review of the human and animal evidence for the carcinogenicity of crystalline silica, the International Agency for Research on Cancer has assigned this substance a Group 2A (probably carcinogenic to humans) classification. (OSHA notes that IARC's findings apply to all of the crystalline forms of silica, including quartz, cristobalite, tridymite, tripoli, and fused silica.)

Because OSHA believes that the primary objective of the present rulemaking in construction, maritime, and agriculture should be the promulgation of consistent PELs in all OSHA-regulated sectors, the Agency is not at this time initiating a full 6(b) rulemaking on crystalline silica but is postponing a comprehensive review of silica's cancer-causing potential until the first PEL update. At present, OSHA believes that it is appropriate to propose an 8-hour TWA PEL of 0.1 mg/m^3 for quartz, measured as the respirable silica fraction, in construction, maritime, and agriculture. This limit represents no substantial change in value from OSHA's current formula limit for this substance in construction and maritime, but will simplify employee sampling procedures in these sectors. With regard to the agricultural sector, OSHA preliminarily concludes that, in the absence of a limit, exposure to respirable quartz dust presents a significant risk of silicosis, and perhaps of cancer, to workers. As OSHA's Preliminary Regulatory Impact Analysis for the agricultural sector shows, many agricultural workers are potentially exposed to silica in the course of their work. OSHA believes that promulgation of the 0.1 mg/m^3 limit will substantially reduce this risk for these workers. In addition, promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

SILICA, CRYSTALLINE QUARTZ (TOTAL DUST)

CAS: 14808–60–7; Chemical Formula: SiO_2

H.S. No. 2142

The current OSHA limit for silica-containing dusts (measured as total dust) in construction and maritime is expressed as mppcf and derives from the following formula: 250/%SiO_2+5. There is no PEL in agriculture. NIOSH has no REL for silica measured as total dust; the ACGIH TLV®–TWA is 0.3 mg/m^3. OSHA is proposing an 8-hour TWA PEL of 0.3 mg/m^3 for crystalline quartz (total dust) in agriculture and is additionally expressing the PEL for this substance in mg/m^3 rather than as a formula to simplify exposure monitoring. This action would make OSHA's PEL for crystalline quartz (total dust) consistent across all regulated sectors.

Crystalline quartz occurs naturally in certain types of soil (e.g., in sandy soil) and can also be produced by heating amorphous or other types of silica to high temperatures. Crystalline silica consists of colorless crystals and is used or liberated in the manufacture of glass, porcelain, and pottery, in metal casting, sandblasting, and granite cutting, and in the production of refractory, grinding, and scouring compounds (Clayton and Clayton 1981, p. 3020; Proctor, Hughes, and Fischman 1988, p. 440). Agricultural workers are often exposed to silica when they till silicaceous soil.

Silica exposure causes a chronic and disabling pulmonary disease, called

silicosis, and may cause cancer in exposed workers. The animal and human studies describing silica's toxicity do not generally differentiate between total dust and the respirable fraction (i.e., do not identify the particle size of the particulate causing the effect). It is therefore difficult to determine which studies relate to total dust and which involve the respirable particulate. However, there is general agreement that particle size is the principal factor affecting silica's toxic potential (Merchant 1986). Accordingly, OSHA is proposing a less stringent limit for crystalline quartz (total dust) than for crystalline quartz (respirable fraction); the proposed PELs for these substances are 0.1 mg/m³ and 0.3 mg/m³, respectively. The toxicology writeup for crystalline silica appears in this preamble under the entry for "Crystalline silica—quartz (respirable)" and the reader is referred to that section for the details of silica's toxicity in animals and humans.

Based on the voluminous evidence of crystalline silica's toxicity, OSHA preliminarily concludes that the proposed 8-hour TWA PEL of 0.3 mg/m³ is necessary in agriculture to protect workers in this sector from the significant risk of silicosis and other diseases, including cancer, potentially associated with exposure to crystalline quartz (total dust). The Agency considers these diseases to be material impairments of health and preliminarily concludes that the proposed PEL will substantially reduce these risks. Furthermore, OSHA believes it appropriate to propose an 8-hour TWA PEL of 0.3 mg/m³ for quartz, measured as total dust, for construction and maritime. This limit represents no substantial change from OSHA's current formula limit for these sectors, but will simplify employee sampling procedures. Promulgation of this limit will also make the PEL for crystalline silica, quartz (total dust) consistent across all OSHA-regulated sectors.

SILICA, CRYSTALLINE—TRIDYMITE
CAS: 15468–32–3; Chemical Formula: SiO_2
H.S. No. 1358

OSHA's PEL for tridymite in the construction and maritime industries is expressed as the formula $250/\%SiO_2+5$. There is no limit in agriculture. The ACGIH recommends an 8-hour TWA limit of 0.05 mg/m³, measured as silica dust. The ACGIH limit is based on a study conducted by King, Mohanty, Harrison, and Nagelschmidt (1953/Ex. 1–85) that found tridymite to be the most active of the free silica forms when injected intratracheally into rats. NIOSH has a REL for this substance of 0.05 mg/m³ as a 10-hour TWA. The Agency is retaining its PEL for tridymite in construction and maritime but is now expressing this limit in mg/m³ rather than as the formula; the proposed limit is 0.05 mg/m³, and OSHA is also proposing to extend this limit to agriculture. An 8-hour TWA limit of 0.05 mg/m³ was recently established for tridymite in general industry; promulgation of the proposed 0.05 mg/m³ limit in construction, maritime, and agriculture will thus make OSHA's PEL for tridymite consistent across all regulated sectors.

Tridymite is a transparent, tasteless form of free silica. Tridymite is used as a filtering and insulating medium and as a silicaceous refractory material for furnace linings and silica bricks (Clayton and Clayton 1981, p. 3020). Tridymite is one of the crystalline polymorphs of silica; accordingly, the toxicology writeup that appears in this preamble under the heading for "Crystalline silica quartz (respirable)" also applies to tridymite, and the reader is referred to that section for the details of silica's toxicity in humans and animals.

Although expressed in different units, OSHA's current and proposed limits for tridymite in construction and maritime are comparable. The mg/m³ limit is being proposed merely to simplify the monitoring of tridymite dust concentrations. OSHA is proposing to replace its current formula limit for tridymite in construction and maritime with a numerically equivalent 8-hour TWA limit of 0.05 mg/m³, measured as respirable silica dust, and to extend this limit to agriculture. OSHA is proposing this change to simplify employee exposure monitoring in the construction and maritime industries, and to substantially reduce the significant risk of tridymite-induced silicosis and possible cancer that exists in the agricultural sector in the absence of a limit for this substance.

SILICA, CRYSTALLINE—TRIPOLI
CAS: 1317–95–9; Chemical Formula: SiO_2
H.S. No. 1357

Although OSHA's Table Z-2, which is presently in effect in construction and maritime, does not specifically indicate a limit for tripoli, OSHA's current limit for crystalline quartz (which applies to tripoli) is expressed as mppcf and derives from the formula:

$$\frac{250^*}{\%SiO2+5}$$

*The percentage of crystalline silica in the formula is the amount determined from airborne samples, except in those cases in which other methods have been shown to be applicable.

The 8-hour TLV®–TWA ACGIH limit for tripoli is 0.1 mg/m³, measured as respirable silica dust. NIOSH has a REL of 50 μg/m³ as a TWA for tripoli. OSHA is proposing an 8-hour TWA PEL of 0.1 mg/m³ for tripoli in the construction, maritime, and agriculture sectors. This is the limit recently established for tripoli in general industry.

Tripoli is a colorless, microcrystalline polymorph of silica. It is used in buffing compounds, on buffing wheels, in scouring soaps, powders, and polishes, and for polishing optical lenses (Clayton and Clayton 1981, p. 3020). Because tripoli is a crystalline form of silica, the toxicology writeup that appears in this preamble under the heading for "Crystalline silica quartz (respirable)" also pertains to tripoli.

In construction and maritime, OSHA is proposing to replace its limit for tripoli, which is expressed as the formula presented above, with a comparable 8-hour TWA limit of 0.1 mg/m³, measured as respirable silica dust; the Agency is also proposing to extend this limit to agriculture to reduce the significant risk of tripoli-induced silicosis and possible cancer among workers in this sector. Promulgation of this limit will make the PEL for tripoli consistent across all OSHA-regulated sectors.

SILICA, FUSED
CAS: 60676–86–0; Chemical Formula: SiO_2
H.S. No. 1358

Fused silica is a crystalline form of quartz. As such, it is currently covered in construction and maritime by OSHA's PEL for quartz. OSHA's current limit for quartz dust in construction and maritime operations is the formula 250/%SiO2+5, measured as mppcf; there is no PEL in agriculture, and NIOSH has no REL. The ACGIH has an 8-hour TLV®–TWA limit of 0.1 mg/m³, measured as free silica; the ACGIH adopted this limit in 1985 to simplify the monitoring of quartz dust concentrations. OSHA is proposing a 0.1 mg/m³ 8-hour TWA PEL for fused silica in construction, maritime, and agricultural operations. The limit being proposed for construction, maritime, and agriculture was recently established for fused silica in general industry.

Fused silica is a colorless, odorless, noncombustible solid that is employed in the aerospace industry as an ablative material in rockets and spacecraft. Fused silica can also be used in making camera lenses and finds use in fiber form as a reinforcer of plastics (ACGIH 1986, p. 521).

In construction and maritime, OSHA is replacing its current limit for fused silica, which is expressed as the formula presented above, with a comparable 8-hour TWA PEL of 0.1 mg/m³, measured as respirable silica dust; the Agency is also proposing to extend this limit to agriculture to reduce the significant risk of silicosis and possible cancer that exists in the absence of a limit for fused silica in this sector. OSHA believes that this limit will also simplify employee exposure monitoring in construction and maritime. In addition, promulgation of this limit will make OSHA's PEL for fused silica consistent across all regulated sectors. Because fused silica is a crystalline form of silica, the toxicology writeup that appears in this preamble under the heading for "Crystalline silica quartz (respirable)" also applies to fused silica.

SOAPSTONE, TOTAL DUST
SOAPSTONE, RESPIRABLE DUST
CAS: None; Chemical Formula: 3 MgO–4 SiO_2–H_2O
H.S. No. 1363 (total dust)
H.S. No. 1363A (respirable dust)

OSHA's current exposure limit for soapstone, total dust, in construction and maritime operations is 20 mppcf (6 mg/m³); the Agency has no separate limit for the respirable fraction. There is no limit in agriculture. The ACGIH has established individual TLV®–TWAs for two forms of soapstone: 6 mg/m³ for total dust and 3 mg/m³ for the respirable fraction, both measured as total dust or respirable dust containing less then 1 percent quartz. Because the ratio of total dust mass to the mass of the respirable fraction is 2:1 (ACGIH 1984, p. 480), the 6 mg/m³ total dust limit automatically implies a 3 mg/m³ limit for the respirable fraction. NIOSH has no REL for soapstone. OSHA is proposing permissible exposure limits of 6 mg/m³ as an 8-hour TWA (total dust) and 3 mg/m³ as an 8-hour TWA (respirable dust) for soapstone in construction, maritime, and agriculture. NIOSH (Ex. 8–47, Table N1) concurred with these limits when the Agency recently established them in general industry.

A study by Dreessen and DallaValle (1935/Ex. 1–588) of mill workers exposed to soapstone showed lung changes in these workers, but it is believed that the dusts involved in these exposures were actually composed of steatite talc, which had a tremolite content of 10 percent. Experiments by Miller and Sayers (1941/Ex. 1–595) showed no measurable toxic effects in guinea pigs injected intraperitoneally with various samples of soapstone.

In construction and maritime, OSHA is proposing to express its limit for soapstone (total dust) in mg/m³, rather than mppcf, to simplify employee sampling and analysis. The total dust limit being proposed for the construction and maritime industries is an 8-hour TWA of 6 mg/m³, which is equivalent to the current limit of 20 mppcf, and the new limit of 3 mg/m³ for respirable dust has been implicit in the existing total dust limit. In agriculture, OSHA is proposing both limits to reduce the risk of tremolite-induced pulmonary effects in exposed workers. Promulgation of these limits will also make OSHA's PELs for this substance consistent across all regulated sectors.

SULFUR DIOXIDE
CAS: 7446–09–5; Chemical formula: SO_2
H.S. No. 1375

OSHA's current limit for sulfur dioxide (SO_2) in the construction and maritime industries is 5 ppm as an 8-hour TWA. There is no limit in agriculture. The Agency is proposing to revise this limit to 2 ppm as an 8-hour TWA, to add a 5 ppm STEL, and to extend these limits to agriculture. Although NIOSH recommends a limit of 0.5 ppm for sulfur dioxide, NIOSH did concur (Ex. 8–47, Table N1) with OSHA's proposed limits when the Agency established them recently in general industry. The ACGIH has a TLV®–TWA of 2 ppm and a TLV®–STEL of 5 ppm for sulfur dioxide. Promulgation of the proposed limits will make the PELs for this substance consistent across all OSHA-regulated sectors.

Sulfur dioxide is a colorless, nonflammable gas or liquid with a suffocating odor. It is used in treating wood pulp for paper manufacturing, in ore and metal refining, and in the extraction of lubricating oils; as a bleaching, disinfecting, and fumigating agent; as a food additive and preservative; and as a reducing agent (ACGIH 1986, p. 542 (87)). Sulfur dioxide is generated as an unwanted byproduct of combustion, and especially during the burning of coal or heavy oil. When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Workplace exposure to sulfur dioxide causes both acute and chronic effects. The chronic effects of exposure include permanent pulmonary impairment, which is caused by repeated episodes of bronchoconstriction. A number of human and animal studies demonstrate this effect (Skalpe 1964/Ex. 1–438; Smith, Peters, Reading, and Castle 1977/Ex. 1–805; Archer and Gillam 1978/Ex. 1–711; Ministry of Health (Canada) 1976/Ex. 1–1208; Lewis, Campbell, and Vaughan 1969, as cited in ACGIH 1986/Ex. 1–3, p. 542).

In a study of Norwegian paper pulp mill workers, Skalpe (1964/Ex. 1–438) reported that average SO_2 concentrations were believed to range from 2 to 36 ppm. Results showed a significantly higher frequency of respiratory disease symptoms, including coughing, expectoration, and dyspnea, among workers less than 50 years of age (i.e., those with the shortest exposure). Workers older than 50, however, did not display symptomatology different from that of controls.

More recently, Smith, Peters, Reading, and Castle (1977/Ex. 1–805) studied a group of smelter workers exposed, on average, to less than 2 ppm SO_2 but concurrently exposed to respirable particulate at levels generally less than 2 mg/m³. These workers showed a decrement in forced vital capacity (FVC) and forced expiratory volume (FEV_1) of 4.8 percent when compared with controls. These authors concluded that workers exposed to SO_2 levels above 1 ppm had an accelerated loss of pulmonary function. This study has been criticized on the grounds that the control population itself may have been exposed to respiratory toxins and that other contaminants, such as iron sulfites, may have contributed to the pulmonary decrement seen in these smelter workers. (OSHA notes that the first criticism would lead to an underestimate of the severity of the health effects.) On average, 60 percent more of the workers exposed to concentrations of SO_2 greater than 1 ppm reported symptoms of chronic cough than did workers who were exposed to SO_2 at a concentration below 1 ppm. The prevalence of chronic sputum production was elevated for workers who had never smoked and who were exposed above 1 ppm.

Archer and Gillam (1978/Ex. 1–711) studied workers at the same smelter facility and obtained results similar to those of Smith, Peters, Reading, and Castle (1977/Ex. 1–805). Significant reductions in FVC and FEV_1 were found to be associated with chronic exposures to 0.4 to 3 ppm SO_2 (TWA) with concomitant exposure to particulate. These authors also found a corresponding increase in some symptoms of respiratory disease (chronic bronchitis) that was not attributable to smoking. Tomono and coworkers (1961, as cited in ACGIH 1986/Ex. 1–3, p. 542) found that 1.6 ppm was the lowest concentration that

produced bronchoconstriction in 46 healthy male subjects.

NIOSH recommends a 0.5-ppm 8-hour TWA limit for SO_2. In addition to the studies by Archer and Gillam (1977/Ex. 1–711) and Smith, Peters, Reading, and Castle (1977/Ex. 1–805) described above, NIOSH relied on a third study (Ministry of Health (Canada) 1976/Ex. 1–1208) of smelter workers exposed to SO_2 levels of 2.5 ppm for 10 or more years, which showed an increased incidence of respiratory disease in these workers. A fourth study cited by NIOSH (NIOSH 1977m, as cited in ACGIH 1986/Ex. 1–3, p. 542) reported that 10,000 workers exposed to SO_2 at levels of 0.35 ppm showed no adverse exposure-related effects.

Alarie and co-workers (1970 and 1972, as cited in ACGIH 1986/Ex. 1–3, p. 542) found that guinea pigs exposed to SO_2 by inhalation showed no decrement in pulmonary function at SO_2 levels of 5 ppm; monkeys exposed to 1.3 ppm for 78 weeks also showed no deficit (Alarie, Ulrich, Busey et al. 1970 and 1972, both as cited in ACGIH 1986/Ex. 1–3, p. 542). However, in another study, dogs exposed continuously to 5 ppm for 225 days showed increased pulmonary flow resistance and a decrease in lung compliance (Lewis, Campbell, and Vaughan 1969, as cited in ACGIH 1986/Ex. 1–3, p. 542). In addition, rats exposed to 10 ppm SO_2 daily for six weeks developed a thickening of the mucous layer that interfered with effective particle clearance (Dalhamn 1956, as cited in ACGIH 1986/Ex. 1–3, p. 542).

The acute effects of SO_2 exposure have been recognized for years in industrial settings; symptoms of acute overexposure include upper respiratory tract irritation, rhinorrhea, choking, and coughing. These symptoms are so disagreeable that most persons will not tolerate exposure for longer than 15-minutes. Within 5 to 15 minutes of the onset of exposure, workers develop temporary reflex bronchoconstriction and increased airway resistance. Short-term exposure causes measurable bronchoconstriction (Frank, Amdur, Worcester, and Whittenburger 1962, as cited in ACGIH 1986/Ex. 1–3, p. 542; Weir, Stevens, and Bromberg 1972/Ex. 1–401); the ACGIH (1986/Ex. 1–3, p. 542) reports that this bronchoconstriction is dose-related and is manifested as an increase in pulmonary flow resistance.

These acute effects, which may be severe, indicate the need for the 5 ppm STEL. (The Immediately Dangerous to Life or Health (IDLH) level for SO_2 is 100 ppm.) With a 2 ppm 8-hour TWA alone, a worker could be exposed to 100 ppm for 10 minutes or to 30 ppm (the concentration that produces severe respiratory symptoms (Kehoe Ex. 1–339; Skalpes Ex. 1–438)) for 20 minutes. The 15-minute 5 ppm STEL is thus necessary to prevent these effects as well as the bronchoconstriction that can develop in sensitive individuals from brief exposure to relatively low levels of SO_2. Acute bronchoconstrictive effects are precisely the kinds of effect STELs were designed to protect against.

Efforts have been made to quantify the acute no-adverse-effect level for SO_2-induced increased airway resistance. Frank, Amdur, Worcester, and Whittenberger (1962, as cited in ACGIH 1986/Ex. 1–3, p. 542) reported that, at SO_2 concentrations of 1 ppm, 1 in 11 healthy subjects developed pulmonary flow resistance; at concentrations of 5 or 13 ppm, there was a 39- and 72-percent increase, respectively, in such resistance. Weir, Stevens, and Bromberg (1972/Ex. 1–401) noted a statistically significant but reversible increase in small-airway resistance and a decrease in lung compliance at a concentration of 3 ppm; however, Burton et al. (1969) reported no effects, even among smokers, at a level of 2.1 ppm.

N.R. Frank, Professor of Medicine at the University of Washington State, commented during the 1977 hearing (NIOSH 1977m) that sulfur dioxide may not by itself be hazardous to the lungs but that an aerosol of sulfur dioxide and water or SO_2 oxidized to sulfate particulate may increase the toxic potential of SO_2 (Ex. 40, Docket H–039). Dr. Frank also presented evidence showing that a single short-term exposure to very high SO_2 levels (200 to 1000 ppm) can produce lung damage (Ex. 40, Docket H–039).

The lack of chronic effects observed in animals at levels below 5 ppm (Alarie, Ulrich, Busey et al. 1970 and 1972, as cited in ACGIH 1986/Ex. 1–3, p. 542) caused commenters to question whether chronic lung disease results from long-term exposure to SO_2 below the current 5 ppm PEL. Dr. Alarie appeared at the 1977 hearing and testified on animal studies conducted by him and others on sulfur dioxide (NIOSH 1977m, as cited in ACGIH 1986/Ex. 1–3, p. 542). He testified that, in his opinion, the long-term studies in animals support the establishment of a ceiling value for SO_2 but do not indicate that benefits would be gained by reducing the time-weighted average from 5 to 2 ppm. OSHA agrees with Dr. Alarie that a STEL is necessary to minimize high short-term exposures to SO_2; however, OSHA does not agree that no effects have been seen in animals at levels at or below 5 ppm. For example, Lewis, Campbell, and Vaughan (1969, as cited in ACGIH 1986/Ex. 1–3, p. 542) showed that beagles exposed to 5 ppm SO_2 exhibited decreased dynamic compliance and increased flow resistance. In addition, NIOSH (1974b/Ex. 1–235) has reported:

> [M]an is considered to be more sensitive than other mammals to the effects of sulfur dioxide in ranges commonly employed experimentally * * * (Ex. 1–235).

It is therefore not surprising that humans have also been shown to develop respiratory effects, including bronchoconstriction, coughing, and sputum production, at levels below 5 ppm (Smith, Peters, Reading, and Castle 1977/Ex. 1–805; Archer and Gillam 1978/Ex. 1–711; Frank, Amdur, Worcester, and Whittenburger 1962, as cited in ACGIH 1986/Ex. 1–3, p. 542; Weir, Stevens, and Bromburg 1972/Ex. 1–401).

In the prior rulemaking for general industry, several commenters (Exs. 3–1123, 8–57, 86, 86A, 117, 177, and 188) argued that the Ferris et al. study demonstrated a lack of chronic effects caused by exposure to pure SO_2. OSHA disagrees. First, the Ferris et al. study shows deleterious health effects at SO_2 levels down to 3 ppm that become progressively worse at 5 ppm. Second, other studies show adverse health effects in animals exposed to pure SO_2 at levels of 5 ppm. Third, SO_2 causes bronchoconstriction. OSHA therefore believes that a 2 ppm TWA is necessary to protect workers in construction, maritime, and agriculture from these significant risks, which are associated either with exposure to pure SO_2 or to SO_2 and particulates.

Commenters cited Rom et al. (1983) to the effect that pulmonary function does not decline as a result of exposure to SO_2. In 1983, Rom et al. conducted a follow-up study of Smith et al.'s cohort (which was studied originally in 1974). Rom et al. reported a statistically significant improvement in pulmonary function in workers in two of the groups expected to show the greatest decline. For the period 1980 to 1983, the mean annual changes across both groups were negative.

The investigator's explanation for the apparent increase in FVC in the 1973–1980 period is that the pulmonary function tests given by Smith et al. in 1973 were inadequate. However, they also suggest that the use of a more reliable dual-cartridge respirator in the new smelter may have reduced exposure to SO_2, leading to improved pulmonary function. Furthermore, the size of the study is quite small, especially once the cohort is stratified into smokers, ex-smokers, and

nonsmokers. Thus, the small size of this study may have made it difficult to observe any significant SO_2-related declines in pulmonary function.

A study by Holness et al. (1985) examined the change in pulmonary function in a group of nickel refinery workers both before and after shutdown of the smelter. Average SO_2 concentration in the smelter was 0.47 ppm. Pulmonary function was assessed twice during a workweek and again following a 6-month shutdown period during which no exposures to SO_2 or particulate occurred. Pulmonary function exams conducted on Monday showed that, after adjusting lung function values for the effects of smoking, smelter workers had significantly lower FVC and FEV values than office workers. On Thursday, smelter workers exhibited further declines in FVC and FEV_1, although the magnitude of the change in FEV_1 was not statistically significant. Area sampling for SO_2 and particulate suggested a direct relationship between the change in FVC and FEV_1 and exposure to SO_2 and particulate. In addition, the prevalence of respiratory symptoms (adjusted for smoking) was significantly higher among these smelter workers than office workers both prior to and after the shutdown period.

Taken together, the evidence from all of the studies described in this subsection clearly shows that exposure to SO_2 below 5 ppm (both in the pure state and together with particulates) does cause respiratory symptoms, including repeated episodes of bronchoconstriction. The studies by Smith, Peters, Reading, and Castle (1977/Ex. 1–373), Archer and Gillam (1978/Ex. 1–711), and Frank, Amdur, Worcester, and Whittenberger (1962, as cited in ACGIH 1986/Ex. 1–3, p. 542) consistently demonstrate that persons exposed to concentrations of SO_2 below 5 ppm have an accelerated loss of pulmonary function and exhibit adverse pulmonary symptoms, both acute and chronic. OSHA believes that these effects constitute material impairments of health and are significant.

The second point raised by commenters concerned the formation of other toxic and irritating products from the interaction between SO_2 and water or between SO_2 and particles. Some of the participants in the earlier rulemaking, such as Dr. Colucci of the Corn Refiners Association, testified that it would be more protective to identify and limit exposure to each of these by-products, rather than to regulate SO_2 alone. OSHA disagrees with this approach; since these products are all formed from sulfur dioxide, limiting exposure to SO_2 will concurrently limit exposure to these SO_2 by-products. This approach is more straightforward and easier to implement than attempting to identify the myriad decay products that may be formed in different industrial settings. Furthermore, the studies discussed above clearly establish a relationship between airborne SO_2 levels and adverse effects; no quantitative relationship on which to base a PEL has been established for the decay products of SO_2 reactions. Therefore, to reduce the significant risk of respiratory symptoms among exposed workers, OSHA finds that limiting exposure to SO_2 will be effective.

After considering all of the relevant evidence from both the 1977 and the present dockets, OSHA preliminarily concludes that an 8-hour TWA PEL of 2 ppm and a STEL of 5 ppm are necessary to reduce the significant risk of adverse respiratory effects that have been demonstrated to occur in workers exposed to SO_2 above these levels. The Agency believes that the coughing, increase in sputum production, bronchoconstriction, and significant declines in pulmonary function observed in workers exposed to SO_2 at the levels permitted by the current limit constitute material impairments of health and functional capacity, and must be protected against. Accordingly, OSHA is proposing these PELs for workers in construction, maritime, and agricultural workplaces. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

SULFUR TETRAFLUORIDE

CAS: 7783–60–0; Chemical Formula: SF_4

H.S. No. 1378

OSHA has no exposure limit for sulfur tetrafluoride in the construction, maritime, or agriculture industry. The ACGIH has a limit of 0.1 ppm as a ceiling; NIOSH has no REL for this substance. OSHA is proposing a PEL of 0.1 ppm as a ceiling; NIOSH (Ex. 8–47, Table N1) concurred with this limit when the Agency recently established it in general industry. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Sulfur tetrafluoride is a colorless, noncombustible gas. It is used as a fluorinating agent in the production of water and oil repellents and lubricity improvers. Sulfur tetrafluoride is also used in making pesticides (Hawley's 1987, p. 1109; New Jersey Fact Sheet 1986, p. 1).

On contact with moisture, sulfur tetrafluoride produces sulfur dioxide and hydrogen fluoride (HF) (Lester 1971, as cited in ACGIH 1986/Ex. 1–3, p. 546), and it is the release of HF that is primarily responsible for sulfur tetrafluoride's toxic effects (Zapp 1971, as cited in ACGIH 1986/Ex. 1–3, p. 546). A du Pont study (1961, as cited in ACGIH 1986/Ex. 1–3, p. 546) of rats exposed for four hours to 4 ppm sulfur tetrafluoride over a period of 10-days reported that the animals demonstrated nasal discharge, difficulty in breathing, and weakness. Autopsies of these animals revealed evidence of emphysema, but those rats surviving exposure and given a two-week rest period after exposure showed no significant pathological changes. In the same study by du Pont (1961, as cited in ACGIH 1986/Ex. 1–3, p. 546), a four-hour exposure to 20 ppm sulfur tetrafluoride proved lethal to one of two rats. In a study by Clayton (1962/Ex. 1–409), irregular breathing and signs of irritation were observed following exposures to concentrations of 20 ppm and lower; animals receiving lethal amounts of sulfur tetrafluoride showed pulmonary edema on autopsy, and those with sublethal exposures demonstrated no pathologic changes 14 days later.

OSHA is proposing a 0.1-ppm ceiling limit for this highly toxic gas in the construction, maritime, and agriculture industries. The Agency believes that establishing this limit for this previously unregulated chemical will reduce the significant risk of chronic respiratory effects potentially associated with exposure to sulfur tetrafluoride at the levels permitted in these sectors by the absence of any OSHA limit. OSHA considers the chronic respiratory effects caused by exposure to sulfur tetrafluoride to be material impairments of health. In addition, promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

TALC (Containing no asbestos)

CAS: 14807–96–6; Chemical Formula: $Mg_3Si_4O_{10}(OH)_2$

H.S. No. 1381

The current OSHA PEL for nonasbestiform talc (containing less than 1 percent crystalline silica) in the construction and maritime industries is 20 million particles per cubic foot of air (mppcf) as an 8-hour TWA; when expressed as mg/m^3, this is comparable to 3 mg/m^3. There is no limit in agriculture. The ACGIH has a $TLV^®$–TWA of 2 mg/m^3 (15 mppcf) for talc, measured as respirable dust, and this is the limit being proposed by OSHA for construction, maritime, and agriculture operations. NIOSH has no REL for this

substance, but concurred (Ex. 8–47, Table N1) that this limit was appropriate when the Agency established it in general industry.

Talc is a fine powder that is white to gray-white in color; it is found as a mineral, and the main component is a crystalline hydrated silicate of magnesium that is usually in the form of plates but occasionally may be in the form of fibers. Talc is used in industrial products and cosmetics. Talc is also used in insecticides, ceramics, paints, paper, plastics, rubber, and roofing (ACGIH 1986, p. 550).

The health-effects evidence for talc is complicated by the fact that talcs contain amphiboles and other minerals, in addition to platiform talc crystals; adverse health effects appear to be related to the nonplatiform content (that is, to the fiber content) of the talc in question (ACGIH 1986/Ex. 1–3, p. 550). There are conflicting views regarding the extent to which the fibrous constituents are asbestos; however, no health effects information is available that is specifically related to fibrous talc (ACGIH 1986/Ex. 1–3, p. 550).

Numerous epidemiological studies have documented the effects on workers of long-term exposures to talc. In 1942, Porro et al. (1942, as cited in Stokinger 1981b/Ex. 1–1127) published a report in which 15 cases of talc pneumoconiosis, including five postmortem examinations, showed that asbestotic bodies were almost always present in fibrotic areas of the lungs of those workers with talcosis. Siegal and colleagues (1943, as cited in Stokinger 1981b/Ex. 1–1127) noted that the incidence of advanced fibrosis in a group of 221 talc miners and millers was 14.5 percent. These workers were primarily exposed to fibrous talc, which was believed to be responsible for the pathology of the asbestos-like lung lesions. A study by McLaughlin et al. (1949, as cited in Stokinger 1981b/Ex. 1–1127) revealed that talc-induced pneumoconiosis was caused by the fibrous varieties of talc; in animal studies by Schepers and Durkan (1955, as cited in Stokinger 1981b/Ex. 1–1127), the degree of fibrosis in the lung tissue was found to be a function of the length of the talc fibers, rather than of the composition of the talc itself. A paper by Kleinfeld, Giel, Majeranowski, and Messite (1963, as cited in Stokinger 1981b/Ex. 1–1127) reported that postmortem examinations on six talc industry workers showed that the asbestotic bodies found in the lung bronchioles or embedded in fibrous tissue were indistinguishable from the asbestos bodies seen in cases of asbestosis.

Kleinfeld, Messite, Kooyman, and Zaki (1967/Ex. 1–704) later conducted a cohort study of 220 workers who had been employed in a mine that produced talc that had a tremolite and anthophyllite content. Of the 91 deaths in this group, 10 resulted from respiratory cancer and 28 were attributed to pneumoconiosis. The proportional mortality rate from respiratory cancer was four times the expected rate. In 1974, when Kleinfeld, Messite, and Zaki (Ex. 1–705) performed a follow-up study of this group (which at that time consisted of 260 workers [108 deaths]), they found significant differences between the expected and observed mortality in the period 1950 to 1954, but not during 1960 to 1969. These investigators attributed this finding to the reduction in talc dust counts from averages of 25 to 73 mppcf (approximately 4 to 12 mg/m^3) in the years between 1948 and 1965 to averages of 9 to 43 mppcf (approximately 1.5 to 6.5 mg/m^3) in the period 1966 to 1969. This study also showed a decrease of greater than 50 percent in deaths due to pneumoconiosis in the 1965-to-1969 time period.

Studies by NIOSH (Dement and Zumwald 1978, as cited in ACGIH 1986/Ex. 1–3, p. 552) of 398 white male workers employed between 1947 and 1959 in the talc industries found that 74 of these men had died, and that bronchogenic cancer was the cause of death in nine men; only 3.3 deaths from this cause would have been expected. Nonmalignant respiratory disease (NMRD) exclusive of influenza, pneumonia, and tuberculosis accounted for three deaths; 1.5 would have been expected. From these data, NIOSH concluded that a significant increase in mortality due to bronchogenic cancer and NMRD had occurred as a result of occupational exposure to talc dust. NIOSH's report also included a morbidity study of 12 talc industry workers, currently employed, in which chest x-rays, lung function tests, and questionnaires were used. This study concluded that a higher prevalence of cough, phlegm, dyspnea, and irregular opacities in chest x-rays existed in these workers than in potash miners; instances of pleural thickening and calcification were greater than in coal and potash miners; and the pulmonary function of talc workers overall was reduced in comparison with that of coal and potash miners employed for the same length of time. The reductions in pulmonary function among the talc workers were dose- and duration-related.

The ACGIH (1986/Ex. 1–3, p. 552) concluded that serious health effects have been associated in the past (i.e., prior to 1945) with exposures to amphibole-containing talc. However, the ACGIH believes that the introduction of mining improvements has all but eliminated "the excess of death rates from pneumoconiosis and lung cancer" (ACGIH 1986/Ex. 1–3, p. 552).

Two recent studies of the health effects associated with talc exposures (Rubino, Scansetti, Piolatto, and Romano 1976/Ex. 1–801; Selevan, Dement, Wagoner, and Froines 1979/Ex. 1–989) are available. The Rubino, Scansetti, Piolatto, and Romano (1976/Ex. 1–801) study found that miners and millers exposed to an average of 849 to 8470 mppcf-years (miners) or 76 to 651 mppcf-years (millers) showed no increase in the number of observed (compared to expected) deaths from causes other than silicosis. These authors concluded that the disease-causing factor in these workers was silica rather than talc (Rubino, Scansetti, Piolatto, and Romano 1976/Ex. 1–801).

The Selevan, Dement, Wagoner, and Froines (1979/Ex. 1–989) study of 392 workers exposed to talc in five mines found nonmalignant respiratory deaths for millers to be almost eight times the expected rate, while miners experienced more than three times the expected mortality rate for NMRD. The ACGIH (1986/Ex. 1–3, p. 552) believed that the Selevan et al. (1979/Ex. 1–989) study is incomplete because confounding factors were not adequately identified and controlled for.

With regard to NIOSH's findings (Dement and Zumwald 1978), of excess cancer deaths among talc workers, OSHA is currently reviewing the scientific and toxicological data describing the effects of exposure to the nonasbestiform varieties of mineral fibers that are found in talc deposits. OSHA is considering a separate rulemaking to address this issue.

OSHA is proposing an 8-hour TWA limit of 2 mg/m^3 for the respirable dust of talc containing no asbestos fibers and less than 1 percent crystalline silica in the construction, maritime, and agriculture industries. The Agency believes that this limit will protect workers in these sectors from the significant risk of nonmalignant respiratory effects associated with exposure to talc dust; OSHA considers these effects material impairments of health. In addition, promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors. According to the ACGIH (1986/Ex. 1–3), talc may, at

times, occur in a fibrous form. At this time, OSHA has not made any preliminary determinations with regard to the possible health consequences resulting from exposure to talc fibers.

TIN OXIDE

CAS: 7440–31–5; Chemical Formula: SnO

H.S. No. 1395

OSHA has no exposure limit for tin oxide in the construction, maritime, or agriculture industry. The ACGIH has a TLV® of 2 mg/m³ as an 8-hour TWA. NIOSH has no REL for tin oxide. The Agency is proposing a PEL of 2 mg/m³ as an 8-hour TWA for tin oxide in construction, maritime, and agriculture; NIOSH concurred (Ex. 8–47, Table N1) with this limit when the Agency recently established it in general industry. Promulgation of this limit will make the PEL for tin oxide consistent across all OSHA-regulated sectors.

Tin oxide may be a white or yellow-brown powder. It is used as a reducing agent, an intermediate in the preparation of stannous salts used in the plating and glass industries, and as an ingredient in pharmaceuticals and soft abrasives (Hawley's 1987, p. 1089).

Injection of tin dust intraperitoneally into guinea pigs resulted in a nonspecific, well-vascularized chronic granulomatous reaction (Oyanguren, Haddad, and Maass 1958/Ex. 1–652). Chronic exposure to tin oxide fume and dust results in stannosis, a form of pneumoconiosis. The fume of tin oxide is considered to be a more important source of stannosis than the dust (Dundon and Hughes 1950/Ex. 1–732), but other authorities consider the quality of the dust and the duration of exposure equally important (Robertson and Whittaker 1955/Ex. 1–987). The onset of the symptoms of stannosis may be delayed for years; the appearance of the condition is signalled by difficulty in breathing. One worker who had been exposed to unspecified tin oxide levels for 22 years was tested for stannosis and registered a vital breathing capacity 70 percent of normal and a maximal breathing capacity 61 percent of the predicted value (Spencer and Wycoff 1954/Ex. 1–611). More than 150 cases of stannosis have been reported in the world literature (Robertson and Whittaker 1955/Ex. 1–987), and five cases were reported in the United States before 1954. No cases of massive fibrosis caused by exposure to tin oxide dust or fume have been reported (ACGIH 1986/Ex. 1–3, p. 574).

OSHA is proposing an 8-hour TWA PEL of 2 mg/m³ for tin oxide dust and fume in the construction, maritime, and agriculture industries. The Agency believes that this limit will protect workers in these sectors, from the significant risks of reduced pulmonary capacity and other signs and symptoms of stannosis, which are considered material impairments of health that are associated with exposure to this substance. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

TRIMELLITIC ANHYDRIDE

CAS: 552–30–7; Chemical Formula: $C_9H_4O_5$

H.S. No. 1409

OSHA has no exposure limit for trimellitic anhydride in the construction, maritime, or agriculture industry. In 1981, the ACGIH set 0.005 ppm (0.04 mg/m³) as the 8-hour TLV®–TWA limit for this substance. NIOSH has no REL for trimellitic anhydride. The Agency is proposing a 0.005 ppm 8-hour TWA limit for this substance in the construction, maritime, and agriculture industries. NIOSH concurred (Ex. 8–47, Table N1) with this limit when the Agency recently established it in general industry. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Trimellitic anhydride is a colorless solid. It is used in agricultural chemicals, dyes and pigments, paints and coatings, pharmaceuticals, and surface active agents. Trimellitic anhydride is also employed as a curing agent for epoxy and other resins and as an ingredient in vinyl chloride plasticizers and various polymers and polyesters (ACGIH 1986, p. 606).

Exposure to trimellitic anhydride (TMAN) causes irritation of the eyes, nose, skin, and pulmonary tract. NIOSH (1978n, as cited in ACGIH 1986/Ex. 1–3, p. 606) reported in a current intelligence bulletin that trimellitic anhydride should be considered an extremely toxic workplace hazard, because exposure to it can cause noncardiac pulmonary edema and immunological sensitization, as well as upper respiratory tract irritation.

Pulmonary edema has occurred in workers exposed to TMAN at unreported air concentrations; the development of pulmonary edema in these workers without upper respiratory tract irritation suggests that TMAN is a sensitizer (Rice, Jenkins, Gray, and Greenburg 1977/Ex. 1–358). Zeiss, Patterson, Pruzansky, and colleagues (1977/Ex. 1–501) described TMAN-related illnesses among a group of workers synthesizing this substance. These authors believe that there are three separate syndromes associated with TMAN exposure: rhinitis/asthma; a flu-like condition; and irritation of the upper respiratory tract. Another case of TMAN-related occupational sensitization occurred in a worker exposed during the application of an epoxy resin coating (Fawcett, Taylor, and Pepys 1977/Ex. 1–636).

At TMAN concentrations averaging 1.5 and 2.8 mg/m³ in two processes, NIOSH reported that employees reported eye and nose irritation, shortness of breath, coughing, nausea, headache, skin irritation, and throat irritation (NIOSH 1974c/Ex. 1–1181). Pulmonary hemorrhage and hemolytic anemia have been reported in workers exposed to TMAN at unspecified levels (Ahmad, Morgan, Patterson et al. 1979/Ex. 1–460).

Rats have shown intraalveolar hemorrhage after TMAN exposures to concentrations of 0.01 ppm (Amoco Chemical Corporation 1978, as cited in ACGIH 1986/Ex. 1–3, p. 606).

Based on this study, OSHA is proposing to set the PEL in construction, maritime, and agriculture at an 8-hour TWA level of 0.005 ppm. The Agency believes that this limit will protect workers from the severe pulmonary effects, sensitization, and skin and upper respiratory tract irritation observed in workers exposed to this extremely toxic substance. The Agency believes that these effects clearly constitute material impairments of health and functional capacity within the meaning of the Act. OSHA preliminarily concludes that this limit is necessary to substantially reduce these significant risks, which were formerly uncontrolled among workers in these sectors. In addition, promulgation of this limit will make the PEL for trimellitic anhydride consistent across all OSHA-regulated sectors.

WOOD DUST

CAS: None; Chemical Formula: None

H.S. No. 1430A (Hard Wood)

H.S. No. 1430B (Soft Wood)

H.S. No. 1430C (Western Red Cedar)

Before 1980, OSHA regulated wood dust under its nuisance dust standard of 15 mg/m³ (29 CFR 1910.1000, Table Z–3). However, in a 1985 enforcement proceeding before the Occupational Safety and Health Review Commission, wood dust was held not to be covered by the nuisance dust standard, and the Agency did not regulate this substance after this decision (12 OSHC 1785). Consequently, OSHA has no current PELs for wood dust in the construction, maritime, and agriculture industries, although the prior air contaminants standard did establish PELs for wood dust in general industry. The ACGIH has a TLV®–TWA of 1 mg/m³ for hard

wood dust, and a TLV®–TWA of 5 mg/m³ and STEL of 10 mg/m³ for soft wood dust. The ACGIH has no limit for Western Red Cedar. NIOSH has no RELs for soft- and hardwood dusts or Western Red Cedar dust in construction, maritime, and agriculture. OSHA is proposing a single 8-hour TWA of 5 mg/m³ and a STEL of 10 mg/m³ for all hard wood and soft wood dusts except Western red cedar. For Western red cedar, a highly allergenic species of soft wood, the Agency is proposing an 8-hour TWA limit of 2.5 mg/m³. These are the limits recently established for these substances in general industry. Promulgation of these limits will make the PELs for these substances consistent across all OSHA-regulated sectors.

Wood dust is defined as any wood particles arising from the processing or handling of woods. Hard woods derive from the deciduous broad-leaved flowering species of trees, and soft woods include the coniferous species that do not shed their leaves in the winter. Wood dust is produced during sawmill operations and in wood sawing and sanding procedures in furniture manufacture. Wood dust may be used as an industrial wood flour product, as an absorbent for nitroglycerin in the manufacture of dynamite, and as a filler in plastics, linoleum, paperboard, fur cleaning, and polishing agents (Sittig 1985, p. 930; Hawley's 1987, p. 1238).

Exposure to wood dust has long been associated with a variety of adverse health effects, including dermatitis, allergic respiratory effects, mucosal and nonallergic respiratory effects, and cancer. The toxicity data in animals are limited, particularly with regard to exposure to wood dust alone; there are, however, a large number of studies in humans. The discussion below first describes some of the relevant toxicological studies and then presents the record evidence on wood dust.

Animal Studies

Groups of male guinea pigs were injected intratracheally with suspensions containing 75 mg of sheesham or mango wood dust or of hemp or bagasse fibers, or 20 mg of jute fiber (Bhattacharjee, Dogra, Lal, and Zaidi 1979/Ex. 1–463; Bhattacharjee and Zaidi 1982/Ex. 1–464). Animals were sacrificed serially at intervals up to 90 days after injection. Lung examination revealed that, at 90 days, Grade I fibrosis of the lungs had occurred in the guinea pigs injected with mango or jute, while those treated with sheesham or hemp had developed Grade II pulmonary fibrosis.

In another experiment involving guinea pigs, animals were exposed by inhalation to average respirable dust concentrations of 1143 mg/m³ for 30 minutes/day, 5 days/week for 24 weeks (McMichael, DiPalma, Blumenstein et al. 1983, Ex. 1–644). Histopathological examination showed lung changes, described by the authors as moderate to severe, in all exposed guinea pigs. The changes seen included an increase in septal connective tissue components and aggregation of lymphocytes; however, no pulmonary fibrosis or extensive destruction of the parenchymal tissue occurred. The authors of this study concluded that exposure to fir bark dust may cause inflammatory changes in the lung.

Two studies examined the effect of exposing Syrian golden hamsters to beech wood dust by inhalation, with or without concurrent administration of the known carcinogen diethylnitrosamine (DEN) (Wilhelmsson, Hellquist, Olofsson, and Klintenberg 1985/Ex. 1–402; Wilhelmsson, Jernudd, Ripe, and Holmberg 1985/Ex. 1–1042; Drettner, Wilhelmsson and Lundh 1985/Ex. 1–312). In each study, the animals were divided into four separate groups. In Study I, there were 12 animals per group. Two groups were exposed to fresh beech wood dust (a hard wood dust) at a mean total dust concentration of 15 mg/m³ for six hours/day, five days/week for 36 weeks, and one of these groups was also given 1.5 mg of DEN once a week for the first 12 weeks. The third group in Study I was given the DEN doses only (positive control), and the fourth group was given no exposure at all (negative control).

In Study II, there were 24 animals in each of four groups. Two groups of animals were exposed to fresh beech wood dust at a mean total dust concentration of 30 mg/m³ for six hours/day, five days/week for 40 weeks. The positive and negative control groups were treated as in Study I.

In Study I, none of the hamsters had lung or nasal tumors or metaplasia. Four hamsters exposed to wood dust and DEN exhibited squamous cell papillomas of the trachea, as did three animals in the positive control group and one in the negative control group. No differences in organs other than the respiratory organs were seen between the treated and control groups in Study I.

In Study II, all DEN-exposed hamsters had nasal lesions ranging from hyperplasias and dysplasias to papillomas. In addition, half of all DEN-exposed hamsters developed nasal adenocarcinomas, whether or not they had also been exposed to wood dust. Half of the DEN-exposed animals also had papillomas of the larynx and trachea. In the wood-dust-exposure-only group, two of the animals had nasal lesions, one of which was an unclassifiable malignant nasal tumor and the other of which consisted of focal metaplasia with mild dysplasia. The authors concluded that exposure to wood dust did not increase the tumor incidence in DEN-exposed animals but did affect the respiratory tract of all exposed animals.

Human Studies

Dermatitis

There are a large number of case reports, epidemiological studies, and other data on the health effects of wood dust exposure in humans. Dermatitis caused by exposure to wood dusts is common, and can be caused either by chemical irritation, sensitization (allergic reaction), or both of these together. As many as 300 species of trees have been implicated in wood-caused dermatitis.

The chemicals associated with allergic reactions are generally found in the inner parts of a tree, e.g., the heartwood, and the workers most prone to these reactions are those involved in secondary wood processing (e.g., carpenters, joiners, and finishers).

The symptoms of sensitization are redness, scaling, and itching, which may progress to vesicular dermatitis and, after repeated exposures, to chronic dermatitis. The parts of the body most often affected are the hands, forearms, eyelids, face, neck, and genitals. This form of dermatitis generally appears after a few days or weeks of contact.

Allergic Respiratory Effects

Allergic respiratory responses are mediated by the immune system, as is also the case with allergic dermatitis. Many authors have reported cases of allergic reactions in workers exposed to wood dust (Sosman, Schlueter, Fink, and Barboriak 1969/Ex. 1–444; Greenberg 1972/Ex. 1–482; Pickering, Batten, and Pepys 1972/Ex. 1–655; Eaton 1973/Ex. 1–478; Booth, LeFoldt, and Moffitt 1976/Ex. 1–466; Chan-Yeung, Ashley, Corey et al. 1978/Ex. 1–622; Edwards, Brooks, Henderson, and Apol 1978/Ex. 1–950; Innocenti and Angotzi 1980/Ex. 1–1036; Bush and Clayton 1983/Ex. 1–469; Cartier, Chan, Malo et al. 1986/Ex. 1–472). Asthma is the most common response to wood dust exposure, and the allergic nature of such reactions has been demonstrated by the presence of IgE antibodies and positive skin reactions on patch testing. The best-studied of the allergic reactions to wood dust is Western red cedar (WRC)

asthma; it is estimated that 5 percent of the workers handling this species are allergic to it. However, only one study is available that relates exposure level to ventilatory function. In that study, exposure to concentrations of 2 mg/m³ of WRC dust caused significant decreases in forced vital capacity and forced expiratory volume (Vedal, Chan-Yeung, Enarson et al. 1986/Ex. 1–397). These authors also found that exposures to concentrations above 3 mg/m³ produced eye irritation.

Mucosal and Nonallergic Respiratory Effects

This section discusses changes in the structure and function of the nasal mucosa and respiratory tract that are caused by exposure to wood dust. These changes include nasal dryness, irritation, bleeding, and obstruction; coughing, wheezing, and sneezing; sinusitis; and prolonged colds. These symptoms have been observed even at wood dust concentrations below 4 mg/m³.

Bellion, Mattei, and Treves (1964, as cited in NIOSH 1987a/Ex. 1–1005) found that 97 of 225 workers (carpenters, sawmill workers, woodworkers) exposed from 3 to 24 years to the dust of several different hard woods showed radiologic evidence of pulmonary abnormalities. Black, Evans, Hadfield et al. (1974/Ex. 1–299) studied nine woodworkers from a woodworking factory in England. In all of these workers, mucociliary movement was markedly depressed, leading these authors to conclude that exposure to wood dust in the furniture industry for 10 years or more can impair mucociliary clearance. These findings were confirmed in a Danish study involving furniture makers (Solgaard and Andersen 1975/Ex. 1–443; Andersen, Solgaard, and Andersen 1976/Ex. 1–297; Andersen, Andersen, and Solgaard 1977/Ex. 1–296); compared with controls, the mucociliary transport rate was also significantly impaired in these woodworkers, and dose-response effects were noted.

A respiratory survey conducted by Chan-Yeung, Giclas, and Henson (1980/Ex. 1–474) in pulp and paper mill workers in British Columbia showed that workers exposed to wood dust at a mean total dust concentration of 0.5 mg/m³ had a slight but statistically significant decrease in pulmonary function values compared with controls. The authors concluded that the chemical preservatives used to treat the wood could also have been responsible for these adverse effects.

In a cross-sectional survey of 1,157 American woodworkers (both hard and soft wood), Whitehead, Ashikaga, and Vacek (1981/Ex. 1–454) found that exposure to higher (10+ mg-years/m³), as compared with lower (0 to 2 mg-years/m³), dust concentrations was associated with a statistically significant and higher incidence of decreased pulmonary function. However, dose-response effects were observed only for soft wood (i.e., pine) dusts. A later study by Beckman, Ashikaga, and Whitehead (1980, as cited in NIOSH 1987a/Ex. 1–1005) examined subgroups of the workers studied by Whitehead and found no correlation between years of exposure to pine wood dust and pulmonary function.

In a pilot study of 55 workers in a North Carolina hardwood furniture plant, Goldsmith (1983, as cited in NIOSH 1987a/Ex. 1–1005) found that, at mean area wood dust concentrations of 2 mg/m³ or below, peak ventilatory flow correlated significantly with cumulative person-years of exposure. Goldsmith interpreted this finding to mean that inhalation of wood dust may impair large-airway function.

A study of Italian woodworkers showed that the number of wood-dust-exposed workers who had developed anosmia (loss of smell) was significantly higher than in a control group of nonexposed workers (Innocenti, Valiani, Vessio et al. 1985/Ex. 1–1037). Amoore (1986/Ex. 1–1029) confirmed this finding in other workers exposed to hardwood dusts.

Summary of mucosal and nonallergic respiratory effects. A large number of studies have demonstrated that occupational exposure to wood dust causes both statistically significant and nonsignificant increases in respiratory symptoms at exposure levels as low as 2 mg/m³. These symptoms range from irritation to bleeding, wheezing, sinusitis, and prolonged colds. In addition, chronic wood dust exposure causes mucociliary stasis (i.e., the absence of effective clearance) in the nose and, in some workers, also causes changes in the nasal mucosa. Several studies have demonstrated decreased pulmonary function among wood-dust-exposed workers, although other studies have not confirmed these findings.

Carcinogenicity

The association between occupational exposure to wood dust and various forms of cancer has been explored in many studies and in many countries. In 1987, the International Agency for Research on Cancer (IARC) classified furniture manufacturing in Category I (confirmed human carcinogen) and carpentry in Category 2B (suspected human carcinogen). NIOSH (Ex. 8–47) considers both hard and soft wood dust to be potentially carcinogenic in humans; for soft wood dust, NIOSH recommends a separate 6(b) rulemaking (Ex. 8–47, Table N6B). NIOSH concurred, however, with the proposed PEL of 1 mg/m³ TWA for hard wood dust (Ex. 8–47, Table N6A).

The discussion below focuses on selected U.S. studies.

Nasal and sinus cavity cancer. The earliest U.S. study of wood dust exposure and nasal cancer was conducted by Brinton, Stone, Blot, and Fraumeni (Ex. 1–468) in 1976. These authors analyzed cancer death rates between 1950 and 1969 in 132 U.S. counties having at least 1 percent of their population employed in furniture and wood-fixture manufacturing. This study revealed that the age-adjusted mortality rate for cancer of the nasal cavity and sinuses among white males in the "furniture" counties was significantly higher than in nonfurniture counties.

In a later case-control study, these authors (Brinton, Blot, Becker et al. 1984/Ex. 1–467) analyzed cases of nasal and sinus cancers occurring in North Carolina and Virginia between 1970 and 1980. This study identified a significantly elevated risk of adenocarcinomas in males working in the furniture manufacturing industry, but no increased risk among lumber, carpentry, or construction workers. There was no significant increase in the risk of squamous cell carcinoma in workers from any other wood-related industry.

In a study sponsored by the Inter-Industry Wood Dust Task Force, Viren, Vogt, and Dixon (1982, as cited in NIOSH 1987a/Ex. 1–1005) described a death certificate case-control study of nasal cancer deaths for 1963 to 1977 in North Carolina, Mississippi, Washington, and Oregon. Findings of this study included a relative nasal cancer risk of 1.95 for industries involving lumber and wood products; however, no significant relative risk of nasal cancer was seen for workers in the furniture-manufacturing industry.

Imbus and Dyson conducted a study of nasal cancer and North Carolina furniture workers (1985, as cited in NIOSH 1987a/Ex. 1–1005). This study found: (1) That there was a statistically significant increase of nasal cancer among furniture workers; (2) that the nasal cancer rates among North Carolina furniture workers were much lower than those reported for English furniture workers; (3) that the number of nasal cancer deaths among North Carolina furniture workers decreased

between 1956 and 1977; and (4) that a slight excess in nasal cancer may have existed among North Carolina furniture workers but is currently either declining or nonexistent.

At present, the National Cancer Institute is conducting a cohort mortality study of 36,622 workers employed in the wood, metal, and plastic furniture manufacturing industries (Miller et al. 1988, as cited in NIOSH 1987a/Ex. 1–1005). Results are too preliminary to be described at this time.

Summary of evidence for nasal and sinus cavity cancers. NIOSH (1987a/Ex. 1–1005) concluded that the literature clearly demonstrates an association between occupational wood dust exposure and nasal cancer. English studies first identified this link by showing a 10- to 20-times-greater incidence of nasal adenocarcinoma among woodworkers in the furniture industry than among other woodworkers and 100 times greater than in the general population. In the United States, three studies have reported a fourfold risk of nasal cancer or adenocarcinoma in furniture workers, and another study noted a similar relationship between nasal cancer and wood dust exposure. One other study failed to find such an association for furniture workers, but did find an increase among logging and timber industry workers.

Pulmonary cancer. A number of studies investigating the association between wood dust exposure and the development of lung cancer have been conducted. Milham (1974/Ex. 1–943) found a significant excess of malignant tumors of the bronchus and lung in workers who had belonged to the AFL–CIO United Brotherhood of Carpenters and Joiners of America. Only construction workers showed a statistically significant increase in lung cancer rate.

In a study of lung cancer in Florida residents, Blot, Davies, Brown et al. (1982/Ex. 1–465) found that an elevated risk of lung cancer that was statistically significant existed among workers in the lumber and wood industry and in construction; however, smoking may have been a confounding factor in these results.

Summary of evidence for pulmonary cancer. The association between lung cancer and occupational wood dust exposure is inconclusive, although several epidemiological studies have reported increases in lung cancer among wood-dust-exposed workers.

Hodgkin's disease. The data on the relationship between exposure to wood dust and the development of Hodgkin's disease are conflicting. Milham (1967/Ex. 1–750) and Milham and Hesser (1987/Ex. 1–645) concluded, on the basis of a case-cohort study of 1,549 white males dying of this disease between 1940–1953 and 1957–1964, that there was an association between Hodgkin's disease and exposure to wood dust.

Another study (Spiers 1989/Ex. 1–445) concluded that men working in the wood industries in the eastern United States were at special risk for Hodgkin's disease, and suggested that pine pollen exposure might be responsible for the increase.

A Washington State epidemiological study (Petersen and Milham 1974/Ex. 1–654) also found that woodworkers had an increased risk of Hodgkin's disease, and the work of these authors was supported by the results of another study (Grufferman, Duong, and Cole 1976/Ex. 1–484), which showed a nonsignificant increase in the relative risk for Hodgkin's disease among woodworkers.

Summary of evidence for Hodgkin's disease. Although the data are conflicting, several epidemiological studies of U.S. workers do report increases in the incidence of Hodgkin's disease among woodworkers. This excess is particularly apparent among carpenters.

Other cancers. NIOSH (1987a/Ex. 1–1005) concluded that the data on the relationship between occupational exposure to wood dust and the development of cancers other than nasal, Hodgkin's disease, or lung cancers are insufficient and inconclusive.

After a review of the record in the prior rulemaking, OSHA has determined that the health evidence for the toxicity of wood dust cannot be separately distinguished for soft wood and hard wood. In addition, the Agency was convinced in that rulemaking that most operations involve both kinds of wood and are performed on the same machines and equipment and in the same facility. Thus, any controls installed to reduce exposures would of necessity need to be sufficient to reduce airborne dust levels to the lower of the two limits (i.e., to the proposed wood dust limit of 1 mg/m³).

Some commenters involved in the earlier rulemaking found fault with several of these studies on the grounds that they involved British or other non-U.S. woodworkers (see, for example, Exs. 8–34, 191, 3–628, and 3–917), involved only a small number of subjects (see, for example, Exs. 8–34, 168, and 191), had inconsistent results (see, for example, Ex. 8–34), or failed to demonstrate a dose-response relationship between wood dust exposure and the health effect of concern (see, for example, Exs. 8–34, 3–628, 3–917, and 191). The Inter-Industry Wood Dust Coordinating Committee (IWDCC) stated:

> [T]he observations in the European studies are not representative of conditions in U.S. workplaces, especially under modern conditions * * *.
>
> The English and other European experience does not provide an accurate predictive model for the incidence of nasal cancer. * * * The excesses of nasal cancer observed in the European studies simply have not been observed in the United States at any time * * * (Ex. 3–748, pp. 2, 52).

OSHA agrees with the IWDCC that the incidence of nasal cancer seen in the United States is substantially lower than that seen in other countries, particularly in Great Britain. However, the Agency does not agree that excesses in nasal cancers, and particularly of nasal adenocarcinomas, have not been observed in American woodworkers. Several U.S. studies have reported excesses in nasal cancer risks among employees in the wood industries (Brinton, Stone, Blot, and Fraumeni 1976/Ex. 1–468; Brinton, Blot, Becker et al. 1984/Ex. 1–467; Viren, Vogt, and Dixon 1982, and Imbus and Dyson 1985, both as cited in NIOSH 1987a/Ex. 1–1005).

In response to those commenters who argued that none of the studies described by OSHA presented sufficient dose-response data to be used as a basis for establishing a limit, the Agency emphasized that it was not relying on any single study to determine that wood dust presents a significant risk of material health impairment. Instead, OSHA made this determination on the basis of the findings in the dozens of studies reporting on the respiratory, irritant, allergic, and carcinogenic properties of wood dust. The Agency preliminarily finds the results of these studies biologically plausible and their findings reproducible and consistent. It is true that some of these studies, like all human studies, have limitations of sample size, involve confounding exposures, have exposure measurement problems, and often do not produce the kind of dose-response data that can be obtained when experimental animals are subjected to controlled laboratory conditions. What the large group of studies being relied upon by OSHA to establish the significance of the risk associated with exposure to wood dust do show is that the overall weight of evidence that such exposures are harmful and cause loss of functional capacity and material impairment of health is convincing beyond a reasonable doubt.

The Agency preliminarily finds that the health evidence clearly indicates that occupational exposure to wood dust poses a significant risk of material health impairment at the 10-mg/m³ (or particulate PEL) level. OSHA therefore believes that establishing an 8-hour PEL of 5 mg/m³ and a 15-minute STEL of 10 mg/m³ in construction, maritime, and agriculture for all wood dusts (except Western red cedar) will substantially reduce this significant risk. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

For construction, maritime, and agriculture, OSHA proposes an 8-hour TWA PEL of 2.5 mg/m³ for Western red cedar wood dust, based on its widely recognized ability to cause immune-system-mediated allergic sensitization. A study by Brooks, Edwards, Apol, and Edwards (1980) that was submitted by the United Petitioners (Ex. 82D) reports that

> a high prevalence of occupational asthma was observed among workers exposed to WRC wood dust (Ex. 82D, p. 315).

At the hearing, Dr. Brooks described occupational asthma as follows:

> [T]here are spasms of the bronchial tubes, there is reduced air flow on expiration * * * [the extent of which depends] on the extent of the exposure, and also * * * on the duration of the exposure * * * as a consequence of this sensitization and airway injury from the sensitization and the, asthmatic reaction and the various biochemical and cellular changes that occur, there develops an associated process * * * the airways develop an increased sensitivity and an increased broncho-spastic responsiveness to many different non-specific stimuli. So such things as cold air, dust, fumes, gases that are non-specific and wouldn't normally * * * [affect] most individuals [will affect] the individual with occupational asthma. And it's [such] hyper-reactive airways that cause individuals to continue to have disability and to continue to have symptoms once they leave the work place * * *. They develop this nonspecific bronchial hyper-reactivity which may last the rest of their life (Tr. pp. 12–339 to 12–343).

The 1980 study by Brooks, Edwards, Apol, and Edwards found a dose-related relationship between total WRC dust level and prevalence of asthma in employees in jobs with the greatest dust exposures. The Brooks et al. study found asthma in zero percent of WRC workers exposed at 0.5 mg/m³; however, at 3.56 mg/m³, this percentage rose to 5 percent.

In the prior rulemaking, the United Petitioners submitted a 1988 paper by Goldsmith and Shy that found that there is a clearly defined asthma syndrome produced by WRC (Ex. 3–362). OSHA preliminarily finds these studies convincing evidence of WRC's allergenic potential; in addition, the Agency believes that a threshold for occupational asthma exists and lies between 2 and 3.4 mg/m³. Based on this evidence, OSHA preliminarily concludes that an 8-hour PEL of 2.5 mg/m³ is necessary to protect workers from the significant and often permanent effects of immune-mediated occupational asthma associated with exposure to WRC dust at levels above this limit.

In construction, maritime, and agriculture, OSHA is proposing a PEL of 5 mg/m³ as an 8-hour TWA and 10 mg/m³ as a 15-minute STEL for hard and soft wood dust, with the exception of Western red cedar, for which a PEL of 2.5 mg/m³ (8-hour TWA) is being proposed. OSHA preliminarily concludes that promulgation of these exposure limits will substantially reduce the significant risk of material impairment in the form of pulmonary dysfunction (including changes in peak flow, interference with mucociliary clearance, respiratory symptoms, and chronic effects) that is associated with exposure to wood dust at the higher levels that would be permitted in the absence of any limit. In addition, promulgation of these limits will make the PELs for these substances consistent across all OSHA-regulated sectors.

YTTRIUM AND COMPOUNDS
CAS No: 7440–65–6 (Yttrium); Chemical Formula: Varies with Compound
H.S. No. 2169

In general industry, construction, and maritime, OSHA's current PEL for yttrium and compounds (measured as yttrium) is 1 mg/m³ as an 8-hour TWA; NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. The ACGIH TLV®–TWA for yttrium and its compounds is 1 mg/m³ as an 8-hour TWA. OSHA is retaining its current limit of 1 mg/m³ as a 8-hour TWA in construction and maritime and is proposing to extend this limit to agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Yttrium is a metallic element that is odorless, lustrous, and dark gray (ACGIH 1986, p. 641(86)). Examples of yttrium compounds of commercial interest are yttrium oxide, yttrium acetate, yttrium bromide, yttrium chloride, yttrium phosphide, yttrium sulfate, yttrium vanadate, and yttrium nitrate hexahydrate. Yttrium is used to make alloys, in the manufacture of lasers, and in nuclear technology (ACGIH 1986, p. 641(86); Hawley's 1987, p. 1246). Yttrium compounds are used as reagents in analytical chemistry, as ingredients in electronic components, in phosphors for color television tubes, as mantles in gas and acetylene lights, and in many other applications (ACGIH 1986, p. 641(86); Hawley's 1987, p. 1246).

Yttrium and its compounds cause liver and lung damage in experimental animals. In general, yttrium's oral toxicity is low, although this substance is highly toxic when administered parenterally (Clayton and Clayton 1981, p. 1678). Acute toxicity varies with the compound and species. The intraperitoneal LD_{50}s in rats for yttrium chloride, yttrium nitrate, and yttrium oxide, respectively, are 45 mg/kg, 362 mg/kg, and 230 mg/kg (RTECS 1989, "Yttrium chloride"; RTECS 1989, "Yttrium oxide"; Sax and Lewis 1989, p. 3508). Rats injected intraperitoneally with 60 mg/kg of elemental yttrium every other day for 5 months survived, and autopsy failed to reveal accumulation of yttrium in the bones of these animals (MacDonald, Nusbaum, and Alexander et al. 1952, in ACGIH 1986). Rats exposed to yttrium oxide in the form of a single 50-mg intratracheal dose developed emphysema and diffuse granulomatous nodules within 8 months of the exposure (Clayton and Clayton 1981, p. 1682). Rats exposed to an unspecified concentration of yttrium chloride developed liver edema (with portal congestion), pleural effusions, and pulmonary hyperemia (Stokinger 1963, in Clayton and Clayton 1981, p. 1682).

There are no reports of yttrium-related exposure effects in humans; however, based on effects seen in animals, exposure to yttrium or its compounds is likely to cause lung and liver damage in overexposed individuals.

Based on this evidence, OSHA preliminarily concludes that workers in agriculture are at risk of experiencing lung and liver effects at the exposures permitted by the absence of an OSHA PEL. Accordingly, OSHA is proposing an 8-hour TWA PEL of 1 mg/m³ for yttrium and its compounds in agriculture. In addition, promulgation of this limit will make the PEL for yttrium consistent across all OSHA-regulated sectors.

Preliminary Conclusions For All Respiratory Toxicants

As Table C6–2 and the discussions above show, limits for the respiratory toxins have been established to control employee exposures to or below the airborne concentrations of these substances that have been associated with the development of acute or chronic respiratory effects. For most of these substances, the evidence is sufficient to identify the NOE or low-

effect levels that are related to these effects in humans or animals. Accordingly, OSHA preliminarily concludes that maintaining employee exposures at or below these limits will greatly decrease the likelihood that employees will be at significant risk of respiratory effects when they are exposed to these substances in the workplace. Because the chronic pulmonary disease caused by exposure to toxic dusts is often incapacitating, such exposures can effectively end the working life of severely affected individuals. Less-serious pulmonary disease can result in lost workdays, both as a result of the associated symptoms themselves and as a consequence of increased susceptibility to respiratory infections. The effects of exposure to acute pulmonary toxins, such as ozone or trimellitic anhydride, range from reduced lung function to life-threatening pulmonary edema. OSHA has preliminarily determined that these adverse pulmonary effects constitute material impairments of health. The Agency believes that the proposed limits will substantially reduce these significant occupational risks. In addition, promulgation of these limits will make OSHA's PELs for these substances consistent across all regulated sectors.

7. Substances for Which Proposed Limits Are Based on Avoidance of Cardiovascular Effects

Introduction. OSHA is proposing to revise or establish limits for eleven chemicals on the basis of their adverse effects on the cardiovascular system. Table C7–1 lists these cardiovascular toxins, along with their CAS numbers, HS numbers, 1987–1988 ACGIH TLV®s, and NIOSH RELs (if any). In addition, Table C7–1 shows OSHA's current PELs for these substances in construction and maritime. OSHA has no PELs for these substances in agricultural workplaces. The right-hand column in Table C7–1 shows the PELs that were recently established for these cardiovascular toxins in general industry; these are the limits being proposed today for construction, maritime, and agricultural workplaces. Promulgation of these limits will thus make OSHA's PELs for these 11 substances consistent across all regulated sectors.

Description of the Health Effects. Although the cardiovascular system can be adversely affected in many different ways by exposure to toxic substances, the adverse effects caused by exposure to the eleven chemicals shown in Table C7–1 are limited to three categories: (1) Cardiac sensitization; (2) vasodilation; and (3) atherosclerosis.

TABLE C7–1.—SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON AVOIDANCE OF CARDIOVASCULAR EFFECTS

H.S. Number/Chemical Name	CAS No.	Current OSHA PEL in Construction, and Maritime [1]	1987–1988 ACGIH TLV [2]	NIOSH REL [3]	Proposed OSHA PEL in Construction Maritime, and Agriculture [1]
2008 Antimony and compounds–	7440–36–0	0.5 mg/m³ TWA	0.5 mg/m³ TWA	0.5 mg/m³ TWA	0.5 mg/m³ TWA.
1070 Carbon disulfide	75–15–0	20 ppm TWA, Skin	10 ppm TWA, Skin	1 ppm TWA, 10 ppm Ceiling (15-min).	4 ppm TWA, 12 ppm STEL, Skin.
1087 Chloropentafluoroethane	76–15–3		1000 ppm TWA		1000 ppm TWA.
2058 Dichlorodifluoromethane	75–71–8	1000 ppm TWA	1000 ppm TWA		1000 ppm TWA.
2060 Dichlorotetrafluoroethane	76–14–2	1000 ppm TWA	1000 ppm TWA		1000 ppm TWA.
1170 Ethylene glycol dinitrate	628-96-6	1 mg/m³ Ceiling, Skin	0.3 mg/m³ TWA, Skin	0.1 mg/m³ Ceiling (20-min) [4].	0.1 mg/m³ STEL, Skin.
1180 Fluorotrichloromethane	75–69–4	1000 ppm TWA	1000 ppm Ceiling		1000 ppm Ceiling.
1290 Nitroglycerin	55–63–0	2 mg/m³ TWA, Skin	0.5 mg/m³ TWA, Skin	0.1 mg/m³ Ceiling (20-min) [4].	0.1 mg/m³ STEL, Skin.
2124 Pentachlorophenol	87–86–5	0.5 mg/m³ TWA, Skin	0.5 mg/m³ TWA, Skin		0.5 mg/m³ TWA, Skin.
1364 Sodium azide	26628-22-8		0.1 ppm Ceiling		0.1 ppm Ceiling, Skin (as HN_3); 0.3 mg/m³ Ceiling, Skin (as NaN_3).
1403 1,1,2-Trichloro-1,2,2-trifluoroethane	76–13–1	1000 ppm TWA	1000 ppm TWA, 1250 ppm STEL.		1000 ppm TWA, 1250 ppm STEL.

[1] OSHA's PELs do not currently apply in Agriculture; OSHA's TWA limits are for 8-hour exposures, its STELs are for 15 minutes unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time.

[2] The ACGIH TLV®-TWA is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times in any working day, with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

[3] NIOSH TWA limits are for 10 hour/day, 40 hour/week exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

[4] This is the limit recommended by NIOSH for ethylene glycol dinitrate or nitroglycerin alone or for a mixture of the two.

Because these effects can have potentially disabling or life-threatening outcomes, OSHA has preliminarily concluded that these effects clearly constitute material impairments of health and functional capacity.

Cardiac sensitization is a form of sensitization that is not mediated by the immune system; instead, chemicals that are cardiac sensitizers make the heart sensitive to the effects of a class of biological compounds called sympathomimetic amines. The physiological action of sympathomimetic amines is to stimulate the heart to beat faster; the hormone adrenaline (also called epinephrine) is an example of a sympathomimetic amine. Adrenaline is normally secreted into the bloodstream when the body anticipates an increase in physical exertion, such as occurs when someone is frightened. To increase the heartbeat rate, individuals must be exposed to a concentration (dose) of epinephrine that is equal to or higher than the no-effect level for this substance. The effect of a cardiac sensitizer is to lower the no-effect level so that the heartbeat rate is stimulated by exposure to a lower concentration (or dose) of adrenaline. The region of the heart that becomes sensitized is the pacemaking and conduction system, which determines the rhythm and rate of the heartbeat. Unregulated or unnecessary interference with this region of the heart can result in arrhythmia, an abnormality in the rhythm or rate of the heartbeat (Levy 1985/Ex. 1–210). The clinical consequences of arrhythmia vary among individuals, e.g., a young person with a healthy heart may not be adversely or seriously affected by an arrhythmia. However, fatal arrhythmias have occurred in healthy young people, and arrhythmias can result in cerebral or myocardial ischemia, shock, or

congestive heart failure in older people or in individuals whose cardiovascular systems have already been compromised.

Vasodilators are compounds that cause blood vessels to expand, resulting in a decrease in blood pressure (hypotension) and a decrease in the amount of blood reaching the organs. Acute hypotension is a common cause of shock ("Harrison's Principles of Internal Medicine," 10th ed., Petersdorf et al. 1983). Chronic hypotension may result in a number of symptoms, including lethargy, weakness, easy fatigability, and dizziness or faintness.

Atherosclerosis is a serious disease produced by a degenerative process in the arteries. Plaques containing lipids, complex carbohydrates, blood products, and calcium form on the inside walls of arteries, usually on the major blood vessels. These plaques are also called atheromas; their presence makes arteries narrower. Depending on which arteries in the body contain atheromas, different clinical consequences may result; these include renal hypertension, stroke, and myocardial ischemia (inadequate circulation of blood to the myocardium) (Balazs, Hanig, and Herman 1986/Ex. 1–176). Some chemicals can enhance or accelerate the formation of atheromas and thereby encourage the development of atherosclerosis, a major cause of coronary heart disease.

Dose-Response Relationships and Cardiac Effects

For several of the chemicals shown in Table C7–1, the proposed limits are based primarily on epidemiological studies, health surveys, and case reports indicating that occupationally exposed workers subjected to concentrations above a no-adverse-effect level experience these cardiovascular effects. However, human data for several other of these chemicals are scarce. For these chemicals, the proposed limits are based on the results of studies in laboratory animals.

Chemically induced cardiovascular disease occurs in a pattern that corresponds to a typical effect-level dose-response relationship; that is, an exposure level and exposure duration exist below which the substance appears unlikely to exert an adverse effect. Thus, the limits proposed for substances in this group are designed to maintain exposures below this apparent no-adverse-effect level. For the eleven substances in this group of cardiovascular toxins, the available toxicologic data and OSHA's preliminary findings are described below.

ANTIMONY AND COMPOUNDS
CAS: 7440–36–0; Chemical Formula: Varies
H.S. No. 2008

OSHA's current PEL for antimony and its compounds (measured as antimony, or Sb) in general industry, construction, and maritime is 0.5 mg/m³ as an 8-hour time-weighted average. There is no PEL in agriculture. Both NIOSH and the ACGIH also have an 8-hour TWA limit of 0.5 mg/m³ for this substance and its compounds. OSHA is proposing to establish an 8-hour TWA PEL of 0.5 mg/m³ for antimony and its compounds in agriculture. This is the limit recently established for these substances in general industry.

Antimony is a shiny, silvery or gray metal or a yellow crystalline solid (New Jersey Department of Health 1986). Some of the compounds of antimony in industrial use include antimony lactate, antimony pentachloride, antimony pentafluoride, antimony potassium tartrate, antimony tribromide, antimony trichloride, antimony trifluoride, and antimony trioxide. These compounds find use in various applications: fabric dyeing; metal-coloring; catalytic reactions; insecticidal applications; the production of porcelain, pottery, and pigments; and in decolorizing glass. When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Antimony and its compounds pose hazards to exposed individuals by several routes of exposure: inhalation, ingestion, and eye or skin contact. Experimental animals exposed to antimony and its compounds by inhalation develop pulmonary irritation; autopsy of acutely poisoned animals reveals enlargement of splenic follicles and fatty degeneration of the liver (NIOSH/OSHA 1988). The oral LD_{50} in rats for elemental antimony is 100 mg/kg (Genium 1989). For antimony trifluoride, the oral LD_{50} in mice is 804 mg/kg (Genium 1989); for antimony trichloride, the oral LD_{50} in rats is 525 mg/kg (RTECS 1990). For antimony pentachloride, the LC_{50} in rats is 720 mg/m³ for 2 hours (RTECS 1990).

Guinea pigs exposed chronically (duration not specified) to 45 mg/m³ of antimony trioxide developed extensive pneumonitis; at autopsy, they also exhibited fatty degeneration of the liver (Dernehl, Nau and Sweets 1945). Rabbits and rats exposed for 100 hours/month for 10 or 14.5 months to 100 to 125 mg/m³ concentrations of antimony (rats) or to 89 mg/m³ concentrations of antimony (rabbits) developed lipoid pneumonia (Gross 1955). Female rats exposed to antimony trioxide at a concentration of 3.2 or 4.2 mg/m³ for 6 hours/day, 5 days/week for 1 year developed lung tumors (EPA 1983). Rats, rabbits, and dogs exposed 7 hours/day to antimony sulfide (at an antimony concentration of 3.07 to 5.6 mg/m³) for 8 weeks developed functional disorders of the heart and parenchymatous degeneration of the myocardium (Brieger, Semisch, Stasney, and Piatner 1954).

In humans, exposure to antimony compounds causes, depending on the specific compound, irritation of the eyes, mucous membranes, and skin, pneumoconiosis, chronic cough, gastrointestinal symptoms, and cardiac effects (Potkonjak and Pavlovich 1983; Taylor 1966). Workers exposed to antimony trisulfide, antimony trioxide, or antimony trichloride have developed pulmonary fibrosis, electrocardiogram changes, heart muscle changes, and sudden death from heart attack (NIOSH/OSHA 1988). In a group of abrasive industry workers examined after exposure to mean antimony levels of 3 to 5 mg/m³ for 8 months to 2 years, 37 of 75 were found to have changes in their electrocardiograms, 14 of 75 had elevated blood pressure readings, and 7 of 111 had ulcers; six sudden heart-related deaths and two other deaths from chronic heart disease had occurred among these workers (Brieger, Semish, Stasney, and Piatner 1954). Dermal effects, known as "antimony spots" have been recognized in antimony workers for years, but the pneumoconiotic potential of these compounds has only been recognized in the last 20 years or so (McCallum 1963). Antimony spots take the form of papules and pustules around the sweat and sebaceous glands (McCallum 1963). Exposure to antimony halides or oxides causes irritation of the eyes, nose, and upper respiratory tract in exposed workers (Taylor 1966; Cordasco 1974). Increased rates of spontaneous abortions and premature births have been reported in metallurgical workers in the Soviet Union (Belyaeva 1967); there is also some evidence that exposure to antimony may cause an increased risk of lung cancer (Proctor, Hughes, and Fischman 1988, p. 79).

Based on the evidence discussed above, OSHA preliminarily concludes that, in the absence of a limit, agricultural workers who are exposed to antimony or its compounds are at increased risk of irritation of the eyes, nose, and upper respiratory tract; pneumoconiosis; skin rashes and pustules; spontaneous abortion and premature birth of offspring; and,

perhaps, of lung cancer. The Agency believes that the proposed permissible exposure limit of 0.5 mg/m³ (measured as antimony) will reduce these significant risks among agricultural workers. In addition, promulgation of this limit will make OSHA's PEL for these substances consistent across all regulated sectors.

CARBON DISULFIDE

CAS: 75–15–0; Chemical Formula: CS_2

H.S. No. 1070

In construction and maritime, OSHA's current limit for carbon disulfide is 20 ppm as an 8-hour TWA, with a skin notation. OSHA has no PEL for this substance in agriculture. The 1987–1988 ACGIH TLV®–TWA for this substance is 10 ppm, with a skin notation. NIOSH has recommended exposure limits of 1 ppm as a 10-hour TWA and 10 ppm as a 15-minute ceiling. NIOSH also concurs (Ex. 8–47, Table N1) with the limits being proposed. OSHA is proposing an 8-hour TWA PEL of 4 ppm and with a 12-ppm 15-minute STEL for carbon disulfide, with a skin notation. These are the limits recently established for carbon disulfide in general industry.

Carbon disulfide is a clear, colorless, or faintly yellow liquid with a strong, disagreeable odor. This substance is used in the manufacture of soil disinfectants; as a veterinary anthelmintic; as a solvent and corrosion inhibitor; as a space and commodity fumigant; as a catalyst; and as a chemical intermediate in rayon and other manufacturing processes (HSDB 1985).

Carbon disulfide is a narcotic and a central nervous system toxin in animals. The oral LD_{50} in rats is 3188 mg/kg, and the lowest lethal inhalation dose in the same species is 25 g/m³ for 2 hours (RTECS 1989). Animals acutely poisoned by exposure to carbon disulfide show excitement followed by muscular weakness, collapse, coma, and death by respiratory failure (HSDB 1985); at autopsy, animals showed degenerative changes in the ganglion cells of the retina and optic nerve (Grant 1986). Carbon disulfide has shown embryotoxic and fetotoxic effects in rats, mice, and rabbits by the oral and inhalation routes of administration (RTECS 1990); it also has reproductive effects in animals of several species (RTECS 1990). In humans, carbon disulfide causes adverse cardiovascular effects and damages the central and peripheral nervous systems (Proctor, Hughes, and Fischman 1988, p. 120). Occupational exposure to carbon disulfide has caused mental disturbances, including confusion, irritability, insomnia, and overt psychosis (Rom 1983, p. 318).

OSHA is proposing to reduce the exposure limits for carbon disulfide on the basis of the results of several studies of British and Finnish workers (Tiller, Schilling, and Morris 1968/Ex. 1–92; Seppalanin and Tolonen 1974/Ex. 1–100; Tolonen et al. 1975/Ex. 1–392; Tolonen, Nurminen, and Hernberg 1979/Ex. 1–158; Sweetnam, Taylor, and Elwood 1987; and Nurminen and Hernberg 1985). These studies, taken as a whole, indicate that long-term exposure to carbon disulfide concentrations at levels between 10 and 40 ppm is associated with an excess risk of adverse neurological effects and with excess mortality for coronary heart disease.

The study by Tiller et al. (1968/Ex. 1–92) of British rayon workers was the first to relate exposure to carbon disulfide with the development of coronary heart disease. These authors found that, among men employed for more than 10 years in the rayon industry and followed from 1950 to 1964, those exposed to carbon disulfide had death rates from coronary heart disease that were more than twice the rate in other rayon workers. Thus, the Tiller et al. (1968/Ex. 1–92) study demonstrated that 10 years or more of exposure to carbon disulfide was associated with a significantly elevated risk of coronary disease.

The United Kingdom's threshold limit value for carbon disulfide, which had been 20 ppm in the 1960s, was reduced to 10 ppm in the 1970s. To examine the effect of this reduced limit on occupational risk, Sweetnam et al. (1987) conducted a follow-up study on the cohort first described by Tiller et al. (1968/Ex. 1–92). The health status and cause of death for 2,848 members of this cohort were ascertained up to the end of 1982. Exposure scores representing cumulative exposure to carbon disulfide were developed for each cohort member, based on an analysis of personal and area sampling results, job category, and time spent in each job category. Sweetnam et al. (1987) found the pattern of mortality similar to that found by Tiller, Schilling, and Morris (1968/Ex. 1–92): among spinner operators, who had the highest CS_2 exposures of any job category, 73 deaths from ischemic heart disease (IHD) were identified, compared with 42.5 expected deaths (SMR=172), a finding that was statistically significant. A statistically significant trend was found between cumulative exposure since first exposure and incidence of IHD mortality, which indicates a dose-related effect. A second (and perhaps more important) finding of this study was that recent (or current) exposure to carbon disulfide, as well as total cumulative exposure, were both risk factors for IHD. The authors established this association by examining the relationship between IHD mortality risk and each worker's total CS_2 exposure in the two years preceding death or the two years before the end of the study. The third result of this study was that workers with current CS_2 exposure also had significantly higher risk than workers whose exposure had ceased. The dose-related relationship between increased IHD mortality risk and recent exposure to carbon disulfide suggested to the authors of this study that the effect of carbon disulfide on the cardiovascular system was direct and reversible.

Thus, the Sweetnam et al. (1987) follow-up study determined that there is a relationship between the risk of IHD mortality and increased cumulative exposure to CS_2. Among workers who terminated exposure, this risk declined to non-statistically significant levels after one year of no exposure. However, risk continued to be elevated among workers who continued to be exposed or who had not been exposure-free for a full year. OSHA believes that the findings of this study indicate that cumulative CS_2 dose from time of first exposure is a risk factor for IHD, and that this elevated risk continues unless exposure is terminated. That is, OSHA believes that workers who have been exposed to CS_2 in the past continue to be at increased risk as long as they are exposed to CS_2, even when their recent exposure is to lower levels (approximately 10 ppm, the current limit in the United Kingdom).

This interpretation appears to have been confirmed by Nurminen and Hernberg (1985) in their follow-up study of 343 Finnish rayon workers who had been exposed to carbon disulfide for at least five years. Health status data were obtained for these workers for the period 1967 to 1982. In 1972, a preventive program had been instituted that included the establishment of a 10-ppm exposure limit and the removal of workers at high risk of coronary disease from continued exposure to carbon disulfide. Median exposure levels (calculated largely from area samples) for the period 1975 to 1980 did not exceed 5 to 6 ppm, and third-quartile exposure levels did not exceed 10 ppm. These levels were about half those reported for the period 1967 to 1975.

Nurminen and Hernberg (1985) reported a 4.7-fold increase in IHD mortality incidence for the period 1967 to 1972, i.e., for the period prior to the

establishment of the protective measures described above. Five years after these measures were instituted, only 19 percent of the cohort continued to be exposed to carbon disulfide (compared to 53 percent of the cohort exposed in 1972). The relative risk for the first seven years of follow-up (1967 to 1974) was 3.2, compared with a relative risk of 1.0 for the last eight years (1974 to 1982). The excess risk of IHD mortality thus declined steadily throughout the follow-up period; this trend was statistically significant. The authors concluded that "* * * the cardiotoxic effects of CS_2 are reversible in the sense that the cessation of, or a radical decrease in, exposure reduces the risk of cardiovascular mortality to background levels" (Nurminen and Hernberg 1985, p. 34). Thus, the Nurminen and Hernberg (1985) study shows that reducing exposure levels below 10 ppm (combined, in their case, with a rigorous medical removal program to terminate exposure for employees who had developed signs or symptoms of coronary heart disease) can reduce the significant risk of IHD mortality to baseline levels.

It should be noted, however, that this study should not be interpreted to mean that 10 ppm is sufficiently protective. The average exposure of this group was to concentrations of about 5 ppm. In addition, this workforce was highly selective, because workers having a risk factor for cardiovascular disease had been removed. As many as 50 percent of all middle-aged men have some risk factors for cardiovascular disease; however, a medical screening program as rigorous as the one in place for the workers in this study would undoubtedly screen out perhaps as many as half the workforce. Furthermore, a workforce this selective would be expected to have a below-average rate of cardiovascular disease; instead, it should be noted that they actually had an average cardiovascular disease risk.

In the earlier PEL rulemaking, OSHA proposed a 1-ppm 8-hour TWA for carbon disulfide, and many commenters, including NIOSH (Exs. 8–47 and 193), the AFL–CIO (Ex. 194), and Dr. James Melius, Director of the Division of Occupational Health and Environmental Epidemiology of the New York Department of Health (Ex. 152), supported this proposed limit. Some participants (Exs. 3–747, 3–1158, 8–19, 8–45, 31, 125, and 174; Tr. pp. 4–74 to 4–107) in the prior rulemaking argued that the evidence in the studies described above did not support a limit of 1 ppm; these commenters argued that the cohorts in which excess deaths from cardiovascular disease had been seen included workers who had been exposed for many years to levels of carbon disulfide much higher than the 10- to 40-ppm levels generally reported in these studies.

OSHA responded to these commenters in the preamble to the final rule for air contaminants in general industry (54 FR 2537) by stating that, in the Agency's opinion, both cumulative exposure and current exposure are risk factors for IHD among CS_2-exposed workers. Further, OSHA stated that the Agency believes that this excess risk continues for as long as exposure continues.

A recent epidemiologic study by MacMahon and Monson (1988/Ex. 125) also supports carbon disulfide's cardiotoxicity in the workplace setting. The cohort in this study consisted of 10,418 men employed between 1957 and 1979 in the four principal viscose rayon plants in the United States, and the mortality status of the cohort was ascertained up to mid-1983. Cohort members were placed into general exposure categories according to job title; these categories were highest, intermediate, variable, least, and none. The authors found no significant increase in overall mortality in the 4,448 employees with the highest potential for CS_2 exposure compared with the mortality among 3,311 employees with no CS_2 exposure. However, there was a statistically significant excess of arteriosclerotic heart disease (ASHD) among the most heavily exposed workers (242 deaths versus 195.6 expected). No clear relationship was observed between exposure duration or latency and excess ASHD mortality; however, the data suggest that the risk was higher among employees exposed to CS_2 for 15 or more years and among employees hired prior to 1960.

In addition, MacMahon and Monson (1988/Ex. 125) found a statistically significant increase in the SMR (SMR=150) for ASHD among members of the cohort who had been exposed to CS_2 in the year immediately preceding death or in the year immediately preceding the termination date of the study (Ex. 125, Attachment B, Table 7, p. 702); however, there was no general pattern of increased SMRs among cohort members whose time since last exposure exceeded one year. This finding is consistent with the results of the British studies, which also found an increased risk of heart disease among recently exposed employees but not among employees who had left their jobs.

In the prior rulemaking, one commenter (1988/Ex. 125) interpreted the MacMahon and Monson study as meaning that U.S. workers employed since 1960 were not at risk of ASHD (Tr. 4–96). However, OSHA disagrees with this view and believes that the results of the MacMahon and Monson (1988/Ex. 125) study are supportive of and consistent with those of the British and Finnish studies discussed above, for the following reasons. First, all of the studies clearly demonstrate a positive association between exposure to carbon disulfide and an increased risk of mortality from heart disease. Second, studies from all three countries link the excess risk to cumulative CS_2 exposure. Third, studies from all three countries demonstrate that this significant risk can be substantially reduced or eliminated by reducing or stopping exposure, even after a considerable CS_2 dose has accumulated; both the U.S. and British studies report a significantly increased risk of death from heart disease among workers who were recently exposed but no increased risk among workers whose exposures had ended one year or longer prior to death or prior to the end of the study. Moreover, the Finnish study reported steady declines in heart disease mortality among workers after exposure levels were reduced to below 10 ppm and a rigorous medical screening and removal program was instituted. These findings would appear to demonstrate that current or continued exposure to carbon disulfide at the levels presently encountered in these facilities is as important a risk factor for heart disease mortality as cumulative exposure. Therefore, reducing exposures to below 10 ppm does not by itself appear to eliminate risk.

In addition to evidence that carbon disulfide is a cardiovascular toxin, there is a substantial body of evidence that exposure to carbon disulfide presents a fetotoxic hazard and that this substance may also be a teratogen. Some of the early (pre-1977) animal data on reproductive effects were reviewed in the NIOSH criteria document (1977b/Ex. 1–260) on carbon disulfide. There are also two more recent and relevant reports (Cai and Bao 1981 and Hemminki and Niemi 1982). Cai and Bao (1981) reported increased incidences of menstrual disturbances and of pregnancy toxemia, a potentially lethal condition, among rayon workers and also presented evidence that CS_2 can cross the placental barrier and be secreted into mothers' milk. The Hemminki and Niemi (1982) study found a significantly elevated incidence of

spontaneous abortions among women employed in viscose rayon facilities in Finland; however, data on the specific CS_2 exposure levels were generally lacking.

The Rohm and Haas Company submitted a summary (Ex. 10–5) of the evidence on the reproductive toxicity of carbon disulfide to the docket in the prior rulemaking; this information shows that carbon disulfide has caused fetal deaths and malformations in CS_2-exposed laboratory animals. Rohm and Haas described studies in which oral administration of CS_2 to female rats during gestation produced both teratogenic and fetotoxic effects. These effects were magnified in the F_2 offspring of the prenatally exposed F_1 generation, which suggests that CS_2 has a multi-generational effect that continues to cause malformations in successive generations.

Jones-Price et al. (1984) found both maternal and fetal toxicity in CD rats exposed orally to 200, 400, or 600 mg/kg/day CS_2 during days 6 through 15 of gestation. No dose-related increases in the incidence of teratogenicity were observed. In another report, these authors found significant dose-related increases in percent resorptions/litter, percent non-live (dead or resorbed)/litter, and percent of fetuses affected (non-live and malformed)/litter among New Zealand White rabbits exposed orally to 25, 75, or 150 mg/kg/day during days 6 through 19 of gestation. The percentage of malformed fetuses per litter increased with dose and was statistically significant at the highest dose tested.

In an inhalation study, Hardin, Bond, Sikov et al. (1981/Ex. 1–699) exposed rats and rabbits to 20 or 40 ppm CS_2 for 6.5 hours per day during days 1 through 19 (rats) or 1 through 24 (rabbits) of gestation. No embryotoxic or fetotoxic effects were noted, indicating that 40 ppm is a no-effect level for these effects in rats and rabbits. It would thus appear that the lowest-reported-effect level (25 mg/kg/day) documented by Jones-Price et al. (1984) for embryotoxicity/fetotoxicity in rabbits corresponds to an equivalent airborne exposure of 58 ppm; this lowest-reported-effect level is in close agreement with the no-effect level reported by Hardin et al. (1981/Ex. 1–699) for the same species.

OSHA believes that this evidence, which shows that fetotoxic and teratogenic effects are associated with exposure to carbon disulfide, warrants concern, particularly in light of the fact that CS_2 has been shown to cause multigenerational effects in rats. Based on the significant risks of reproductive effects and cardiovascular disease, OSHA has preliminarily concluded that a substantial reduction in the PEL for carbon disulfide is justified for workers in the construction, maritime, and agriculture industries. The 4 ppm level proposed thus provides a 10-fold "safety factor" for fetotoxic effects in animals. Secondly, 10 ppm does not appear to be sufficiently protective to eliminate significant risk of cardiovascular disease. The additional protection of 4 ppm is needed for these effects. The Agency preliminarily finds that the proposed limits of 4 ppm as an 8-hour TWA and 12 ppm as a STEL, with a skin notation, will protect workers in these sectors from these significant risks, which constitute material health impairments that are potentially associated with exposure to this substance. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

CHLOROPENTAFLUOROETHANE
CAS: 76–15–3; Chemical Formula: $ClCF_2CF_3$
H.S. No. 1087

OSHA has no limit for chloropentafluoroethane (FC–115) in construction, maritime, or agriculture. The ACGIH TLV® for this substance is 1000 ppm as an 8-hour TWA; NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL for chloropentafluoroethane of 1000 ppm in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Chloropentafluoroethane is a colorless, nonflammable gas that has a mild, ether-like odor. Chloropentafluoroethane is used primarily as a refrigerant; a dielectric gas; an aerosol propellant; and in food preparation (HSDB 1989).

At high concentrations, this substance is an asphyxiant and a cardiac sensitizer. At lower concentrations and in liquid (pressurized) form, contact of FC–115 with the skin causes frostbite and burns (Braker and Mossman 1980, p. 176). Rats, mice, rabbits, and dogs have tolerated six-hour daily exposures to 100,000 ppm FC–115 for 90 days without adverse effects (Clayton, Hood, Nick, and Waritz 1966/Ex. 1–952), and laboratory animals have tolerated exposure to 200,000 ppm for 3.5 hours daily, five days per week, for four weeks (Weigand 1971/Ex. 1–1102). However, dogs exposed to a 10- or 20-percent concentration of FC–115 (approximately 100,000 to 200,000 ppm) showed central nervous system depression (Aviado and Belej 1975), and rats exposed to 190,000 ppm for between 4 and 26 minutes developed apnea (cessation of breathing) (HSDB 1989). FC–115's potential for cardiac sensitization caused one of 13 unanesthetized dogs to develop cardiac sensitization after exposure to 150,000 ppm intravenously (Trochimowicz, Azar, Terrill, and Mullin 1974/Ex. 1–448; Reinhardt, Azar, Maxfield, et al. 1971). Several other studies indicate that unanesthetized dogs, rats, and monkeys exposed to concentrations between 100,000 ppm and 200,000 ppm may show increased blood pressure, accelerated heart rate, myocardial depression, or altered pulmonary effects under certain conditions (Belej and Aviado 1975/Ex. 1–462; Friedman, Cammarato, and Aviado 1973/Ex. 1–416; Aviado and Belej 1975/Ex. 1–616).

Based on this evidence in humans and animals, OSHA is proposing an 8-hour TWA permissible exposure limit of 1000 ppm for chloropentafluoroethane to protect workers in construction, maritime, and agriculture from the significant risk of FC–115-related cardiac effects. This limit includes a reasonable safety factor in light of the seriousness of the effect and the need to account for interspecies variation. The Agency believes that these health effects constitute material impairments of health and functional capacity and that the PEL is necessary to reduce these risks. Promulgation of this limit will also make OSHA's PEL for this substance consistent across all regulated sectors.

DICHLORODIFLUOROMETHANE
CAS: 75–71–8; Chemical Formula: CCl_2F_2
H.S. No. 2058

OSHA's current PEL for dichlorodifluoromethane in general industry, construction, and maritime is 1000 ppm as an 8-hour TWA; there is no PEL in agriculture. The ACGIH TLV®–TWA for dichlorodifluoromethane is 1000 ppm; NIOSH has no REL for this substance but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA of 1000 ppm for dichlorodifluoromethane in agriculture. This action will make the PEL for this substance consistent across all OSHA-regulated sectors.

Dichlorodifluoromethane, also called Fluorocarbon 12, is a colorless, nonflammable gas. It is odorless in concentrations below 20 percent by volume; at higher concentrations, it has a mild ether-like odor similar to that of carbon tetrachloride (Braker and Mossman 1980, p. 231). Dichlorodifluoromethane is used as a refrigerant, a blowing agent for polymeric foams, and a fixative for

frozen tissue sections; formerly, it was widely used as an aerosol propellant (ACGIH 1986, p. 181; HSDB 1990).

Dichlorodifluoromethane is an irritant and a narcotic in humans and animals; at very high concentrations, inhalation of this substance causes asphyxiation. Dichlorodifluoromethane also causes cardiac sensitization in animals. The LC_{50} in rats is 800,000 ppm for 30 minutes (RTECS 1990). Dogs, monkeys, and guinea pigs exposed to a 200,000-ppm concentration of dichlorodifluoromethane for several hours daily for several days exhibited the following signs: Tremors, ataxia, salivation, and lacrimation (Grant 1986, p. 322). Rats exposed to a 10,000-ppm concentration of this substance for 6 hours/day for 90 days and dogs exposed to a 5,000-ppm concentration on the same regimen showed no adverse effects (Leuschner, Neumann and Hubscher 1983). Cardiac arrhythmias were observed in five of 12 dogs exposed both to a 50,000-ppm concentration of dichlorofluoromethane and to intravenous epinephrine (Reinhardt, Azar, Maxfield et al. 1971). Carcinogenicity and teratogenicity studies involving this substance have been negative (Maltoni, Lefemine, Tovoli, and Perino 1988; Brar, Jackson, Traitor et al. 1976).

In humans, dichlorodifluoromethane causes eye and upper respiratory tract irritation as well as central nervous system depression. Volunteers exposed to a 200,000-ppm concentration of dichlorodifluoromethane for a brief (not further specified) time reported experiencing eye irritation and central nervous system effects; all effects reversed after termination of exposure (Proctor, Hughes, and Fischman 1988, p. 187). When the concentration was reduced to 110,000 ppm and the duration was increased to 11 minutes, volunteers experienced amnesia, cardiac arrhythmias, and a decrease in their level of consciousness (Proctor, Hughes, and Fischman 1988, p. 187). At an exposure level of 40,000 ppm for 80 minutes, subjects experienced ringing in the ears, a feeling of apprehension, and generalized paresthesia; their speech was also slurred (Proctor, Hughes, and Fischman 1988, p. 187). Two volunteers exposed to 10,000 ppm for 2.5 hours showed psychomotor impairment on testing (Azar, Reinhart, Maxfield, et al. 1972). Exposing volunteers to a 1000-ppm concentration of dichlorodifluoromethane for 8 hours/day for 17 days, however, caused no exposure-related effects (Stewart, Newton, Baretta, et al. 1978). A man who accidentally punctured a refrigerator coil containing dichlorodifluoromethane subsequently developed bronchopneumonia and died; he is believed to have aspirated the freezing liquid or vapor (Laurain 1964). A man chronically exposed to dichlorodifluoromethane at work (concentrations unknown) developed chronic pharyngolaryngeal edema, with the following signs and symptoms: dysphagia, dysphonia, and inspiratory dyspnea; these effects disappeared after the man moved to another job (Tanturri, Pia, and Benzi 1988).

Based on this evidence in humans and animals, OSHA preliminarily concludes that workers in agriculture are at significant risk of experiencing these exposure-related effects and that implementation of the proposed 8-hour TWA PEL of 1000 ppm will substantially reduce these risks. Adoption of the proposed limit will also ensure consistency in OSHA's limit for this substance in all regulated sectors.

DICHLOROTETRAFLUOROETHANE

CAS: 76–14–2; Chemical Formula: $CClF_2CClF_2$

H.S. No. 2060

The current OSHA limit for dichlorotetrafluoroethane in general industry, construction, and maritime is an 8-hour TWA PEL of 1000 ppm. OSHA does not currently have a limit for this substance in agriculture. The ACGIH has a TLV®–TWA of 1000 ppm for dichlorotetrafluoroethane; NIOSH has no REL but concurs (Ex. 8–47, Table N3A). OSHA is proposing an 8-hour TWA PEL of 1000 ppm for this substance in agriculture; this action will ensure consistency in the limit for dichlorotetrafluoroethane in all OSHA-regulated sectors.

Dichlorotetrafluoroethane is a colorless, nonflammable gas with a faint ether-like odor at high concentrations (Merck 1983, pp. 373–374). It is shipped as a liquefied gas. This substance is used as a solvent; in fire extinguishers; as a refrigerant, blowing agent, and dielectric fluid; and in air conditioning systems (Hawley's 1987, p. 382; Hazardous Substance Fact Sheet 1986, p. 1).

At extremely high concentrations, dichlorotetrafluoroethane can cause asphyxia. It also causes cardiac sensitization, central nervous system depression, and respiratory and eye irritation in humans and experimental animals (Proctor Hughes and Fischman 1988, p. 193; Gosselin 1984, P. II–160). The LC_{50} values for rats, mice, and rabbits are 720,000, 700,000, and 750,000 ppm, respectively, for 30-minute exposures (RTECS 1987). Local inflammation of the skin and eyes was caused by repeated spraying of the skin or eyes of rats or rabbits with dichlorotetrafluoroethane (Quevauviller 1965; Quevauviller et al. 1964, in ACGIH 1986, p. 191). Exposures to concentrations of 300,000 to 400,000 ppm disturbed the equilibrium of rats and guinea pigs exposed to these concentrations for 2 hours (Scholz 1962, in ACGIH 1986, p. 191). Guinea pigs exposed for 2 hours to concentrations ranging from 8000 to 47,000 ppm developed irregular breathing patterns but showed no other signs of toxicity (Nuckolls 1933, in ACGIH 1986, p. 191). Although dogs survived single 8-hour exposures to a concentration of 200,000 ppm, a single 16-hour exposure or repeated 8-hour exposures to the same concentration proved to be lethal. After twenty-one 8-hour exposures to concentrations ranging from 142,000 to 150,000 ppm, dogs were alive but showed blood changes, incoordination, and convulsions (Yant 1933). One of 12 dogs experienced serious cardiac arrhythmia after exposure to a 25,000 ppm concentration of dichlorotetrafluoroethane and an injection of epinephrine (Reinhardt, Azar, Maxfield 1971). Another study reported that exposure to a dichlorotetrafluoroethane concentration of 50,000 to 800,000 induced cardiac sensitization in dogs (Reinhardt, Azar, Maxfield et al. 1971; Mullin, Azar, Reinhardt et al. 1972). Chronic exposure of rats to 10,000 ppm and dogs to 5,000 ppm for 6 hours/day for 90 days caused no clinical, biochemical, or histologic changes visible at autopsy (Leuschner et al. 1983, in Proctor, Hughes, and Fischman 1988, p. 193).

In humans, exposure to dichlorotetrafluoroethane vapor causes mild and transient central nervous system damage (Gosselin, Smith, and Hodge 1984, p. II–160). Contact with the cryogenic liquid can cause frostbite or burns of the skin or eyes (Proctor, Hughes, and Fischman 1988, p. 193).

Based on this evidence in humans and animals, OSHA primarily concludes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing eye and respiratory irritation and central nervous system depression. OSHA believes that an 8-hour TWA PEL of 1000 ppm will substantially reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ETHYLENE GLYCOL DINITRATE

CAS: 628–96–6; Chemical Formula: $CH_2NO_3CH_2NO_3$

H.S. No. 1170

NITROGLYCERIN
CAS: 55-63-0; Chemical Formula: $CH_2NO_3CHNO_3CH_2NO_3$
H.S. No. 1290

OSHA's current exposure limits for ethylene glycol dinitrate (EGDN) and nitroglycerin (NG) in construction and maritime are both 0.2 ppm (1.0 mg/m³) as ceiling concentrations. These limits also bear skin notations. There are no PELs for these substances in agriculture. The ACGIH TLV®s for EDGN and NG are 0.05 ppm (0.3 mg/m³) as 8-hour TWAs, with skin notations. OSHA is proposing limits for EDGN and NG in construction, maritime, and agriculture of 0.1 mg/m³ as 15-minute STELs and is retaining the skin notations; these limits are based on NIOSH's recommended limits. These are the limits recently established for these substances in general industry.

EDGN is an oily, yellowish, explosive liquid, and NG is a pale yellow, viscous liquid. EDGN and NG are frequently encountered together in dynamite and other high explosives. EDGN is used exclusively as an anti-freezing agent in such explosives. NG is also used in rocket propellant (HSDB 1989), as a medication for cardiovascular disease, and in veterinary medicine to treat asthma in dogs (Merck 1983).

Both NG and EDGN have moderate acute toxicity when administered orally to rats or mice; the oral LD_{50}s for NG in rats and mice are 105 and 115 mg/kg, respectively, and the oral LD_{50} for EDGN in rats is 616 mg/kg (RTECS 1990). Animals given NG or EDGN orally exhibit marked cardiovascular and other symptoms, including decreased blood pressure, tremors, ataxia, lethargy, altered respiration, cyanosis, prostration, convulsions, and death from either respiratory or cardiac arrest. In addition, both NG and EDGN produce methemoglobinemia in animals following acute exposure. Chronic exposure (2 ppm, 8 hours/day, for 1000 days) of cats to EDGN caused anemia; fatty changes in the heart, liver, and kidney; and hyperplasia of the bone marrow (HSDB 1989). Reproductive and developmental effects have been noted in rats exposed to NG by skin painting (2400 mg/kg for 30 days) and intraperitoneal injection (11 or 220 mg/kg during days 7–17 of pregnancy) (RTECS 1990).

The cardiovascular effects seen in animals have also occurred in humans; low-level exposure to NG or EDGN causes headache (due to vasodilation), dizziness, nausea, skin rash, and methemoglobinemia. More severe exposure results in hypotension, flushing, palpitations, delirium, and central nervous system depression. Consumption of alcohol can aggravate these effects. Exposure to NG or EDGN can also result in persistent tachycardia and reduced pulse pressure (Carmichael and Lieben 1963).

Workers exposed to NG or EDGN for a period of time (i.e., 6 to 10 years) may develop organic nitrate dependence, which causes severe and life-threatening symptoms to appear after periods of non-exposure (such as after a weekend). In such cases, affected individuals complain of sudden chest pains that resemble angina pectoris and then die suddenly, often before treatment can be administered (Carmichael and Lieben 1963; Proctor, Hughes, and Fischman 1988, p. 247, 376).

A number of investigations provide information on the exposure levels that result in adverse effects in humans. Trainor and Jones (1966/Ex. 1–107) reported that exposure to EGDN:NG at a level of 0.7 mg/m³ for 25 minutes was sufficient to produce decreased blood pressure and a slight headache in humans. These authors also reported that workers at a munitions plant developed headaches when exposed to EGDN:NG concentrations between 0.1 and 0.53 mg/m³ (0.36 mg/m³ average). Morikawa, Muraki, Ikoma et al. (1967/Ex. 1–55) found that workers in an explosives plant exposed to low concentrations of EGDN:NG (0.066 ppm (approximately 0.5 mg/m³) was the highest average level) had a much higher incidence of abnormal pulse waves than did controls (143 out of 1,271 versus 0 out of 175). Abnormal pulse waves often indicate a clinically significant defect in the functioning of the heart and/or circulatory system (Braunwald 1978/Ex. 1–1001).

In its criteria document for NG and EGDN, NIOSH (1978h/Ex. 1–234) refers to a report of a dynamite worker who died when exposed to EGDN:NG concentrations between 0.3 and 1.4 mg/m³, as well as to another report of two workers who died suddenly following exposure to EGDN:NG at concentrations ranging from 1.7 to 2.7 mg/m³. NIOSH (1978h/Ex. 1–234) observed that skin absorption may have contributed significantly to the exposures causing these deaths.

During the rulemaking for general industry, OSHA received several comments (Exs. 3–661, 8–66, 121, 190, and 154) on the strength of the evidence and significance of the adverse effects associated with exposure to EGDN and NG. Specifically, these commenters questioned the relevance of the toxicologic data discussed above in light of the improved working conditions prevailing today in NG/EDGN manufacturing plants. In addition, the Institute of Makers of Explosives (IME) asserted that "headaches are transitory phenomena which pose no significant health risk" (Ex. 190, p. 5).

OSHA does not share the IME's view of the significance of chemically induced headaches. The Agency believes that such headaches impair performance, cause pain and suffering, affect the safety of the victim and his or her co-workers, and contribute to absenteeism. In the case of EGDN:NG-induced headaches, however, headaches have a greater meaning: They are an early warning of vasodilation, an indicator of systemic toxicity. OSHA also believes that the report of an EGDN:NG-induced death in an explosives manufacturing facility is both convincing and troubling. The Trainor and Jones (1966/Ex. 1–107) study, the NIOSH criteria document (1978h/Ex. 1–234), and the Morikawa, Muraki, Ikoma et al. (1967/Ex. 1–55) study indicate that the health effects associated with exposure to very low levels of EGDN:NG (i.e., in the range of 0.1 to 1.4 mg/m³) are acute, may occur after brief exposures, and have been shown to be lethal.

According to NIOSH (Ex. 150), the 15-minute 0.1-mg/m³ limits being proposed for these substances will protect against "angina pectoris, other signs and symptoms of cardiac ischemia or heart damage, and against sudden death * * * since all of these * * * seem to be related to compensatory vasoconstriction induced by repeated exposure to NG or EGDN" (Ex. 150). NIOSH also reports that a preliminary study of mortality resulting from heart disease and other causes among NG workers by Reeve, Bloom, Rinsky, and Smith (1983a and 1983b) suggests an association between NG exposure and cardiovascular disease mortality; at the facilities where this increase in cardiovascular disease occurred, exposures were being maintained near or below 0.02 ppm (0.2 mg/m³) (Ex. 150).

Hypotension and headache have been observed in populations exposed to EGDN:NG at levels below 0.5 mg/m³ for brief periods (25 minutes), and fatalities have occurred after EGDN:NG exposures at concentrations between 0.3 and 1.4 mg/m³, in one instance, and between 1.7 and 2.7 mg/m³, in another.

These substances present severe explosive hazards as well. Methods to reduce exposures to EGDN:NG must take this factor into account. OSHA has considered the explosion factor in various actions it has taken in regard to general industry, such as extending compliance periods. OSHA is not aware

of these problems in construction, maritime, or agriculture; therefore, the Agency requests comment on whether such problems exist in these sectors.

OSHA's current standard for these substances in construction and maritime is 0.2 ppm (1.2 mg/m³ for EGDN and 2.0 mg/m³ for NG); since worker deaths have occurred at or near this level, OSHA is proposing short-term limits for EGDN and NG of 0.1 mg/m³, with skin notations, for these substances in these two sectors and in agriculture. OSHA preliminarily concludes that these limits are necessary to prevent fatalities and to protect workers in these sectors against the significant risks of vasodilation and cardiac effects, which are considered material impairments of health. Because EGDN:NG is readily absorbed through the skin and can produce systemic effects by this exposure route, OSHA is retaining the skin notations for both substances in construction and maritime and is proposing one in agriculture. In addition, promulgation of these limits will make OSHA's PELs for these substances consistent across all regulated sectors.

FLUOROTRICHLOROMETHANE
(TRICHLOROFLUOROMETHANE)
CAS: 75–69–4; Chemical Formula: CCl_3F
H.S. No. 1180

Fluorotrichloromethane (trichlorofluoromethane), also known as FC–11, is a member of a large family of chemicals, the chlorofluorocarbons. OSHA has no PEL for this substance in construction, maritime, or agriculture. The ACGIH TLV® for FC–11 is 1000 ppm as an instantaneous ceiling. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing a ceiling PEL of 1000 ppm for fluorotrichloromethane in construction, maritime, and agriculture. This is the limit recently established in general industry.

FC–11 is a colorless, nonflammable, low-boiling liquid or gas (depending on temperature). Its primary uses are as an aerosol propellant, a refrigerant, a fire extinguisher, a solvent and degreasing agent, and a blowing agent in the production of polyurethane foam (HSDB 1985).

The lowest lethal inhalation dose of FC–11 in 50 percent of mice (LC_{50}) is 100,000 ppm for 30 minutes (RTECS 1990). Before death, these mice developed cardiac arrhythmias, and dogs inhaling 25,000 ppm had decreased myocardial function (Aviado and Belej 1975). Monkeys were the most sensitive of the species tested to FC–11's cardiotoxic effects (Clayton and Clayton 1981, p. 3085); Drinking Water and Health, National Research Council 1977). Exposure to 5000 ppm FC–11 has induced cardiac sensitization and arrhythmia in dogs that were intravenously injected with epinephrine (Reinhardt, Azar, Maxfield, Smith, and Mullin 1971/Ex. 1–78).

Acute overexposure to FC–11 by inhalation has caused cardiac sensitization (arrhythmia) or bronchial constriction and death in exposed humans (Drinking Water and Health, National Research Council 1977). Volunteers exposed to 1000 ppm for 8 hours/day, 5 days/week for 18 exposure episodes evidenced no changes in their electrocardiograms or pulmonary function tests (Clayton and Clayton 1981, p. 3081). Slowed heart rate is the usual response in humans inhaling low (not further specified) concentrations of FC–11 (Clayton and Clayton 1981, p. 3085).

The cardiac sensitization exhibited by FC–11-exposed individuals is an acute effect. OSHA believes that, in the absence of a limit, workers in construction, maritime, and agriculture could be exposed to sufficiently high concentrations of FC–11 to sensitize the heart to sympathomimetic amines; OSHA considers this effect to be a material impairment of health. Accordingly, OSHA preliminarily concludes that workers in these sectors are at significant risk of experiencing arrhythmia and the other exposure-related effects of FC–11 and that the proposed 1000-ppm ceiling limit will substantially reduce this significant risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PENTACHLOROPHENOL
CAS: 87–86–5; Chemical Formula: C_6HCl_5O
H.S. No. 2124

In general industry, construction, and maritime, OSHA's permissible exposure limit for pentachlorophenol is 0.5 mg/m³ as an 8-hour TWA; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.5 mg/m³, with a skin notation, for pentachlorophenol. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing to establish an 8-hour TWA PEL of 0.5 mg/m³, and a skin notation, for pentachlorophenol in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Pentachlorophenol is a colorless to light brown solid with a phenol-like odor and a pungent taste. It is used as a chemical intermediate, and as a herbicide, fungicide, molluscicide, and wood preservative (ACGIH 1986, p. 461; Hawley's 1987, p. 881). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Pentachlorophenol causes eye, skin, and respiratory tract irritation, hyperpyrexia, increased metabolic rate, and circulatory effects in humans and animals; in animals it is also fetotoxic. The oral LD_{50}s in various species are 27 mg/kg in rats, 117 mg/kg in mice, and 168 mg/kg in hamsters, respectively (RTECS 1990). In rats, the dermal LD_{50} is 95 mg/kg; the lowest lethal dermal dose in rabbits is 40 mg/kg (RTECS 1990). The LC_{50} in mice is 225 mg/m³; in rats, it is 355 mg/m³ (RTECS 1990). When 10 mg of this substance was placed on the skin of rabbits, it caused mild irritation (RTECS 1990). Acutely poisoned animals showed accelerated respiration, elevated body temperature, tachycardia, and neuromuscular weakness before dying of cardiac failure; rigor mortis is instantaneous (Deichmann et al. 1942, in IARC 1979, Vol. 20, p. 314). Animal studies have shown that the immune system is sensitive to pentachlorophenol exposure (Kerkvliet, Baecher-Steppan and Schmitz et al. 1982). Mice given 50 to 500 ppm technical grade pentachlorophenol in their diets showed signs of greatly reduced immunocompetence, expressed as an increased susceptibility to the growth of transplanted tumors (Kerkvliet, Baecher-Steppan and Schmitz et al. 1982). Thirty male rats given 20 mg/l of pentachlorophenol showed neurochemical effects after 3 to 14 weeks of exposure (National Research Council 1986, in HSDB 1989). In a 2-year study, rats were given oral doses of 10 and 30 mg/kg/day of pentachlorophenol; the animals in the high-dose group showed decreased body weights, increased serum glutamic-pyruvate transaminase activity, and increased urinary specific gravity (Schwetz et al. 1978, in Clayton and Clayton 1981, p. 2607). Hamsters given daily oral doses of 1.25 mg/kg pentachlorophenol from days 5 to 10 of gestation had an increase in fetal deaths and resorptions; the no-effect level was 2.5 mg/kg/day (National Research Council 1986, in HSDB 1989). Pentachlorophenol caused signs of fetotoxicity, including resorptions, subcutaneous edema, dilated ureters, and anomalies of the skull, ribs, and vertebrae, in the offspring of rats dosed orally at a level of 5 to 50 mg/kg per day

(Schwetz, Keeler and Gehring et al. 1974). In an NTP (1989) bioassay, two pentachlorophenol mixtures (technical grade and Dowicide EC–7) were tested for carcinogenicity in male and female B6C3F1 mice. Groups of 50 male and 50 female mice were fed diets containing 100 or 200 ppm technical grade pentachlorophenol, or 100, 200, or 600 ppm Dowicide EC–7, for 2 years. The study found clear evidence of carcinogenicity among male mice fed either mixture, as shown by increased incidences of adrenal medullary and hepatocellular neoplasms, and among female mice fed Dowicide EC–7, as shown by increased incidences of adrenal medullary and hepatocellular neoplasms and hemangiosarcomas. The study found some evidence of carcinogenicity among female mice fed technical grade pentachlorophenol, as evidenced by increased incidences of hemangiosarcomas and hepatocellular neoplasms.

In humans, exposure to pentachlorophenol dust and mist concentrations in excess of 1 mg/m^3 causes pain and irritation of the nose and throat and violent sneezing and coughing (Deichmann and Keplinger 1981; Demidenko 1969, in ACGIH 1986, p. 461). The signs and symptoms of pentachlorophenol poisoning may include profuse diaphoresis, hyperpyrexia, tachycardia, tachypnea, weakness, nausea, vomiting, abdominal pain, headache, anorexia, progressive coma, and death (Wood, Rom, White, and Logan 1983). Acute poisoning also can affect renal and hepatic function (Wood, Rom, White, and Logan et al. 1983). The lowest lethal oral dose reported in humans is 29 mg/kg (RTECS 1990). A worker experienced exfoliation of the epidermal layer of the hands 1 to 2 days after handling a 20 percent sodium pentachlorophenate solution; recovery occurred within 5 days (Nomura 1953, in Hayes 1982, p. 475). After washing a paint brush in a solvent containing 0.4 percent pentachlorophenol, a worker's hands became painful and red; the pain lasted for about 2 hours (Hayes 1982, p. 475). A 32-year-old white male exposed to lumber pretreated with pentachlorophenol suffered from chloracne characterized by many small yellow/white papules over most of his body; the condition improved after 6 weeks of treatment (Cole, Stone, Gates, and Culver 1986). Five incidents of poisoning, including two fatalities, have been reported in workers from two small wood preservative plants; symptoms included fever, severe hyperpyrexia, increased anion gap, and renal insufficiency (Wood, Rom, White, and Logan 1983). Chronic exposure to this substance is associated with an increase in conjunctivitis, chronic sinusitis, bronchitis, polyneuritis, and dermatitis (Proctor, Hughes, and Fischman 1988, p. 398). Repeated exposure to commercial pentachlorophenol caused aplastic anemia in four individuals, pure red cell aplasia in two people, and subsequent Hodgkin's disease and acute leukemia in two of these individuals (Roberts 1983).

Based on this evidence in humans and animals, OSHA preliminarily concludes that pentachlorophenol causes sensory irritation and systemic toxicity. OSHA believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that establishing a TWA limit of 0.5 mg/m^3, and a skin notation, is necessary to significantly reduce these risks of material health impairment. In addition, promulgation of this PEL and the skin notation will make OSHA's limit for this substance consistent across all regulated sectors.

SODIUM AZIDE

CAS: 26628–22–8; Chemical Formula: NaN_3

H.S. No. 1364

There is no current OSHA PEL for sodium azide in construction, maritime, or agriculture. The current ACGIH TLV® for this substance is 0.1 ppm as an instantaneous ceiling. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limits being proposed. OSHA is proposing a ceiling limit of 0.1 for sodium azide as HN_3 and 0.3 mg/m^3 as a ceiling limit for sodium azide NAN_3, both with skin notations. These are the limits recently established in general industry.

Sodium azide is a colorless, crystalline solid. It is used as a chemical intermediate; an herbicide, fungicide, nematocide, and soil fumigant; in automobile air bags; and to decompose nitrites (HSDB 1989).

Sodium azide is a material of high acute toxicity that acts by direct vasodilation. The oral LD_{50}s for rats and mice have been reported to be 27 mg/kg and 19–27 mg/kg, respectively (RTECS 1989). There have been several reported cases of accidental human poisonings involving ingestion of mg quantities of sodium azide; symptoms reported from these cases have included nausea, headaches, sweating, faintness, tachycardia, hypotension, and hyperventilation. Sodium azide is known to produce hypotension in laboratory animals as well as humans. An intravenous dose of 1 mg/kg was reported to lower blood pressure in cats (Graham 1949/Ex. 1–109). In the 1950s, the medicinal usefulness of sodium azide as a hypotensive agent was tested in 30 hypertensive patients. Their hypertension was reduced, but observed side effects included headaches; in addition, 20 of 30 patients developed increased sensitivity to sodium azide, necessitating a reduction in the dose (Black, Zweifach, and Speer 1954/Ex. 1–163). Hicks (1950) reported that repeated intraperitoneal injections of 5 to 10 mg/kg in rats caused demyelination of the nerve fibers of the central nervous system. Alben and Fager (1972) showed that sodium azide formed strong complexes with hemoglobin and blocked oxygen transport in the blood.

In addition to these effects, sodium azide has been reported to cause testicular damage in rats given intraperitoneal injection of doses sufficient to cause intoxication. Reported doses given to monkeys have caused damage to the central nervous system, resulting in blindness and attacks of rigidity.

Acute inhalation by humans of hydrazoic acid vapor (which forms when sodium azide contacts water) results in lowered blood pressure, eye irritation, bronchitis, headache, weakness, and collapse (Fairhall et al. 1943/Ex. 1–130; Graham 1949/Ex. 1–109). The exposure levels that produce these effects were not reported by these authors. Haas and Marsh (1970/Ex. 1–121) reported that exposure to concentrations of hydrazoic acid vapor as low as 0.5 ppm "cause[d] some discomfort to laboratory personnel."

Because of its hypotensive effect in humans, OSHA preliminarily concludes that ceiling limits of 0.1 ppm (measured as HN_3) and 0.3 mg/m^3 (measured as NaN_3) should be established for sodium azide to reduce the significant risk of cardiovascular and irritation effects potentially posed to construction, maritime, and agriculture workers at the levels permitted by the absence of an OSHA limit. To reduce this significant risk substantially, OSHA is proposing to establish these ceiling limits for sodium azide, as well as a skin notation. OSHA believes that the skin notation will alert employers to the fact that sodium azide readily penetrates intact skin and that dermal exposure can contribute significantly to overall worker exposure. In addition, promulgation of these limits will make OSHA's PELs for these substances consistent across all regulated sectors.

1,1,2-TRICHLORO-1,2,2-TRIFLUOROETHANE

CAS: 76–13–1; Chemical Formula: CCl_2FCClF_2
H.S. No. 1403

1,1,2-Trichloro-1,2,2-trifluoroethane (FC–113) is a member of the chlorofluorocarbon family. The current OSHA PEL for this substance in construction and maritime is an 8-hour TWA of 1000 ppm. The ACGIH has an 8-hour TLV®–TWA of 1000 ppm and a 15-minute STEL of 1250 ppm for FC–113. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limits being proposed. The Agency proposes to retain the TWA limit and to add a 1250 ppm STEL in construction and maritime. OSHA has no PEL for this substance in agriculture, and proposes adding a 1000 ppm 8-hour TWA PEL and a 1250 ppm STEL for FC–113 in agricultural workplaces. These are the limits recently established for this substance in general industry.

FC–113 is a colorless, noncombustible liquid. It is used as a chemical intermediate in the production of polychlorotrifluoro-ethylene resins and polychlorotrifluoro-ethylene-vinylidine, as a fire extinguishant and as a foam blowing agent, a drying agent, a degreasing solvent, and a dry cleaning solvent. FC–113 is also used as a refrigerant.

FC–113 has a low potential for acute lethality; the 4-hour LC_{50} in rats has been reported to range from 52,000 to 68,000 ppm (ACGIH 1986). In experimental animals, the principal effects of exposure to FC–113 include tachycardia, myocardial depression, hypotension, and cardiac sensitization. These effects have been seen in monkeys and dogs exposed to 25,000–50,000 ppm (HSDB 1989). Exposure of animals to high concentrations (i.e., 12,000 ppm) has also been found to cause central nervous system depression, respiratory irritation, and mild liver changes (ACGIH 1986).

Cardiac sensitization has been found to result from lower, short-term exposures. Reinhardt, Mullin, and Maxfield (1973/Ex. 1–114) observed that 10 out of 29 dogs exposed to 5000 ppm FC–113 for 5 minutes and simultaneously injected with epinephrine developed serious arrhythmias. Similar experiments, in which the dogs were exposed to 2000 to 2500 ppm of this substance for longer periods of time (from 30 minutes to 6 hours) and simultaneously administered epinephrine, resulted occasionally in arrhythmia (Aviado 1975). However, when the experiment was repeated using four 6-hour exposures to 1000 ppm in conjunction with an injection of epinephrine, no arrhythmias were observed. Cardiac sensitization has also been reported in monkeys exposed to 2000 ppm FC–113 for 6 hours (EPA 1983).

A study by Stopps and McLaughlin (1967/Ex. 1–122) of human volunteers revealed that exposure to 2500 ppm FC–113 for 1.5 hours resulted in impairment of psychomotor performance (described as lethargy and inability to concentrate). This effect was not observed at concentrations below 2500 ppm. Within the first one-half to one hour of exposure to 2500 ppm or more, subjects reported subjective sensations including loss of concentration, a tendency to somnolence, and a feeling of "heaviness" in the head. OSHA believes that the results of the Stopps and McLaughlin (1967/Ex. 1–122) study described above demonstrate that FC–113 can induce subjective effects in humans on short-term exposure. Thus, OSHA preliminarily concludes that a STEL is necessary to prevent these effects among construction, maritime, and agriculture workers. The proposed STEL includes a small safety factor below the human NOEL to account for variabilities in toxic responses.

The evidence described above indicates that FC–113 can exert toxic effects at levels of exposure comparable to the levels that are permitted by excursions above the current OSHA TWA limit of 1000 ppm; OSHA believes that such levels pose a significant risk of cardiac sensitization to exposed workers. The Agency considers the cardiac sensitization induced by FC–113 a material impairment of health and functional capacity. OSHA therefore preliminarily concludes that a STEL of 1250 ppm will provide a wider margin of safety against cardiac sensitization and will reduce the risk of impaired psychomotor performance by limiting the potentially high, short-term exposures permitted by the 1000 ppm 8-hour TWA limit alone. Thus, OSHA proposes limits of 1000 ppm TWA and 1250 ppm STEL for 1,1,2-trichloro-1,2,2-trifluoroethane in construction, maritime, and agriculture to substantially reduce the significant risks associated with exposure to this substance. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

Preliminary Conclusions. Of all the physiological systems, the cardiovascular system is especially vulnerable to occupational hazards because cardiovascular diseases are already so prevalent in our society. According to Levy (1985/Ex. 1–210), "an estimated 40 million Americans have some form of cardiovascular disease." The major risk factors, as revealed by epidemiology, are age, male sex, hypertension, cigarette smoking, the existence of low-density and high-density plasma lipoproteins, cholesterol, and diabetes (Levy 1985/Ex. 1–210). Many American workers exposed to the chemicals grouped on the basis of their cardiovascular effects have one or more of these risk factors and are therefore particularly susceptible to exposure to cardiovascular toxicants. Although the precise interactions among these risk factors and exposures to cardiovascular toxins are difficult to demonstrate with accuracy, few would argue that they do not occur.

OSHA preliminarily concludes that the potential for cardiovascular system damage associated with exposure to these cardiac sensitizers, vasodilators, and atherosclerosis-causing substances poses a significant risk to employees in a broad range of workplaces. The effects experienced by exposed workers include arrhythmia, low blood pressure, stroke, and blockage of the flow of blood to the myocardium. Reducing the exposure limits for these cardiovascular toxins from levels where such effects could occur to concentrations where their occurrence is unlikely will substantially reduce these risks. OSHA believes that the health evidence for these cardiovascular toxins forms a reasonable basis for proposing new or revised limits. At the time of the final rule, OSHA will establish new or revised limits for these substances if the Agency determines that such limits will substantially reduce significant risk.

8. Substances for Which Proposed Limits Are Based on Avoidance of Systemic Toxicity

Introduction. For a number of substances, OSHA's proposed limits are based primarily on evidence that exposure is associated with general systemic toxicity. This group of substances is unique among the groupings discussed in this preamble in that no single organ system can be identified as the target of low-dose exposure to these chemicals. Instead, these substances have been shown either to affect several organ systems simultaneously or to cause a variety of nonspecific adverse signs and symptoms that are indicative of general toxicity.

The 73 substances belonging to this group and their CAS numbers, HS numbers, 1987–1988 ACGIH TLV®s and NIOSH RELs are shown in Table C8–1. The current PELs for these substances in construction and maritime are shown in the second column of Table C8–1, and the limits being proposed in construction, maritime, and agriculture

are shown in the right-hand column of the table. Promulgation of the proposed limits in construction, maritime, and agriculture will make the PELs for these substances consistent across all OSHA-regulated sectors.

TABLE C8-1.—SUBSTANCES FOR WHICH PROPOSED LIMITS A

H.S. number/chemical name	CAS No.	Cu
1005 Acetonitrile	75-05-8	40
1006 Acetylsalicylic acid (Aspirin)	50-78-2	.....
1019 Aluminum (Welding fumes) (as Al)	7429-90-5	.....
2004 2-Aminopyridine	504-29-0	0.5
2007 Anisidine (o,p isomers)	29191-52-4	0.5
2010 Arsine	7784-42-1	0.0
2012 Arsenic, organic compounds (as As)	7440-38-0	0.5
2015 Barium, soluble compounds (as Ba)	7440-39-3-	0.5
1046 2-Butoxyethanol	111-76-2	50
2024 tert-Butyl chromate (as CrO_3)	1188-85-1	0.1 S
1052 n-Butyl glycidyl ether (BGE)	2426-08-6	50
1067 Captan	133-06-2	.....
2026 Carbaryl (Sevin) ®	63-25-2	5 m
1088 beta-Chloroprene	126-99-8	25
2045 Cyanides (as CN)	Varies	5 m
1109 Cyclohexylamine	108-91-8	.....
1112 Cyhexatin	13121-70-5	.....
2050 2,4-D (2,4-Dichlorophenoxyacetic acid)	94-75-7	10
1113 Dichlorodiphenyltrichloroethane (DDT)	50-29-3	1 m
1120 2-N-Dibutylaminoethanol	102-81-8	2 p
2061 Dieldrin	60-57-1	0.2
2063 Difluorodibromomethane-	75-61-6	10
1139 Diglycidyl ether	2238-07-5	0.5
2064 Diisopropylamine	108-18-9	5 p
2067 Dimethylformamide	68-12-2	10
2068 1,1-Dimethylhydrazine	57-14-7	0.5
2070 Dinitro-o-cresol	534-52-1	0.2
1159 Ethanolamine-	141-43-5	3 p
1167 Ethylene chlorohydrin	107-07-3	5 p
2082 Ethylenediamine	107-15-3	10
2084 Fluorides (as F)	Varies	2.5
1189 Glycidol	556-52-5	50
2088 Heptachlor	76-44-8	0.5
1198 Hexafluoroacetone	684-16-2	.....
1207 Hydrogen cyanide	74-90-8	10
2093 Hydrogen selenide (as Se)	7783-07-5	0.0
1210 Hydrogenated terphenyls	61788-32-7	.....
1223 2-Isopropoxyethanol	109-59-1	.....
1227 Isopropyl glycidyl ether (IGE)	4016-14-2	50
2098 Lead, inorganic	7439-92-1	0.2
1273 4,4'-Methylene bis (2-chloroaniline)	101-14-4	.....
2111 Molybdenum, soluble compounds (as Mo)	7439-98-7	5 m
2112 Monomethyl hydrazine	60-34-4-	0.2

ITS ARE BASED ON AVOIDANCE OF SYSTEMIC TOXICITY

	Current OSHA PEL in construction and maritime *	1987–1988 ACGIH TLV® **	NIOSH REL***	Proposed OSHA PEL in construction, maritime, and agriculture*
	40 ppm TWA	40 ppm TWA,	20 ppm TWA; 60 ppm STEL, Skin.	40 ppm TWA; 60 ppm STEL
		5 mg/m³ TWA		5 mg/m³ TWA
		5 mg/m³ TWA		5 mg/m³ TWA
	0.5 ppm TWA	0.5 ppm TWA		0.5 ppm TWA
	0.5 mg/m³ TWA, Skin	0.5 mg/m³ TWA, Skin		0.5 mg/m³ TWA, Skin
	0.05 ppm TWA	0.05 ppm TWA	2 μg/m³ Ceiling	0.05 ppm TWA (15-min)⁺
	0.5 mg/m³ TWA	0.2 mg/m³ TWA		0.5 mg/m³ TWA
	0.5 mg/m³ TWA	0.5 mg/m³ TWA		0.5 mg/m³ TWA
	50 ppm TWA, Skin	25 ppm TWA, Skin		25 ppm TWA, Skin
	0.1 mg/m³ Ceiling, Skin.	0.1 mg/m³ Ceiling, Skin.	1 μg/m³ TWA (as Cr VI)	0.1 mg/m³ Ceiling, Skin
	50 ppm TWA	25 ppm TWA	5.6 ppm Ceiling (15-min)	25 ppm TWA
		5 mg/m³ TWA		5 mg/m³ TWA
	5 mg/m³ TWA	5 mg/m³ TWA–	5 mg/m³ TWA	5 mg/m³ TWA
	25 ppm TWA, Skin	10 ppm TWA, Skin	1 ppm Ceiling (15-min)⁺	10 ppm TWA, Skin
	5 mg/m³ TWA, Skin	5 mg/m³ TWA, Skin	5 mg/m³ Ceiling (10-min)	5 mg/m³ TWA, Skin
		10 ppm TWA		10 ppm TWA
		5 mg/m³ TWA		5 mg/m³ TWA
	10 mg/m³ TWA	10 mg/m³ TWA		10 mg/m³ TWA
	1 mg/m³ TWA, Skin	1 mg/m³ TWA	Reduce exposure to lowest reliably detectable concentration⁺.	1 mg/m³ TWA, Skin
	2 ppm TWA, Skin	2 ppm TWA, Skin		2 ppm TWA
	0.25 mg/m³ TWA, Skin	0.25 mg/m³ TWA, Skin–.	Reduce exposure to lowest reliably detectable concentration⁺	0.25 mg/m³ TWA, Skin
	100 ppm TWA–	100 ppm TWA		100 ppm TWA
	0.5 ppm Ceiling	0.1 ppm TWA	0.2 ppm Ceiling⁺ (15-min)	0.1 ppm TWA
	5 ppm TWA, Skin	5 ppm TWA, Skin		5 ppm TWA, Skin
	10 ppm TWA, Skin	10 ppm TWA, Skin		10 ppm TWA, Skin
	0.5 ppm TWA, Skin	0.5 ppm TWA, Skin, A2.	0.06 ppm Ceilng⁺ (2-hour).	0.5 ppm TWA, Skin
	0.2 mg/m³ TWA, Skin	0.2 mg/m³ TWA, Skin	0.2 mg/m³ TWA	0.2 mg/m³ TWA, Skin
	3 ppm TWA	3 ppm TWA; 6 ppm STEL.		3 ppm TWA; 6 ppm STEL
	5 ppm TWA, Skin	1 ppm Ceiling, Skin		1 ppm Ceiling, Skin
	10 ppm TWA	10 ppm TWA		10 ppm TWA
	2.5 mg/m³ TWA	2.5 mg/m³ TWA	2.5 mg/m³ TWA	2.5 mg/m³ TWA
	50 ppm TWA	25 ppm TWA		25 ppm TWA
	0.5 mg/m³ TWA, Skin	0.5 mg/m³ TWA, Skin		0.5 mg/m³ TWA, Skin
		0.1 ppm TWA, Skin		0.1 ppm TWA, Skin
	10 ppm TWA, Skin	10 ppm Ceiling, Skin–	4.7 ppm Ceiling (10-min)	4.7 ppm STEL, Skin
	0.05 ppm TWA	0.05 ppm TWA–		0.05 ppm TWA
		0.5 ppm TWA		0.5 ppm TWA
		25 ppm TWA–		25 ppm TWA
	50 ppm TWA	50 ppm TWA; 75 ppm STEL.	50 ppm Ceiling (15-min)	50 ppm TWA; 75 ppm STEL
	0.2 mg/m³ TWA	0.15 mg/m³ TWA	<100 μg/m³ TWA	50 μg/m³ TWA
		0.02 ppm TWA, Skin, A2.	3 μg/m³ TWA⁺ (lowest detectable concentration).	0.02 ppm TWA, Skin
	5 mg/m³ TWA	5 mg/m³ TWA		5 mg/m³ TWA
	0.2 ppm Ceiling, Skin	0.2 ppm Ceiling, Skin, A2.	0.04 ppm Ceiling⁺ (2-hour)	0.2 ppm Ceiling, Skin

TABLE C8-1.—SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BA

H.S. number/chemical name	CAS No.	Cu
2114 Nickel, metal and insoluble compounds (as Ni)	7440-02-0	1 m
2115 Nicotine	54-11-5	0.5
2125 Perchloromethyl mercaptan	594-42-3	0.1
2126 Phenol	108-95-2	5 p
1317 Phenylhydrazine	100-63-0	5 p
1318 Phenylphosphine	638-21-1	.....
1321 Phosphine	7803-51-2	0.3
2130 Phosphorus (yellow)	7723-14-0	0.1
1330 Piperazine dihydrochloride	142-64-3	.....
1340 n-Propyl nitrate	627-13-4	25
2136 Pyrethrum	8003-34-7	5 m
2137 Pyridine	110-86-1	5 p
2140 Selenium and compounds (as Se)	7782-49-2	0.2
2143 Silver, metal and soluble compounds (as Ag)	7440-22-4	0.0
1366 Sodium fluoroacetate	62-74-8-	0.0
2145 Strychnine	57-24-9	0.1
2150 Tellurium and compounds (as Te)	13494-80-9	0.1
1386 Tetraethyl lead (as Pb)	78-00-2	0.1
1388 Tetramethyl lead (as Pb)	75-74-1	0.1
2156 Tetramethyl succinonitrile	3333-52-6-	0.5
2158 Tetryl (2,4,6-Trinitrophenyl-methyl-nitramine)	479-45-8	1.5
2160 Thiram-	137-26-8	5 m
2161 Tin (metal and inorganic compounds) (as Sn)	7440-31-5	2 m
1412 Trimethyl benzene	25551-13-7-	25
1416 Tungsten insoluble compounds (as W)	7440-33-7	5 m
1417 Tungsten soluble compounds (as W)	7440-33-7	1 m
1426 Vinylidene chloride (1,1-Dichloroethylene)	75-35-4	.....
1430 Welding fumes (Total particulate)		
1437 Zinc oxide (Fume)	1314-13-2	5 m
1439 Zirconium compounds (as Zr)	7440-67-7	5 m

* OSHA's PELs do not currently apply in agriculture; OSHA's TWA limits are for 8-hour exposures; its STELs
eriod of time.

** The ACGIH TLV®-TWA is for an 8-hour exposure; its STELs are 15-minutes limits not to be exceeded more
and its ceilings are peaks not to be exceeded for any period of time. An A2 designation means that the ACGIH cl

*** NIOSH TWA limits are for 10 hour/day, 40 hour/week exposures unless otherwise specified, and its ceiling

†NIOSH considers this substance a potential occupational carcinogen and recommends that exposures be redu

RE BASED ON AVOIDANCE OF SYSTEMIC TOXICITY—Continued

	Current OSHA PEL in construction and maritime *	1987–1988 ACGIH TLV® **	NIOSH REL***	Proposed OSHA PEL in construction, maritime, and agriculture*
0	1 mg/m³ TWA	1 mg/m³ TWA	15 µg/m³ TWA+	1 mg/m³ TWA
5	0.5 mg/m³ TWA, Skin	0.5 mg/m³ TWA, Skin		0.5 mg/m³ TWA, Skin
0	0.1 ppm TWA	0.1 ppm TWA		0.1 ppm TWA
2	5 ppm TWA, Skin	5 ppm TWA, Skin	5.2 ppm TWA; 15.6 ppm Ceiling (15-min).	5 ppm TWA, Skin
0	5 ppm TWA, Skin	5 ppm TWA, 10 ppm STEL, Skin, A2.	0.14 ppm Ceiling+ (2-hour)	5 ppm TWA; 10 ppm STEL, Skin
		0.05 ppm Ceiling		0.05 ppm Ceiling
2	0.3 ppm TWA	0.3 ppm TWA, 1 ppm STEL.		0.3 ppm TWA; 1 ppm STEL
0	0.1 mg/m³ TWA	0.1 mg/m³ TWA		0.1 mg/m³ TWA
3		5 mg/m³ TWA		5 mg/m³ TWA
4	25 ppm TWA	25 ppm TWA; 40 ppm STEL.		25 ppm TWA; 40 ppm STEL
7	5 mg/m³ TWA	5 mg/m³ TWA		5 mg/m³ TWA
1	5 ppm TWA	5 ppm TWA		5 ppm TWA
2	0.2 mg/m³ TWA	0.2 mg/m³ TWA		0.2 mg/m³ TWA
4	0.01 mg/m³ TWA	0.1 mg/m³ TWA (metal); 0.01 mg/m³ TWA (soluble compounds).		0.01 mg/m³ TWA
–	0.05 mg/m³ TWA, Skin	0.05 mg/m³ TWA; 0.15 mg/m³ STEL, Skin.		0.05 mg/m³ TWA; 0.15 mg/m³ STEL, Skin
9	0.15 mg/m³ TWA	0.15 mg/m³ TWA		0.15 mg/m³ TWA
9	0.1 mg/m³ TWA	0.1 mg/m³ TWA		0.1 mg/m³ TWA
2	0.1 mg/m³ TWA, Skin	0.1 mg/m³ TWA, Skin		0.075 mg/m³ TWA, Skin
1	0.15 mg/m³ TWA, Skin	0.15 mg/m³ TWA, Skin		0.075 mg/m³ TWA, Skin
–	0.5 ppm TWA, Skin	0.5 ppm TWA, Skin	1 ppm Ceiling (15-min)	0.5 ppm TWA, Skin
8	1.5 mg/m³ TWA, Skin	1.5 mg/m³ TWA, Skin		1.5 mg/m³ TWA, Skin
8	5 mg/m³ TWA	5 mg/m³ TWA		5 mg/m³ TWA
5	2 mg/m³ TWA	2 mg/m³ TWA		2 mg/m³ TWA
–	25 ppm TWA	25 ppm TWA		25 ppm TWA
7	5 mg/m³ TWA	5 mg/m³ TWA; 10 mg/m³ STEL.	5 mg/m³ TWA	5 mg/m³ TWA; 10 mg/m³ STEL
7	1 mg/m³ TWA	1 mg/m³ TWA; 3 mg/m³ STEL.	1 mg/m³ TWA	1 mg/m³ TWA; 3 mg/m³ STEL
4		5 ppm TWA; 20 ppm STEL.	Controlled as specified for vinyl chloride in 29 CFR 1910.1017, with eventual goal of zero exposure+.	1 ppm TWA
		5 mg/m³ TWA		5 mg/m³ TWA
2	5 mg/m³ TWA	5 mg/m³ TWA; 10 mg/m³ STEL.	5 mg/m³ TWA; 15 mg/m³ Ceiling (15-min).	5 mg/m³ TWA; 10 mg/m³ STEL
7	5 mg/m³ TWA	5 mg/m³ TWA; 10 mg/m³ STEL.		5 mg/m³ TWA; 10 mg/m³ STEL

TELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any

more than 4 times in any working day, with a minimum of 60 minutes between successive STEL exposures;
GIH classifies the substance as a suspected human carcinogen.
ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.
e reduced to the lowest feasible concentration.

Description of the Health Effects. For each substance included in this grouping, limits have been proposed to protect workers in construction, maritime, and agriculture against a variety of adverse exposure-related effects that are manifested at multiple target organ sites. In some instances, the nature of the toxic effects associated with exposure is well-defined and clearly understood (for example, CNS depression, histological organ changes, embryotoxicity, methemoglobinemia, conjunctivitis, liver and kidney damage, testicular damage). The effects of exposure to other substances in this group, however, have been demonstrated only by such nonspecific indicators as dizziness, respiratory irritation, hematuria, chest tightness, weight loss or decreased rate of weight gain, lethargy, loss of appetite, nervousness, or gastrointestinal disturbances. Although the specificity of the systemic effect caused by exposure to the substances in this group may vary, all of these substances have been shown to be biologically active in mammalian species, to interfere significantly with biological processes, and to impair normal organ function.

Table C8–2 summarizes the toxic exposure-related effects reported for these substances in humans and experimental animals; this evidence supports the limits being proposed for these substances. This table shows the variety of adverse health effects that adoption of the proposed rule's limits will minimize or prevent. The table also shows that, for the vast majority of substances in this group, the risks of exposure have been defined in studies of humans or animals and are known to include respiratory effects, neurological effects, adverse effects on the reproductive system, organ damage, hematopoietic effects, sensitization, and mucosal irritation. All of these effects are indicative of generalized systemic effects rather than localized effects occurring at the site of chemical contact, and all of them are considered material impairments of health within the meaning of the Act.

Dose-Response Relationships and Systemic Effects. As Table C8–2 shows, adverse toxic reactions have been reported to occur in humans for more than half of the substances in this group; for these substances, it has thus been established conclusively that exposure is associated with adverse health effects in humans. Experimental animal data comprise the principal evidence for the toxicologic action of the remaining substances. As is the case for many substances for which limits are being proposed in this rulemaking, apparent no-observed-effect levels, supplemented by the use of appropriate margins of protection, provide the basis for setting the limits. The systemic effects caused by exposure to substances in this group appear to follow a no-observed-effect (NOE) dose-response pattern. That is, as intensity and/or duration of exposure decreases, the severity of the effect on organ systems also decreases until a point is reached (the NOE level) where there is no detectable effect, at least at observable levels, on organ systems. No-effect exposure levels have been identified in humans and animals for several of the substances in this group; where no-effect levels have been identified (i.e., for diglycidyl ether and phenylphosphine), they have provided the primary basis for the proposed limits. In instances where no-effect levels have not been reported (e.g., for n-butyl glycidyl ether, trimethylbenzene, and acetylsalicylic acid), OSHA has used safety factors and expert judgment to derive an NOE value.

TABLE C8–2.—SUMMARY OF ADVERSE HEALTH EFFECTS REPORTED FOR SUBSTANCES PRODUCING GENERAL SYSTEMIC TOXICITY

H.S. number/chemical name	Effects reported in humans	Effects reported in animals
1005 Acetonitrile	Tightness in chest; flushing of face	Embryotoxicity; teratogenicity at maternally toxic doses; liver, blood changes.
1006 Acetylsalicylic acid	Mucosal irritation; respiratory sensitization; internal bleeding.	Teratogenicity at high doses.
1019 Aluminum welding fumes–	No data	Respiratory effects.
2004 2-Aminopyridine	Headache, nausea; increased blood pressure	Convulsions; respiratory distress.
2007 Anisidine	Methemoglobinemia; sensitization	Methemoglobinemia, cancer.
2010 Arsine–	Hemolysis; renal failure	Blood changes.
2012 Arsenic, organic compounds	Optic neuritis; encephalitis	Hepatitis.
2015 Barium, soluble compounds	Baritosis; irritation	Irritation; central nervous system effects.
1046 2–Butoxyethanol	Mild sensory irritation	Severe hemoglobinuria; lung, kidney, liver changes; hemolytic anemia; increased osmotic fragility in erythrocytes.
2024 tert-Butyl chromate	Lung, sinus cancer; narcosis; skin necrosis	Central nervous system effects; skin necrosis.
1052 n-Butyl glycidyl ether–	Dermatitis; skin sensitization	Delirium; depression.
1067 Captan–	Recurrent urticaria	Decreased fertility index in males; polyploid carcinoma of duodenum.
2026 Carbaryl	Cholinesterase inhibition	Irritation; reproductive effects; nervous system effects.
1088 beta-Chloroprene	CNS depression; lung, liver, kidney injury; conjunctivitis; necrosis of cornea; lowering of blood pressure.	Minimal systemic effects.
2045 Cyanides	Headache; central nervous system effects; skin corrosion.	Ocular effects, respiratory irritation.
1109 Cyclohexylamine	Acute toxicity; sensitization	Mutagenic and reproductive effects.
1112 Cyhexatin	No data	Microscopic changes in liver, kidneys, adrenal glands.
2050 2,4-D (2,4-Dichlorophenoxy-acetic acid)	Coma; eczema; muscular weakness; peripheral neuropathy.	Central nervous system effects; liver and kidney damage; reproductive and teratogenic effects.
1113 Dichlorodiphenyl-trichloro-ethane (DDT)	Mild systemic poisoning	Cancer of liver, lungs, lymph system.
1120 2-N-Dibutylamino-ethanol	No data	Weight loss; elevated liver- and kidney-to-body-weight ratios.
2061 Dieldrin	Convulsions; headache; nausea	Convulsions; fetotoxicity; cancer.
2063 Difluorodibromomethane–	No data	Respiratory irritation; central nervous system and liver effects.
1139 Diglycidyl ether	Mucosal irritation	CNS depression; clouding of cornea; respiratory irritation; hematopoietic effects.

TABLE C8–2.—SUMMARY OF ADVERSE HEALTH EFFECTS REPORTED FOR SUBSTANCES PRODUCING GENERAL SYSTEMIC TOXICITY—Continued

H.S. number/chemical name	Effects reported in humans	Effects reported in animals
2064 Diisopropylamine	Irritation; corneal edema; dermatitis	Irritation.
2067 Dimethylformamide	Liver, kidney, lung, cardiovascular damage; cancer.	Irritation; lung, liver, kidney damage; reproductive effects.
2068 1,1-Dimethylhydrazine	Dyspnea; nausea; liver damage	Central nervous system and blood effects; cancer.
2070 Dinitro-o-cresol	Hyperpyrexia; nausea; tachycardia	Coma; metabolic stimulation; liver and kidney damage.
1159 Ethanolamine	No data	Pulmonary, hepatic, and renal lesions; decreased alertness; temporary weight loss.
1167 Ethylene chlorohydrin	Liver and brain damage; mucosal irritation; gastrointestinal disturbances.	Respiratory depression; liver and kidney damage.
2082 Ethylenediamine	Sensory irritation; skin, pulmonary sensitization.	Irritation; liver and kidney damage.
2084 Fluorides	Sensory irritation; central nervous system effects; fluorosis.	Irritation; convulsions; teeth mottling.
1189 Glycidol	No data	Pneumonitis; emphysema.
2088 Heptachlor	Convulsions; blood dyscrasias; aplastic anemia.	Liver toxicity; cancer.
1198 Hexafluoroacetone	No data	Renal dysfunction; increased lung weight; testicular damage; hematopoietic effects; fetotoxicity.
1207 Hydrogen cyanide	Cyanide poisoning; weakness; mucosal irritation; colic; nervousness; enlargement of thyroid.	None reported.
2093 Hydrogen selenide	Severe irritation; nausea; fatigue	Severe respiratory irritation.
1210 Hydrogenated terphenyls	No data	Decreased weight gain; liver, kidney damage; lung changes, bronchopneumonia.
1223 2-Isopropoxyethanol	No data	Anemia; hemoglobinuria; lung congestion.
1227 Isopropyl glycidyl ether	Mucosal irritation	Reduced weight gain; hemoglobin increase; emphysematous changes in lungs; CNS depression.
2098 Lead (inorganic)	Central nervous system damage, reproductive effects, urinary tract damage, blood effects.	Nervous system effects; reproductive effects.
1273 4,4'-Methylene-bis (2-chloroaniline)	Bladder cancer; hematuria	Cyanosis; methemoglobinemia; liver, lung tumors.
2111 Molybdenum, soluble compounds	Irritation; anemia, liver damage; pneumoconioses.	Irritation; central nervous system effects; liver and kidney damage.
2112 Monomethyl hydrazine	Convulsions; blood effects; liver damage	Central nervous system effects; liver damage.
2114 Nickel, metal and insoluble compounds	Dermal sensitization; cancer; kidney damage	Pulmonary damage; lung cancer.
2115 Nicotine	Convulsions; prostration; tachycardia	Central nervous system stimulation and depression; convulsions; teratogenicity.
2125 Perchloromethyl mercaptan	Severe respiratory irritation; liver and kidney damage.	Severe respiratory irritation.
2126 Phenol	Irritation; central nervous system effects	Irritation; central nervous system effects.
1317 Phenylhydrazine	Skin sensitization	Anemia; irregular growth; general weakness; blood vessel tumors.
1318 Phenylphosphine	No data	Mild hemolytic anemia; testicular degeneration; hind leg tremor; nausea; loss of appetite; hypersensitivity to sound and touch.
1321 Phosphine	Pulmonary edema; gastrointestinal disturbances; dizziness.	Respiratory irritation.
2130 Phosphorous (yellow)	Severe respiratory irritation; liver damage; anemia; "phossy jaw" (bone necrosis).	Eye, skin burns; liver, kidney damage; severe respiratory irritation.
1330 Piperazine dihydrochloride	Skin burns; sensitization; asthma	No data.
1340 n-Propyl nitrate	No data	Cyanosis; methemoglobinemia; hypotension; respiratory depression.
2136 Pyrethrum	Dermatitis; irritation; skin sensitization	Central nervous system effects; irritation.
2137 Pyridine	Irritation; central nervous system effects; liver and kidney damage.	Irritation; narcosis; liver and kidney damage.
2140 Selenium and compounds	Irritation; chills; fever; garlic breath	Central nervous system effects; irritation; hair loss; liver damage.
2143 Silver, metal and soluble compounds	Localized or generalized argyria; irritation; kidney and liver damage.	Argyria; kidney damage.
1366 Sodium fluoroacetate	No data	Fluctuation in growth rate; tissue changes.
2145 Strychnine	Convulsions; respiratory paralysis	Convulsions; paralysis.
2150 Tellurium and compounds	Neurotoxicity; garlic breath; liver and kidney damage.	Central nervous system effects; teratogenicity.
1386 Tetraethyl lead		
1388 Tetramethyl lead		
2156 Tetramethyl succinonitrile	Headaches; convulsions; coma	Convulsions; respiratory paralysis.
2158 Tetryl	Irritation; dermal sensitization; pulmonary sensitization; liver damage.	Kidney damage.
2160 Thiram	Irritation; skin sensitization; metabolic stimulation.	Demyelination; dermal sensitization; teratogenicity.
2161 Tin (metal and inorganic compounds)	Irritation; anemia; gastrointestinal symptoms	Granulomas; internal bleeding; anemia; liver damage.
1412 Trimethylbenzene	Nervousness, tension, anxiety; asthmatic bronchitis; hypochromic anemia.	CNS depression; Lymphopenia; neutrophilia.

TABLE C8-2.—SUMMARY OF ADVERSE HEALTH EFFECTS REPORTED FOR SUBSTANCES PRODUCING GENERAL SYSTEMIC TOXICITY—Continued

H.S. number/chemical name	Effects reported in humans	Effects reported in animals
1416 Tungsten insoluble compounds	No data	Gross changes in liver and spleen; lung tissue changes.
1417 Tungsten soluble compounds	No data	Generalized cellular asphyxiation; colic; incoordination; dyspnea.
1428 Vinylidene chloride	No data	Nasal irritation; liver cell degeneration; retarded weight gain; embryotoxicity; kidney adenocarcinoma.
1430 Welding fumes (total particulate)	Pulmonary irritation	Pulmonary irritation.
1437 Zinc oxide (fume)	Metal fume fever; gastritis	No data.
1439 Zirconium compounds	No data	Toxic effects from zirconium tetrachloride due to liberation of hydrochloric acid.

The following discussions describe OSHA's preliminary findings for these systemic toxicants and present a summary of the material impairments of health potentially associated with exposure to these substances.

ACETONITRILE

CAS: 75-05-8; Chemical Formula: CH_3CN

H.S. No. 1005

OSHA's occupational exposure limit for acetonitrile in construction and maritime is 40 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a 40 ppm TLV®-TWA and a 60-ppm TLV®-STEL, in addition to a skin notation. NIOSH has a TWA-REL of 20 ppm for acetonitrile. OSHA is proposing to retain the 40-ppm 8-hour TWA in construction and maritime, to add a STEL of 60 ppm for 15 minutes in these two sectors, and to extend both limits to agriculture. These are the limits recently established for acetonitrile in general industry.

Acetonitrile is a colorless liquid with an aromatic odor. It is most widely used in industry as a specialty solvent and chemical intermediate.

In animal studies, acetonitrile has been found to be embryotoxic and teratogenic in rodents exposed to levels sufficiently high to cause maternal toxicity (Berteau, Levinskas, and Rodwell 1982/Ex. 1-179; Willhite 1983/Ex. 1-43). A 13-week inhalation study conducted by the National Toxicology Program (Hazelton Laboratories, Inc. 1983, as cited in ACGIH 1986/Ex. 1-3, p. 8) found pathological changes in the liver and some blood changes in mice and rats exposed to concentrations of 400 ppm acetonitrile.

The human evidence describing the toxic effects associated with exposure to acetonitrile consists of a report by Pozzani, Carpenter, Palm et al. (1959/Ex. 1-106), who exposed human subjects to acetonitrile vapor, and a case report by Amdur (1959/Ex. 1-143), who described a poisoning incident involving acetonitrile. None of three subjects exposed to 40 ppm for 4 hours reported any adverse responses during the exposure period, but one subject experienced tightness of the chest a few hours after termination of exposure, as well as a cooling sensation in the lungs the following day. None of the subjects had elevated blood cyanide levels; one subject showed a slightly elevated urinary thiocyanate level. Pozzani et al. (1959/Ex. 1-106) also exposed two subjects to 80 ppm and 160 ppm of acetonitrile for 4 hours. When exposed to 80 ppm, subjects reported no adverse response; however, at 160 ppm, one subject experienced slight flushing of the face and chest tightness a few hours after exposure (Pozzani, Carpenter, Palm et al. 1959/Ex. 1-106).

In addition to the Pozzani et al. (1959/Ex. 1-106) study, NIOSH (1978g/Ex. 1-262) cites a report by Amdur (1959/Ex. 1-143), who investigated an incident in which 16 painters became ill (with one death) after using an acetonitrile-containing material in a confined space. Amdur (1959/Ex. 1-143) reported no further incidents after adequate ventilation was installed and acetonitrile levels were maintained at about 17 ppm. NIOSH concluded that exposure to 40 ppm produced only minimal effects and that no observable effects were produced at 17 ppm (NIOSH 1978g/Ex. 1-262, p. 97).

Based on this evidence, OSHA preliminarily concludes that long-term exposure to 40 ppm causes no or only minimal effects and that a short-term limit of 60 ppm will provide protection against the facial flushing and chest tightness experienced by workers exposed for several hours to levels above this concentration. In construction and maritime, OSHA is therefore proposing to retain the 8-hour TWA of 40 ppm for acetonitrile and to add a STEL of 60 ppm; OSHA is also proposing to extend both limits to agriculture. The Agency preliminarily concludes that the proposed limits will prevent the significant risk of acute illness (and, in one case, death) observed in workers exposed to excessive short-term levels of acetonitrile; the Agency believes that these health effects clearly constitute material impairments of health. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

ACETYLSALICYLIC ACID (ASPIRIN)

CAS: 50-78-2; Chemical Formula: $CH_3COOC_6H_4COOH$

H.S. No. 1006

In construction, maritime, and agriculture, there is no PEL for acetylsalicylic acid. The ACGIH has a TLV® of 5 mg/m³ for this substance as an 8-hour TWA. NIOSH has no REL but concurs (Ex. 8-47, Table N1) with the limit being proposed. OSHA is proposing a 5 mg/m³ 8-hour TWA PEL for acetylsalicylic acid in construction, maritime, and agriculture. This is the limit recently established in general industry.

Acetylsalicylic acid is a white crystal or powder that is essentially odorless and has a slightly bitter taste. It is used in medicine as an anti-inflammatory drug, an anti-pyretic, and as an analgesic (Hawley's 1987, p. 103).

The work of O'Brien (1968/Ex. 1-47) reports that a normal therapeutic dose of 600 mg aspirin will interfere with platelet aggregation in subjects exposed for a period of 5 days or more. Hart (1947/Ex. 1-137) also reported that 150 mg is the smallest oral dose of acetylsalicylic acid that will produce this effect. Unpublished data from the Dow Chemical Company (cited in ACGIH 1986/Ex. 1-3, p. 10) indicate that exposure to aspirin concentrations exceeding 100 mg/m³ are tolerated except for occasional skin irritation. However, no data are available on the long-term effects on organ systems of inhalation exposure to aspirin. Secondary sources report that aspirin is

an acute irritant to the gastric mucosa and respiratory tract.

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ for acetylsalicylic acid in the construction, maritime, and agriculture sectors. The Agency preliminarily concludes that this limit will protect workers in these sectors from experiencing adverse blood effects and gastric and respiratory irritation, which are potentially associated with exposure to this substance in the workplace. OSHA believes that these effects constitute material health impairments and that the proposed limit is necessary to substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ALUMINUM (WELDING FUMES)
CAS: 7429–90–5; Chemical Formula: Al
H.S. No. 1019

OSHA has no permissible exposure limit for aluminum welding fumes in the construction, maritime, or agriculture industry. The ACGIH TLV®–TWA for these fumes is 5 mg/m³. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing a TWA–PEL for aluminum welding fumes of 5 mg/m³, measured as aluminum. This is the limit recently established for these fumes in general industry.

The proposed PEL addresses the aluminum fume that is released in the welding process; this limit is being proposed to keep total aluminum particulate concentrations low enough to prevent aluminum particle accumulation in the lungs of exposed workers. However, to the extent either that other toxic substances or materials are released in the welding process or that conditions are conducive to the formation of toxic gases, employers are required to pay attention to the permissible exposure limits for these substances as well. For example, in appendix B of the 1987–88 Threshold Limit Values and Biological Exposure Indices (ACGIH 1987/Ex. 1–16), the ACGIH states that "reactive metals and alloys such as aluminum and titanium are arc-welded in a protective, inert atmosphere such as argon. These arcs create relatively little fume, but an intense radiation which can produce ozone" (ACGIH 1987/Ex. 1–16, appendix B, p. 42). In such an instance, employers would be required to meet any ozone limits in effect (PELs of 0.1 ppm TWA and 0.3 ppm STEL are being proposed for ozone) as well as the PEL for aluminum welding fumes.

The ACGIH states that "most welding, even with primitive ventilation, does not produce exposures inside the welding helmet above 5 mg/m³. That which does * * * should be controlled" (ACGIH 1987/Ex. 1–16, appendix B, p. 43). In those rare instances where internal helmet exposures do exceed 5 mg/m³, employees are at risk from the irritant effects of hot metal fumes, which are deposited in the lung deeply and accumulate there.

Because workers in construction, maritime, and agriculture who are exposed to arc welding fumes have previously not been protected by a permissible exposure limit, OSHA is proposing a PEL of 5 mg/m³ TWA for these fumes (measured in the breathing zone of the welder); the details of the appropriate positioning of the sampler should be determined on the basis of guidance in the Field Operations Manual (OSHA 1984). The Agency preliminarily concludes that the proposed limit will protect welders and other workers in the vicinity of the welding from experiencing the significant irritation potentially associated with inhalation of these fumes. OSHA believes that the respiratory irritation caused by exposure to these fumes constitutes a material health impairment and that the proposed PEL is necessary to substantially reduce this significant risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

2–AMINOPYRIDINE
CAS: 504–29–0; Chemical Formula: $C_5H_6N_2$
H.S. No. 2004

In general industry, construction, and maritime, OSHA's current permissible exposure limit for 2-aminopyridine is 0.5 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV® of 0.5 ppm as an 8-hour TWA for this substance; NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 0.5 ppm for 2-aminopyridine in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all regulated sectors.

2-Aminopyridine is a moderately flammable solid that occurs as white leaflets, colorless crystals, or as a white powder; it has a characteristic, intense odor. 2-Aminopyridine is used in the synthesis of antihistamines and other pharmaceuticals; as an intermediate in the synthesis of herbicides; and in tire manufacture (ACGIH 1986, p. 24; Merck 1983, p. 71; Hawley's 1987, p. 62; Grayson 1985, pp. 979–980). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

2-Aminopyridine is a systemic poison that causes headache, respiratory distress, intoxication, convulsions, and death. The LD_{50} in mice by intraperitoneal injection is 28 mg/kg; the LD_{50} in the same species by intravenous administration is 23 mg/kg (RTECS 1990). Acutely poisoned animals showed excitement, tremors, and convulsions before death (Clayton and Clayton 1981, p. 2731). Cats injected intravenously with a 1-mg/kg dose of 2-aminopyridine showed increased blood pressure and respiratory rate, with central nervous system stimulation and muscle twitching. 2-Aminopyridine is readily absorbed through the skin and may cause convulsions by this route (Clayton and Clayton 1981, p. 2732). Application of a 0.02 M solution of 2-aminopyridine to rabbit eyes from which the corneal epithelium had been removed caused slight or no injury (Grant 1986, p. 383).

A worker in a chemical plant who was acutely overexposed to aminopyridine by inhalation and skin absorption died (Spolyar 1951). In another case, a worker exposed to a 5-ppm concentration of 2-aminopyridine for 5 hours complained of headache and nausea, developed increased blood pressure, but subsequently recovered (Clayton and Clayton 1981, p. 2732).

Based on this evidence in humans and animals, OSHA proposes to establish an 8-hour TWA PEL for 2-aminopyridine of 0.5 ppm in agriculture; adoption of this proposed limit would establish the same PEL for workplaces in all OSHA-regulated industry sectors. The Agency preliminarily concludes that occupational exposure to 2-aminopyridine causes systemic poisoning in the form of central nervous system effects and convulsions. Accordingly, OSHA believes that, in the absence of a permissible exposure limit, workers in agriculture are potentially at significant risk for these exposure-related effects and that the proposed PEL will substantially reduce these risks.

ANISIDINE (o-, p-isomers)
CAS: 29191–52–4; Chemical Formula: $NH_2C_6H_4OCH_3$
H.S. No. 2007

OSHA's current limit for the ortho- and para-isomers of anisidine in general industry, construction, and maritime workplaces is 0.5 mg/m³ as an 8-hour time-weighted average; this limit also has a skin notation, which indicates that percutaneous absorption is a significant

route of exposure for this substance. OSHA has no PEL for anisidine in agriculture. The ACGIH's current TLV®–TWA for this substance is 0.5 mg/m³, with a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N6A) with the limit being proposed. OSHA is proposing to establish a TWA PEL of 0.5 mg/m³, and a skin notation, for anisidine in agriculture. Promulgation of this limit will make OSHA's PEL for anisidine consistent across all regulated sectors.

Anisidine has two isomers that find use in industry: the ortho-isomer, which is a reddish-brown or yellowish oil, is used in the chemical synthesis of guaiacol and various dyes, while the para-isomer, which is a fused crystalline mass, is used to produce azo dyes (ACGIH 1986, p. 31). Both isomers have the fishy odor that is characteristic of amines (Sittig 1985, p. 80).

Acute exposure to anisidine causes methemoglobinemia in animals; chronic exposure causes cancer (Proctor, Hughes, and Fischman 1988, p. 76). The oral LD_{50}s in rats are 2 and 1.4 g/kg, respectively, for the o- and p-isomer (RTECS 1990). The dermal LD_{50} in rats for para-anisidine is 3200 mg/kg (RTECS 1990). Subacutely poisoned animals showed hematological changes, anemia, and kidney damage (Proctor, Hughes, and Fischman 1988, p. 77). When fed to mice or rats for 7 weeks, o-anisidine hydrochloride caused enlargement of the spleen, reductions in weight gain, an increased incidence of bladder hyperplasia (mice) and non-neoplastic lesions of the thyroid gland and kidney (rats) (IARC 1982, pp. 74–75). Oral administration of p-anisidine to mice and rats in a subchronic study also caused weight gain reductions and darkened spleens in animals of both species (IARC 1982, p. 75). In cancer bioassays in mice and rats, the oral administration of o-anisidine hydrochloride, but not p-anisidine hydrochloride, caused statistically significant increases in the incidence of transitional-cell carcinomas of the urinary bladder (IARC 1982, p. 76). Based on this evidence, the International Agency for Research on Cancer (IARC) has concluded that the evidence for the carcinogenicity of ortho-anisidine in animals is sufficient (IARC 1982, p. 77).

Workers exposed to a 1.9-mg/m³ concentration of anisidine for 3.5 hours per day for 6 months complained of headaches and dizziness and had an increase in sulfhemoglobin, methemoglobin, and Heinz bodies in their red blood cells (ACGIH 1986, p. 31). Anisidine is also an irritant and sensitizer of the skin in humans (HSDB 1986). Based on effects seen in animals, IARC believes that o-anisidine should be regarded "* * * as if it presented a carcinogenic risk to humans" (IARC 1982, p. 77).

The evidence described above demonstrates that anisidine is a chemical asphyxiant that produces anoxia by binding to hemoglobin and that it is also an irritant and sensitizer of the skin and a probable human carcinogen. OSHA therefore preliminarily concludes that the absence of a limit for anisidine poses a significant risk of skin effects, methemoglobinemia, and potential carcinogenicity in exposed workers in agriculture. The Agency believes that the proposed TWA PEL of 0.5 mg/m³, with a skin notation, is necessary to substantially reduce these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ARSENIC and organic compounds
CAS: 7440–38–2; Chemical Formula:
Varies with compound
H.S. No. 2012

OSHA's limit for arsenic and the organic compounds of arsenic in general industry, construction, and maritime is 0.5 mg/m³ (measured as arsenic) as an 8-hour TWA. There is no PEL in agriculture. The ACGIH TLV®–TWA is 0.2 mg/m³; NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL of 0.5 mg/m³ for arsenic and the organic compounds of arsenic in agriculture. This action would make OSHA's limit for these substances consistent across all regulated sectors.

Arsenic is a nonmetallic element that is silver-gray and crystalline in appearance; examples of its organic compounds are arsenic diethyl, arsenic dimethyl, 1-(p-arsenosophenyl)urea, arsine-tri-1-piperidinium chloride, (3-(p-arsonophenyl)ureido)dithiobenzoic acid, and arsphenoxide (Sax and Lewis 1989, pp. 296–307). Arsenic is an ingredient in rodenticides, insecticides, herbicides, and other products, and the organic compounds are used in both human and animal medicine (Gosselin, Smith, and Hodge 1984, p. III–42). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Arsenic and its organic compounds are considerably less toxic than the oxides and other inorganic compounds, and most of the literature therefore deals with the inorganic compounds. The LD_{50} in rats for arsenic is 763 mg/kg (RTECS 1990), and the LC_{50} for arsenic dimethyl in the same species is 3900 mg/m³ (Stevens, DiPasquale, and Farmer 1979). Four of five cows given a single 10-mg/kg oral dose of the monosodium salt of methanarsenic acid died (Dickinson 1972, in Am. J. Vet. Res. 33:1889–1892), and chronic feeding of 50 mg/kg of the same substance caused toxic hepatitis in rabbits (Exon, Harr, and Claeys 1974, in Nutr. Rep. Int. 9:351–357). Carcinogenicity assays that have involved the oral administration of dimethylarsenic acid in mice or the subcutaneous injection of dimethylarsenic acid in the same species have been negative (IARC 1980, Vol. 23, p. 112).

Human toxicity data for pure arsenic or the organic compounds are rare. Some of the drugs used to combat syphilis in the early part of the century contained organic arsenic compounds, and therapeutic administration of these drugs often caused optic neuritis, which sometimes progressed to loss of central vision and optic atrophy (Grant 1986, p. 118). The administration of one such drug, arsphenamine, is known to have caused encephalitis, with headache, loss of consciousness, convulsions, and paralysis of some of the cranial nerves (Grant 1986, p. 119).

In agriculture, OSHA is proposing an 8-hour TWA PEL of 0.5 mg/m³ for arsenic and its organic compounds (measured as As). The Agency preliminarily concludes that this limit is necessary to reduce the significant risk of arsenic toxicity potentially associated with exposure to these substances. OSHA considers arsenic intoxication a material impairment of health and believes that the new limit is necessary to substantially reduce this risk among workers in agriculture. In addition, promulgation of this limit will make the PEL for arsenic and its organic compounds consistent across all OSHA-regulated sectors.

ARSINE
CAS: 7784–42–1; Chemical Formula:
AsH_3
H.S. No. 2010

OSHA's permissible exposure limit for arsine in general industry, construction, and maritime workplaces is 0.05 ppm as an 8-hour TWA. The Agency has no limit for this substance in agriculture. The ACGIH TLV®–TWA for arsine is also 0.05 ppm. NIOSH has a REL for arsine of 2μg/m³ as a 15-minute ceiling. OSHA is proposing a TWA PEL for arsine of 0.05 ppm in agricultural workplaces. This action will make OSHA's PEL for arsine consistent across all regulated sectors.

Arsine is a colorless gas that has a garlic-like odor (ACGIH 1986, p. 39). Arsine is evolved when nascent hydrogen is liberated in a solution that contains inorganic arsenic; for example, arsine is liberated when arsenic-containing metals are pickled or treated with reducing acids (Clayton and Clayton 1981, p. 1518). Another source of potential exposures to arsine occurs in sulfuric acid production; if arsenic is not completely removed from the feed gases, arsine is liberated when iron comes into contact with the arsenic-containing acids (Clayton and Clayton 1981, p. 495). Arsine could also be liberated if a metal dropped into a container of arsenical pesticide or if such a pesticide were mixed in a metal vat. Arsine finds industrial use in the laboratory analysis of arsenic and as a doping agent in the semiconductor industry (Proctor, Hughes, and Fischman 1988, p. 83; Clayton and Clayton 1981, p. 1519).

Arsine is a highly toxic gas that causes nearly instantaneous death, acute renal failure, and severe intramuscular hemolysis. When inhaled, arsine is oxidized to form elemental trivalent arsenic and arsenous oxide, both recognized human carcinogens (Proctor, Hughes, and Fischman 1988, p. 831). The extent of the red blood cell breakdown caused by the inhalation of this gas is sufficient to cause the sclera of the eye and the skin to turn an intense orange color (jaundice) (Grant 1986, p. 119; Proctor, Hughes, and Fischman 1988, p. 83). Short-term inhalation of a 250-ppm concentration of this gas may cause death within 30 minutes, and inhalation even of a 10- or 50-ppm concentration for a longer period can be fatal (Proctor, Hughes, and Fischman 1988, p. 83). Symptom onset may be delayed for 2 to 24 hours; the signs and symptoms of arsine poisoning include headache, abdominal pain, weakness, dizziness, vomiting, and dark red or brown urine (hematuria) (Proctor, Hughes, and Fischman 1988, p. 83). Workers exposed for 8 months to arsine at concentrations described as "very small" had substantially reduced hemoglobin levels. Some workers had levels as low as 3.2 g/100 ml blood); these workers were involved in gold extraction operations involving cyanide leaching (Proctor, Hughes, and Fischman 1988, p. 83).

Animals exposed to arsine concentrations between 0.5 and 2 ppm for 3 hours/day for several weeks developed blood changes (ACGIH 1986, p. 39). Arsine has been responsible for several hundred fatal poisonings in humans (ACGIH 1986, p. 39).

Based on this evidence, OSHA preliminarily concludes that workers in agriculture are at significant risk of experiencing hemolysis as a result of exposure to arsine at the levels permitted by the absence of a limit for this substance. Accordingly, OSHA is proposing to substantially reduce this risk by establishing an 8-hour TWA PEL for arsine of 0.05 ppm. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

BARIUM, SOLUBLE COMPOUNDS (as Ba)

CAS: 7440–39–3; Chemical Formula: Ba
H.S. No. 2015

In general industry, construction, and maritime, OSHA's permissible exposure limit for soluble barium compounds (measured as barium) is 0.5 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 0.5 mg/m³ for barium's soluble compounds. NIOSH has no REL for these substances but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing to establish an 8-hour TWA PEL of 0.5 mg/m³ for the soluble barium compounds in agriculture. Promulgation of this limit will make the PEL for these compounds consistent across all OSHA-regulated sectors.

Commonly used soluble barium compounds include barium nitrate, barium oxide, barium carbonate, and barium chloride, all of which are odorless, white solids. These compounds are used as rodenticides, pesticides, disinfectants, and additives; in the manufacture of photographic papers, dyes, chemicals, explosives, matches, and pyrotechnics; in water treatment; as catalysts; and in fireproofing (NIOSH/OSHA Occupational Health Guideline 1981, pp. 1, 4; Hawley's 1987, pp. 118, 120). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Exposure to the soluble barium compounds causes eye, skin, and mucous membrane irritation, central nervous system and gastrointestinal effects, and respiratory disease in humans and animals. Barium compounds can also cause reproductive effects in animals. Barium carbonate has oral LD_{50} values of 418 mg/kg and 200 mg/kg in rats and mice, respectively (RTECS 1990). The oral LD_{50} for barium chloride in rats is 118 mg/kg (RTECS 1990). In contact with the skin of rabbits, 500 mg of barium nitrate caused mild irritation; 100 mg of this substance instilled into the eyes of rabbits caused moderate irritation (RTECS 1990). For barium oxide, the subcutaneous LD_{50} in mice is 50 mg/kg (RTECS 1990). Subcutaneous injection of 5 mg/kg of barium chloride caused acute toxicity and death in rabbits after 2 to 2.5 hours (Fasekas et al. 1953, in Clayton and Clayton 1981, p. 1534). The signs and symptoms of barium salt toxicity in experimental animals include salivation, vomiting, colic, diarrhea, convulsive tremors, slow, hard pulse, and elevated blood pressure. Hemorrhages in the stomach, intestines, and kidneys, followed by muscle paralysis and death, may occur a few hours or days after the exposure (Clayton and Clayton 1981, p. 1534). A recent study in guinea pigs (Hicks, Caldas, Dare, and Hewitt 1986, in Arch. Toxicol. Suppl. 9:416–420) showed that exposure to welding fumes generated in metal arc welding using barium as a flux component caused cardiotoxic and bronchoconstrictive effects. Rats given 1, 10, or 100 ppm barium in their drinking water for 1 to 16 months showed increased systolic blood pressure after exposure for 1 month at the 100-ppm level or 8 months at the 10-ppm level; in the high-dose group, depressed cardiac contraction and electrical hyperexcitability were also seen (Perry, Koop, Perry, and Erlanger 1989, J. Toxicol. Environ. Health 28(3):375–388). Male rats inhaling 1150 mg/m³ of barium carbonate for 24 hours showed evidence of reproductive effects (RTECS 1990). Inhalation of barium carbonate at a concentration of 3130 μg/m³ for 24 hours during the 16th week of pregnancy caused effects on the ovaries and fallopian tubes in female rats (RTECS 1990).

In humans, an oral dose of 800 mg/kg barium carbonate was lethal; convulsions and cardiac effects occurred before death (RTECS 1990). Barium hydroxide and barium oxide are strongly alkaline in aqueous solutions and cause severe eye burns and skin irritation on contact (Grant 1986, p. 134). Symptoms of barium intoxication in humans include nausea, vomiting, colic, and diarrhea; myocardial and general muscular stimulation and tingling of the extremities soon follow (Reeves 1979, in Proctor, Hughes, and Fischman 1988, p. 89). Inhalation of the dust of soluble barium compounds can cause a benign pneumoconiosis called baritosis; radiologic examination shows circumscribed nodules that are evenly distributed throughout both lung fields (Proctor, Hughes, and Fischman 1988, p. 1535). In a factory where bomb casings were heated in barium carbonate, bronchial irritation was reported among

the workers (Elkins 1950, in Clayton and Clayton 1981, p. 1535). In a report of an accidental case of barium carbonate ingestion, symptoms of gastroenteritis, slow pulse rate, and paralysis were seen (Morton 1945, in Clayton and Clayton 1981, p. 1535). Other reports of exposure to barium compounds describe pulmonary nodulation with or without a decrease in lung function (Wende 1956; Gombos 1957, in ACGIH 1986, p. 47).

Based on this evidence, OSHA preliminarily concludes that exposure to the soluble compounds of barium causes eye, skin, and mucous membrane irritation and respiratory, gastrointestinal, and central nervous system effects. OSHA therefore believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed limit of 0.5 mg/m³ as an 8-hour TWA is necessary to significantly reduce the risk of these material health impairments. In addition, promulgation of the proposed PEL will make OSHA's limit for the soluble compounds of barium consistent across all OSHA-regulated sectors.

2-BUTOXYETHANOL

CAS: 111–76–2; Chemical Formula: $C_4H_9OCH_2CH_2OH$

H.S. No. 1046

OSHA's current permissible exposure limit in construction and maritime for 2-butoxyethanol, one of the family of substances known as the glycol ethers, is 50 ppm as an 8-hour TWA, with a skin notation. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 25 ppm, also with a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing a TWA–PEL of 25 ppm, with a skin notation, for this substance in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

2-Butoxyethanol is a colorless liquid with a mild ethereal odor. It is used as a solvent for resins, spray lacquers, varnishes, enamels, and textiles. It is also used in the dry cleaning industry (ACGIH 1986, p. 71; Hawley's 1987, p. 488).

2-Butoxyethanol has long been known to be toxic, with early studies indicating that a single 7-hour exposure to 700 ppm was lethal to laboratory animals (Werner, Mitchell, Miller, and von Oettingen 1943a, as cited in ACGIH 1986/Ex. 1–3, p. 71). Exposures near the lethal level caused systemic toxicity in the form of hemoglobinuria and lung, kidney, and liver changes. Carpenter, Pozzani, Weil, and associates (1956/Ex. 1–303) reported hemolytic anemia and increased fragility of the red blood cells in rats repeatedly exposed to 2-butoxyethanol at 320 ppm for 5 weeks. However, repeated exposure for 12 weeks at 400 ppm was only slightly injurious to dogs (Werner, Mitchell, Miller, and von Oettingen 1943b, as cited in ACGIH 1986/Ex. 1–3, p. 71).

Humans appear to be less susceptible to butoxyethanol poisoning than experimental animals. In humans, several single 8-hour exposures at levels of 200 ppm and 100 ppm caused urinary excretion of butoxyacetic acid; these subjects experienced irritation and discomfort after these exposures (Carpenter, Pozzani, Weil et al. 1956/Ex. 1–303). A recent study has confirmed that the increased erythrocyte osmotic fragility observed in rats exposed to many of the glycol ethers is a very sensitive indicator of toxicity and correlates with the development of hemoglobinuria at higher exposure levels (Moffett, Linnett, and Blair 1976, as cited in ACGIH 1986/Ex. 1–3, p. 71). These findings indicate that the no-effect level in animals is approximately 25 ppm. The ACGIH suggests that 2-butoxyethanol's toxicity may be more likely to occur as a result of skin absorption than as a consequence of inhalation (ACGIH 1986/Ex. 1–3, p. 71).

OSHA preliminary concludes that the current PEL of 50 ppm in construction and maritime is insufficiently protective against the risk of 2-butoxyethanol's irritant, hematological, and other potential systemic effects, which OSHA considers material health impairments. Accordingly, OSHA is proposing to lower the PEL in these two sectors to 25 ppm, to retain the skin notation, and to extend the PEL and the skin notation to agriculture. The proposed limit will reduce the significant risk associated with exposure to 2-butoxyethanol to a level below that at which these toxic effects have been observed in animals and humans. This lower limit will also prevent the discomfort experienced by workers at exposure levels of 40 ppm. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

tert-BUTYL CHROMATE (as CrO_3)

CAS: 1189–85–1; Chemical Formula: $[(CH_3)_3CO]_2CrO_2$

H.S. No. 2024

In general industry, construction, and maritime, OSHA's current permissible exposure limit for tert-butyl chromate (measured as CrO_3) is 0.1 mg/m³ as a ceiling limit; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit for tert-butyl chromate in agriculture. The ACGIH has a TLV®–TWA of 0.1 mg/m³ as a ceiling limit, with a skin notation, for tert-butyl chromate. NIOSH has a REL of 1 μg/m³ (measured as hexavalent chromium) as a 10-hour TWA for this substance. OSHA is proposing to establish a PEL of 0.1 mg/m³ as a ceiling, with a skin notation, for tert-butyl chromate in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

tert-Butyl chromate is a liquid that is used as an organic source of chromium in specialty reactions, in the oxidation of steroids, and in the manufacture of catalysts for the polymerization of olefins (Sittig 1985, pp. 162–163; NIOSH/OSHA 1981). tert-Butyl chromate is a hexavalent chromium compound.

tert-Butyl chromate causes marked eye, skin, and respiratory tract irritation and central nervous system effects in animals. Because tert-butyl chromate is a hexavalent chromium compound, it is likely to cause lung and sinus cancer in humans (Sittig 1985, p. 163). There are no acute toxicity data specific to tert-butyl chromate. Rats exposed repeatedly for 30 to 60 minutes to daily lethal concentrations (dose unspecified) of a mixture of tert-butyl chromate and butyl alcohol showed signs of lacrimation, drowsiness, ataxia, prostration, muscular weakness, twitching, weight loss, rapid and superficial respiration, and necrosis of the skin and subcutaneous tissues (NIOSH/OSHA 1981). At autopsy, extensive epithelial exudation was seen in the liver of these animals (NIOSH/OSHA 1981). In other inhalation studies (species and doses unspecified), tert-butyl chromate caused mild narcosis and rapid respiration (NIOSH/OSHA 1981).

Based on effects seen in animals, workers exposed to tert-butyl chromate are expected to develop pulmonary changes, to experience narcosis, and to develop necrotic lesions of the skin if their skin comes into contact with this substance (Roubal and Krivucova 1960, in ACGIH 1986, p. 80). tert-Butyl chromate can also be absorbed through the skin in toxic amounts, and eye contact with this substance causes severe damage (Hazardous Substance Fact Sheet 1986, p. 1). Contact of the skin with tert-butyl chromate causes deep ulcers and may cause dermal sensitization (Hazardous Substance Fact Sheet 1986, p. 1). Inhalation of tert-butyl chromate vapors may cause ulceration of the nasal septum, with bleeding, discharge, and crusting (Hazardous Substance Fact Sheet 1986, p. 1) Chronic

exposure to low concentrations of tert-butyl chromate also can cause liver and kidney damage (Hazardous Substance Fact Sheet 1986, p. 1).

Based on this evidence, OSHA preliminarily concludes that tert-butyl chromate causes skin, mucous membrane, and respiratory irritation and may cause cancer in exposed workers. OSHA therefore believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed ceiling PEL of 0.1 mg/m^3, with a skin notation, is necessary to significantly reduce the risk of material health impairment among agricultural workers. In addition, promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

n-BUTYL GLYCIDYL ETHER (BGE)
CAS: 2426-08-6; Chemical Formula:
$C_4H_9OCH_2CH_9OH$
H.S. No. 1052

The OSHA limit for n-butyl glycidyl ether in construction and maritime is 50 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH TLV®–TWA is 25 ppm; NIOSH has recommended that occupational exposure to n-butyl glycidyl ether not exceed 5.6 ppm over a 15-minute short-term period. The Agency is proposing a PEL of 25 ppm as an 8-hour TWA for n-butyl glycidyl ether in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

n-Butyl glycidyl ether is a clear, colorless liquid. It is used as a reducing agent and acid receptor, to stabilize chlorinated solvents, and as a chemical intermediate (ACGIH 1986, p. 81; Clayton and Clayton 1981, p. 2201).

OSHA's current PEL of 50 ppm in construction and maritime was adopted from the ACGIH's 1968 TLV® list. The basis for the ACGIH limit was that repeated applications of n-butyl glycidyl ether to the skin of humans caused irritation and sensitization. The NIOSH limit of 5.6 ppm is based, in large part, on mutagenic studies conducted in microbial and mammalian test systems, as well as on some evidence for other members of the glycidyl ether family showing that exposure is associated with testicular atrophy and hematopoietic abnormalities in laboratory animals. After publication of its criteria document, NIOSH received a confidential report prepared for the Shell Development Company by Anderson et al. (1957, as cited in ACGIH 1986/Ex. 1–3, p. 81), who had conducted a rat inhalation study. In this research, rats were exposed to 38 ppm, 75 ppm, 150 ppm, or 300 ppm n-butyl glycidyl ether for 7 hours daily, 5 days per week for 10 weeks. Atrophic testes were found in 5 of 10 rats exposed to 300 ppm, very small testes were found in 1 of 10 rats exposed to 150 ppm, and patchy atrophy was found in the testes of 1 of 10 rats exposed to 75 ppm. No effects were observed in rats exposed at 38 ppm. Based on this additional evidence, NIOSH reaffirmed its REL for n-butyl glycidyl ether in a Current Intelligence Bulletin (NIOSH 1978p). n-Butyl glycidyl ether also causes mild to moderate irritation in contact with the skin of rabbits; instilled into the eyes of animals of the same species, this substance caused severe irritation (RTECS 1990). The oral LD_{50} in rats is 2050 mg/kg, and the dermal LD_{50} in rabbits is 2520 mg/kg (RTECS 1990). The lowest lethal concentration in rats is 670 ppm (RTECS 1990). Animals given intragastric or intraperitoneal injections of n-butyl glycidyl ether developed incoordination and ataxia before death (Hine et al. 1956, in AMA Arch Ind Hlth 14:250–264). Intracutaneous injection of this substance in guinea pigs led to sensitization in 16 of 17 of these animals (Hine et al. 1956).

OSHA believes that this proposal has taken reasonable account of the limited data for this substance; accordingly, OSHA preliminarily concludes that reducing the PEL from 50 ppm to 25 ppm will substantially reduce the significant risk of reproductive effects and will also protect workers in construction, maritime, and agriculture from BGE's irritant and sensitization effects, all of which constitute material health impairments. The Agency therefore proposes a permissible exposure limit of 25 ppm TWA for n-butyl glycidyl ether in construction, maritime, and agriculture. Promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CAPTAN
CAS: 133-06-2; Chemical Formula:
$C_9H_8Cl_3NO_2S$
H.S. No. 1067

OSHA has no PEL for captan in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 5 mg/m^3 for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N6A) with the limit being proposed. The Agency is proposing a PEL of 5 mg/m^3 as an 8-hour TWA for captan in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Captan is a white, crystalline, odorless solid. Captan is used for seed treatment, as a fungicide in paints, in plastics, leather, fabrics, fruit preservation, and as a bacteriostat. When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Skin applications of 900 mg/kg captan produce skin irritation in experimental animals. Long-term feeding studies did not reveal adverse effects in dogs fed captan in the diet at levels of 100 mg/kg/day for 66 weeks or in rats fed 1000 mg/kg/day for 2 years (Martin 1971/Ex. 1–1161; Spencer 1968, as cited in ACGIH 1986/Ex. 1–3, p. 98). Male mice showed decreased fertility at captan levels of 50 or 100 mg/kg/day for 5 days (Collins 1972/Ex. 1–893). Studies on the mutagenicity of captan indicate that the substance acts as an alkylating agent and induces chromosome rearrangements in rats and point mutations in *Neurospora crassa* (Epstein and Legator, as cited in ACGIH 1986/Ex. 1–3, p. 98). Legator and colleagues (1969, as cited in ACGIH 1986/Ex. 1–3, p. 98) reported that captan concentrations of 10 ug/ml inhibited DNA in human embryo cells, and concentrations of 1.5 ug/ml produced chromosomal aberrations in the somatic and germ cells of kangaroo rats. Animal evidence concerning the carcinogenicity of captan is contradictory, although high doses caused significant incidences of polyploid carcinoma of the duodenum and adenomatous polyps in mice (NCI 1977a, as cited in ACGIH 1986/Ex. 1–3, p. 98).

Some captan-exposed individuals experience skin irritation (Spencer 1968, as cited in ACGIH 1986/Ex. 1–3, p. 98). A case of recurrent urticaria caused by captan exposure has been reported and confirmed (Croy 1973/Ex. 1–894), and captan caused high reactivity when administered in a battery of patch tests (Rudner 1977/Ex. 1–967).

OSHA is proposing a PEL of 5 mg/m^3 as a TWA to protect workers exposed to captan in the construction, maritime, and agriculture industries from the significant risk of exposure-related skin irritation, reproductive effects, mutagenicity, and, perhaps, carcinogenicity, all of which constitute material health impairments. The Agency preliminarily concludes that this limit will substantially reduce these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CARBARYL (SEVIN®)
CAS: 63-25-2; Chemical Formula
$C_{12}H_{11}NO_2$

H.S. No. 2026

OSHA's current limit for carbaryl in general industry, construction, and maritime workplaces is 5 mg/m³ as an 8-hour TWA. OSHA has no PEL for carbaryl in agriculture. The 1987–1988 ACGIH TLV®–TWA for carbaryl is 5 mg/m³; the NIOSH recommended exposure limit (REL) for this substance is 5 mg/m³ as a 10-hour time-weighted average. OSHA is proposing to establish a PEL of 5 mg/m³ as an 8-hour TWA for carbaryl in agriculture. This is the limit recently established for carbaryl in general industry.

Carbaryl is an odorless solid that is crystalline and white or grayish in color (Proctor, Hughes, and Fischman 1988, p. 115; ACGIH 1986, p. 99). Carbaryl is widely used in agriculture as an insecticide for corn, vegetables, soybeans, cotton, deciduous fruits, nuts, alfalfa, wheat, and many other crops. It is also used as an insecticide for gardens, turf, ornamentals, forests, livestock, and poultry. In addition, carbaryl is an effective acaricide and molluscicide (HSDB 1985). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Carbaryl is a carbamate pesticide; like other carbamates, this substance causes reversible cholinesterase inhibition in humans and animals. In animals, carbaryl also causes irritation of the eyes and skin when in contact with these organs (RTECS 1920). There is some evidence in experimental animals that carbaryl causes reproductive effects in female animals and developmental effects in the offspring of exposed animals (RTECS 1990; EPA 1987). The oral LD_{50} in rats is 250 mg/kg, and the dermal LD_{50} in rabbits is 2000 mg/kg (RTECS 1990). Dogs exposed by inhalation to a carbaryl dust concentration of approximately 75 mg/m³ showed evidence of cholinesterase inhibition, but rats exposed to the dust at a concentration of 10 mg/m³ for 7 hours/day for 90 days showed no such effects (ACGIH 1986, p. 99).

Dogs fed single oral doses of carbaryl ranging from 250 to 500 mg/kg showed signs of overstimulation of the parasympathetic nervous system (increased respiratory rate, salivation, muscular twitching, incoordination), at the two highest doses; however, red blood cell cholinesterase activity declined by 24 to 33 percent in the 375-mg/kg dose group (EPA 1987). Decreases in red blood cell cholinesterase activity have also been seen after the administration of single oral doses of carbaryl to rats (Carpenter et al. 1961; Weil et al. 1968).

Reproductive effects (reduced fertility, increase in number of resorptions, decrease in number of live fetuses/litter) have been seen in female rats, rabbits, mice, and gerbils (Weil et al. 1972; Murray et al. 1979; Collins et al. 1970), and some studies have shown developmental effects (omphalocele, conjoined nostrils, delayed ossification) among the offspring of pregnant rabbits and mice given carbaryl during gestation (Murray et al. 1979).

In humans, exposure to carbaryl causes a depression in cholinesterase activity that is reversible on cessation of exposure. After a large-scale carbaryl spraying operation in Nigeria, eight applicators had 15-percent reductions in their plasma cholinesterase activity levels (Vanderkar 1965). Male volunteers who ingested doses of carbaryl of up to 0.13 mg/kg/day for 6 weeks showed no signs and reported no symptoms of cholinesterase poisoning (Gosselin, Smith, and Hodge 1984, p. III–87). A human suffered from moderate poisoning after ingesting 250 mg of carbaryl; after a short period (20 minutes) with no symptoms, abdominal pain, profuse sweating, lassitude, and vomiting developed in this individual (Proctor, Hughes, and Fischman 1988, p. 115). Workers exposed for 19 months to carbaryl at concentrations ranging from 0.23 to 0.31 mg/m³ showed no signs or symptoms of cholinesterase inhibition (Proctor, Hughes, and Fischman 1988, p. 115).

The evidence described above shows that carbaryl exposure causes irritation and inhibition of cholinesterase activity in both humans and animals, and that it has reproductive and teratogenic effects in animals. Accordingly, OSHA preliminarily concludes that, in the absence of a limit, workers in agriculture are potentially at significant risk of experiencing these exposure-related effects. The Agency believes that the proposed 8-hour TWA limit of 5 mg/m³ for carbaryl will substantially reduce these risks for workers in this sector. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

beta-CHLOROPRENE

CAS: 126–99–8; Chemical Formula: $CH_2{:}CClCH{:}CH_2$

H.S. No. 1088

The current OSHA limit for beta-chloroprene in construction and maritime is an 8-hour TWA of 25 ppm, with a skin notation. There is no limit in agriculture. The ACGIH has a 10-ppm TLV®–TWA, with a skin notation, and NIOSH (1977c/Ex. 1–277) recommended a ceiling limit of 1 ppm, measured over a 15-minute period. In construction, maritime, and agriculture, the Agency is proposing an 8-hour TWA PEL of 10 ppm; the skin notation is retained in construction and maritime and proposed in agriculture. NIOSH (Ex. 8–47, Table N1) concurs with this limit, which was recently established for this substance in general industry.

beta-Chloroprene is a colorless, highly flammable liquid. It is used in the production of neoprene (ACGIH 1986, p. 135; Hawley's 1987, p. 271).

The ACGIH recommended a reduction in the TLV® for beta-chloroprene from 25 ppm to 10 ppm in 1981, based on a review of the world literature by Trochimowicz, who prepared the 1980 ACGIH documentation, and by Reinhardt (1980, as cited in ACGIH 1986/Ex. 1–3, p. 135). Reinhardt concluded that there was no evidence indicating that the former 25-ppm PEL was not protective, but OSHA believes the systemic effects (in the form of growth retardation) seen in rats and hamsters exposed to 39 ppm beta-chloroprene for 4 weeks or to 50 ppm for a lifetime suggest that the 25-ppm PEL is not sufficiently protective.

When NIOSH established a REL of 1 ppm for beta-chloroprene, NIOSH (1977c/Ex. 1–277) cited three reports on beta-chloroprene-using facilities in the Soviet Union. Katsova (1973, as cited in ACGIH 1986/Ex. 1–3, p. 135) reported finding a significant excess of chromosomal abnormalities in the blood of workers exposed to approximately 5 ppm beta-chloroprene. Volkova, Fomenko, Bagdinov et al. (1976/Ex. 1–1025) reported similar findings in a plant where beta-chloroprene levels ranged from 0.8 to 1.95 ppm. In the third study, Sanotskii (1976/Ex. 1–662) reported abnormal sperm morphology among workers exposed at levels of from 0.28 to 1.94 ppm; a threefold increase in the rate of spontaneous abortion among wives of these workers was also found. In addition, NIOSH (1977c/Ex. 1–277) cited a study by Davtiani, Fomenko, and Andreyeva (1973/Ex. 1–1032) that reported a significant excess of embryonic mortality in female rats that were mated to male rats exposed to 1 ppm beta-chloroprene. These investigators also found chromosomal aberrations in the bone marrow cells of exposed male rats. NIOSH (1977c/Ex. 1–277) also cited a number of reports showing beta-chloroprene to be mutagenic in a variety of test systems. NIOSH concluded that it was prudent to reduce exposure to 1 ppm over a 15-minute period, to reduce the risk of genetic abnormalities being transmitted

to subsequent generations. This exposure represents the lowest concentration that can be measured reliably over a 15-minute period.

However, OSHA notes that sizeable discrepancies exist between the findings from the Russian studies and results from other studies that were undertaken to confirm the Soviet claims. Torkelson and Rowe (1981c, in "Patty's Industrial Hygiene and Toxicology," 3rd rev. ed., Vol. 2B, Clayton and Clayton 1981) offer two possible explanations for these discrepancies:

> beta-Chloroprene is a very unstable compound, which, unless handled with extreme care, * * * [epoxidizes] and polymerizes to toxic compounds. This might explain the alleged effects in animals. Alleged effects in humans may be due to this same cause or to the use of different chemical processes which produce different types of impurities. Many other causes can be postulated, but in our opinion more credence must be given to animal studies in which the sample is known to have been handled with extreme care and to the results of experience in U.S. industry where the method of handling has been reported (Torkelson and Rowe 1981c, p. 3578).

These authors report that when the purity of the sample was carefully controlled, repeated exposures to 25 ppm or less of the vapor have caused no reproductive, teratological, or embryotoxic effects in rats: "Despite frank clinical toxicity in exposed pregnant rats, fetuses showed no teratogenic effects at beta-chloroprene levels as high as 175 ppm" (Torkelson and Rowe 1981c, pp. 3579–80).

The 1-ppm (15-minute STEL) value recommended for this substance by NIOSH is based on studies reported in the Soviet literature; in addition, the REL is set at the analytical limit of detection. OSHA's 10-ppm PEL is based on a 1981 critical review of the world literature (Trochimowicz 1980, as cited in ACGIH 1986/Ex. 1–3, p. 135) and on the observation that only mild systemic effects are observed after exposure to this substance at 38 ppm. OSHA is proposing an 8-hour TWA PEL of 10 ppm, with a skin notation, for beta-chloroprene in the construction, maritime, and agricultural sectors, to substantially reduce the significant risk of reproductive and systemic effects, which constitute material health impairments that are potentially associated with exposure to beta-chloroprene. The Agency preliminarily concludes that the proposed limit will substantially reduce this significant risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CYANIDES (as CN)
CAS: Varies with compound; Chemical Formula: Varies with compound
H.S. No. 2045

In general industry, construction, and maritime, OSHA's permissible exposure limit for cyanides is 5 mg/m^3 as an 8-hour TWA; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for cyanides. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 5 mg/m^3, with a skin notation. NIOSH has a REL of 5 mg/m^3 as a 10-minute ceiling for the cyanide salts. OSHA proposes to establish a TWA PEL of 5 mg/m^3, with a skin notation, for the cyanides in agriculture. Promulgation of this limit will make the PEL for the cyanides consistent across all OSHA-regulated sectors.

The principal cyanides in industrial use are potassium and sodium cyanide and calcium cyanide (Proctor, Hughes, and Fischman 1988, p. 166). Potassium cyanide and sodium cyanide are white deliquescent solids that have a faint, bitter, almond-like odor. Cyanides find use as insecticides and fumigants, in the extraction of gold and silver ores, in electroplating and metal cleaning, in the manufacture of dyes, pigments, nylon, and chelating agents, in the heat treatment of metals, in photography, and as reagents in analytical chemistry (ACGIH 1986, p. 153; Hawley's 1987, pp. 954, 1057). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Cyanides cause systemic effects and eye, skin, and respiratory tract irritation in humans and animals. Sodium cyanide has an oral LD_{50} of 6440 μg/kg in rats (RTECS 1990). The oral LD_{50}s for potassium cyanide in rats, mice, and rabbits are 10 mg/kg, 8.5 mg/kg, and 5 mg/kg, respectively (RTECS 1990). The ocular LD_{50} in rabbits for sodium cyanide is 5 mg/kg; in rabbits, the ocular LD_{50} for potassium cyanide is 7.8 mg/kg (RTECS 1990). Various species (monkeys, cats, dogs, and rats) given daily subcutaneous injections of potassium cyanide at lethal levels showed signs of nystagmus, periods of blindness, and degeneration of the optic nerve, chiasm, and optic tract (Grant 1986, p. 287). A recent study in mice showed that subcutaneous injection of 10 mg/kg potassium cyanide increased the levels of blood ammonia to 2.5 times those of controls; all animals subsequently lapsed into unconsciousness (Yamamoto 1989, in Toxicol. Appl. Pharmacol. 99(3):415–420). The author suggests that hyperammonemia and increased neutral and aromatic amino acids may be important in the loss of consciousness associated with cyanide exposure. An oral dose of 65 g/kg potassium cyanide caused effects on the reproduction and fertility of female rats when administered 14 days before and on days 1 through 22 of pregnancy (RTECS 1990 "Potassium Cyanide").

In humans, the cyanide ion has essentially the same toxicity whatever the route of absorption (Hayes 1982, p. 127). The estimated LC_{50} in humans is 3404 ppm for a 1-minute exposure (NIOSH Criteria Document 1976). After 6 to 8 minutes, exposure to 270 ppm is fatal; 181 ppm is lethal after 10 minutes, and 135 ppm is fatal after a 30-minute exposure (NIOSH Criteria Document 1976). The signs and symptoms of less severe exposure to any of the cyanides include weakness, headache, confusion, nausea, and vomiting (NIOSH Criteria Document 1976). In humid atmospheres, cyanide solutions cause skin and respiratory tract irritation and may cause an allergic contact dermatitis (NIOSH Criteria Document 1976). Cyanide salt solutions cause itching and discoloration or corrosion of the skin due to the alkalinity of these solutions; exposure to solutions as dilute as 0.5 percent potassium cyanide has caused skin irritation and symptoms such as headaches and dizziness (NIOSH Criteria Document 1976). In one case, an individual who ingested 600 mg of potassium cyanide experienced acute pulmonary edema and lactic acidosis (Graham, Laman, Theodore, and Robin 1977, in Proctor, Hughes, and Fischman 1988, p.167). The signs and symptoms of chronic exposure to cyanides include weakness, nausea, headache, and vertigo (NIOSH Criteria Document 1976). Thirty-six silver-reclaiming facility workers repeatedly exposed to cyanide (concentrations not specified) continued to experience symptoms as long as 7 months or more after exposure ended; at least 30 percent of these workers reported experiencing headache, eye irritation, easy fatigue, loss of appetite, and nose bleeds (Blanc et al. 1985, in Proctor, Hughes, and Fischman 1988, p.167). A 39-year old worker suffered from loss of appetite, nervousness, dizziness, headache, nausea, vomiting, and weight loss caused by repeated exposure to cyanide when he put very hot iron into a hardening bath (Wuthrich 1954, in Hayes 1982, p.127). The illness disappeared during the patient's 2- or 3-week absence from work; however, his symptoms returned within 1 month after

return to work (Wuthrich 1954, in Hayes 1982, p. 127).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to any of the cyanides causes eye, skin, and respiratory tract irritation and systemic effects in humans and animals. OSHA therefore believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed limit of 5 mg/m³ as an 8-hour TWA, and a skin notation, is necessary to significantly reduce these risks of material health impairment among these workers. Promulgation of this limit will also make the PEL for these substances consistent across all OSHA-regulated sectors.

CYCLOHEXYLAMINE

CAS: 108–91–8; Chemical Formula: $C_6H_{13}N$

H.S. No. 1109

OSHA has no limit for cyclohexylamine in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 10 ppm. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the proposed limit. OSHA is proposing a PEL of 10 ppm as an 8-hour TWA for cyclohexylamine in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Cyclohexylamine is a liquid with a strong, fishy, amine odor. It is used in the manufacture of insecticides, plasticizers, and dry cleaning soaps, as a corrosion inhibitor, and as a chemical intermediate (ACGIH 1986, p. 161; Merck 1983, p. 392). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Data concerning the acute toxicity of cyclohexylamine were reported by Eastman Kodak in 1958 (ACGIH 1986/Ex. 1–3, p. 161). In rats, the oral LD_{50} of a 5-percent solution in water was between 400 and 800 mg/kg; mice fed a diet of the 1-percent aqueous solution or the undiluted amine had LD_{50}s of between 200 and 400 mg/kg. Injection of the 5-percent aqueous solution in rats produced LD_{50}s of between 5 and 25 mg/kg, while mice injected intraperitoneally with the 1-percent solution had LD_{50}s of between 5 and 10 mg/kg. In guinea pigs, the dermal LD_{50} of undiluted cyclohexylamine is reported to be between 1 and 5 ml/kg. Edema, necrosis, and eschars were reported as a consequence of these dermal exposures. In rabbits, one drop of a 50-percent solution caused complete destruction of the eye. Six-hour inhalation exposures at a vapor concentration of 12,000 ppm caused deaths in rats, but exposure to 1000 ppm caused neither toxic effects nor deaths.

Legator, Palmer, Green, and Petersen (1969/Ex. 1–498) considered cyclohexylamine to be a potential carcinogen, mutagen, or teratogen on the basis of dose-dependent chromosomal abnormalities observed in rats injected intraperitoneally with cyclohexylamine. Khera, Stolz, Gunner et al. (1971/Ex. 1–343) noted adverse effects on rat fertility, and Becker and Gibson (1970/Ex. 1–298) reported embryotoxic effects in mice intraperitoneally injected with cyclohexylamine. In contrast, Kennedy, Sanders, Weinberg et al. (1969, as cited in ACGIH 1986/Ex. 1–3, p. 161) reported no effects of exposure to cyclohexylamine on rabbit and rat fertility, reproduction, embryogenesis, or perinatal and postnatal development.

In general, there is agreement concerning the moderate to severe toxicity of cyclohexylamine and its potential for intense skin irritation and moderate skin sensitization (Sax 1968b, as cited in ACGIH 1986/Ex. 1–3, p. 161). The chemical is well known to be pharmacologically active, having sympathomimetic activity (Barger and Dale 1910/Ex. 1–1104). However, Litchfield and Swan (1971/Ex. 1–346) report that human dietary levels of 5 g/day for 7 to 8 days produced no pharmacologically active levels in the tissues; furthermore, no changes were detected in blood pressure, heart rate, or electrocardiograms of exposed subjects. Chronic experimental toxicity data are lacking, but Watrous and Schulz (1950/Ex. 1–940) have reported that exposure to 4 to 10 ppm of cyclohexylamine caused no symptoms of any kind in acutely exposed employees.

OSHA is proposing an 8-hour TWA PEL of 10 ppm for cyclohexylamine in construction, maritime, and agriculture. The Agency preliminarily concludes that limiting workplace exposures to this substance to the 10-ppm level will protect workers in these sectors from the significant risks of severe skin and eye irritation and sensitization, all material health impairments that are associated with exposure to cyclohexylamine. OSHA believes that this limit is necessary to substantially reduce these significant occupational risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CYHEXATIN

CAS: 13121–70–5; Chemical Formula: $(C_6H_{11})_3SnOH$

H.S. No. 1112

OSHA has no limit for cyhexatin in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 5 mg/m³ for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the proposed limit. The Agency is proposing a 5 mg/m³ TWA–PEL for cyhexatin in construction, maritime, and agriculture. This is the limit recently established for cyhexatin in general industry.

At room temperature, cyhexatin exists in the form of white crystals. Cyhexatin is used as a biocide and it is also an effective miticide (ACGIH 1986, p. 165). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Cyhexatin has oral LD_{50}s of 500, 700, and 190 mg/kg for rabbits, guinea pigs, and rats, respectively (NIOSH 1977i/Ex. 1–1182). Skin exposure to a 1- to 2-percent solution of cyhexatin in goats and cattle caused mild effects; sheep showed mild effects after application of a 0.5-percent solution. One of five sheep died from multiple skin applications of a 1-percent suspension (Johnson, Younger, Witzel, and Radeleff 1975/Ex. 1–336).

The toxicity of cyhexatin is considered to be moderate, although it is greater than the toxicity of most other organic tin compounds. Long-term feeding in rats produced no behavioral changes, mortality, tissue changes, or hematologic or biochemical alterations in response to 2 years of dosing at 12 mg/kg per day; however, dosed animals were smaller than controls. After daily doses by gavage of 24 mg/kg per day for 2 weeks, rats showed microscopic changes in the liver, kidneys, and adrenal glands at autopsy. Six mg/kg is considered to be the no-effect level in rats, and in dogs, the no-effect feeding level is reported to be 3 mg/kg. Rats fed 4 to 6 mg/kg, and rabbits fed 3 mg/kg, showed no ill effects on indices for fertility, gestation, viability, or lactation (Dow Chemical Company 1973d, as cited in ACGIH 1986/Ex. 1–3, p. 165). No inhalation data on animals are available, and there are no human data.

OSHA is proposing an 8-hour TWA limit of 5 mg/m³ for cyhexatin in construction, maritime, and agriculture. OSHA preliminarily concludes that a PEL of 5 mg/m³ will protect workers in these sectors against the significant risk of skin and respiratory irritation, as well as other possible adverse effects associated with exposure in the absence of a limit. The Agency considers these adverse health effects to be material health impairments within the meaning of the Act. Promulgation of this limit will

make OSHA's PEL for this substance consistent across all regulated sectors.

2,4-D (2,4-DICHLOROPHENOXYACETIC ACID)
CAS: 94–75–7; Chemical Formula: $C_8H_6Cl_2O_3$
H.S. No. 2050

In general industry, construction, and maritime, OSHA's permissible exposure limit for 2,4-D is 10 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 10 mg/m³ for 2,4-D. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing to establish an 8-hour TWA PEL of 10 mg/m³ for 2,4-D in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

2,4-D, also called 2,4,dichlorophenoxyacetic acid, is a white to yellow powder with a light phenol-like odor. It is a selective herbicide used to control broadleaf weeds (Hayes 1982, p. 521; ACGIH 1986, p. 167). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

2,4-D causes skin irritation, central nervous system effects, and liver and kidney damage in humans and animals; it also causes reproductive and teratogenic effects in animals. The oral LD_{50}s for rats, mice, guinea pigs, and hamsters are 370 mg/kg, 368 mg/kg, 469 mg/kg, and 500 mg/kg, respectively (RTECS 1990). The dermal LD_{50} in rats is 1500 mg/kg; in rabbits it is 1400 mg/kg (RTECS 1990). Monkeys showed no serious ill effects from a 2,4-D dose of 214 mg/kg (Hill and Carlisle 1947, in Hayes 1982, p. 521). Acute toxicity causes virtually the same effects in all species; death usually occurs as a result of ventricular fibrillation (ACGIH 1986, p. 167). Acutely poisoned animals show stiffness, incoordination, lethargy, stupor, and coma before death (ACGIH 1986, p. 167). Dogs administered a 100-mg/kg dose of 2,4-D developed irritation of the gastrointestinal mucosa, hepatic necrosis, and mild renal tubular degeneration (Drill and Hirtzaka 1953, in EPA Health Advisory 1987, p. 4). Repeated doses of 2,4-D can lead to loss of appetite, weight loss, vomiting, depression, roughness of coat, tenseness, and muscular weakness (Hayes 1982, p. 521). Female rats given 300 mg/kg of 2,4-D orally five times a week for 4 weeks showed depressed growth rates, liver pathology, and gastrointestinal irritation (National Research Council 1977, in HSDB 1985). A single intraperitoneal dose of 240 mg/kg or daily oral doses of 40 mg/kg for 30 days caused an alteration in brain mitochondrial oxidative phosphorylation manifested by an increase in body temperature, increased mitochondrial ATPase activity, and increased oxygen consumption in male rats (Podolak 1981, in Dangerous Properties of Industrial Materials Report 1987, Vol. 7, p. 17). Rats fed doses of 5, 15, or 45 mg/kg/day for 90 days had significant reductions in blood indices at all doses and reduced liver enzyme activities at higher doses; there was also evidence of kidney toxicity at the higher dose (Hazelton Laboratories 1983, in EPA Health Advisory 1987, p. 4). Male and female rats ingesting 2,4-D had increased incidences of malignant neoplasms and lymphosarcomas; female rats developed neoplasms of the mammary gland, while males had carcinomas of the endocrine organs (Reuber 1983, in HSDB 1985). At a dose of 110 mg/kg/day, 2,4-D is teratogenic and embryotoxic in mice (Baage et al. 1973, in Hayes 1982, p. 523). A sow receiving 500 mg/kg of 2,4-D in her diet throughout pregnancy had underdeveloped and apathetic piglets; 10 of 15 died within 24 hours of birth (Bjorklund and Erne 1966, in IARC 1977, Vol. 15, p. 123). Continued 2,4-D feeding of surviving piglets for 8 months caused marked growth depression, persistent anemia, and degenerative changes of the liver and kidneys (Bjorklund and Erne 1966, in IARC 1977, Vol. 15, p. 123).

In humans, the lowest lethal oral 2,4-D dose is 80 mg/kg; before death, gastrointestinal effects, behavioral symptoms, and coma occurred (RTECS 1990). An oral 2,4-D dose of 93 mg/kg caused convulsions (RTECS 1990). Contact of 2,4-D with the skin can cause dermatitis (Seabury 1963; Hayes 1963, in Proctor, Hughes, and Fischman 1988, p. 192). Five female forestry workers who applied a 2,4,5-T and 2,4-D mixture with brushes experienced severe toxic contact eczema on the exposed areas of their skin (Jung and Wolf 1977, in Hayes 1982, p. 524). An intravenous dose of 3.6 g of 2,4-D for medicinal treatment caused stupor, hyporeflexia, fibrillary twitching of some muscles, and urinary incontinence; 24 hours later, the patient still showed muscular weakness, but this ended after another 24 hours (Seabury 1963; Hayes 1963, in Proctor, Hughes, and Fischman 1988, p. 192). The American Medical Association received reports of several cases of illness involving workers engaged in the field application of 2,4-D; symptoms included a burning sensation in the throat and chest, weakness, loss of appetite and weight, and slight albuminuria; these symptoms were a result of inhaling 2,4-D (Queries and Minor Notes 1956; in ACGIH 1986, p. 167). A male student ingested 6 g of a commercial herbicide preparation of the dimethylamine salt of 2,4-D and experienced vomiting and convulsions prior to death; autopsy showed degenerative ganglion cell changes in the brain (Neilson et al. 1965, in EPA Health Advisory 1987, p. 4).

Three cases of peripheral neuropathy resulting from the spraying of 2,4-D have been reported. In one case, paresthesia of the extremities, pain in the legs, and muscle twitching appeared within 4 to 5 days; however, no neurological or electromyographical changes were seen in this case (Goldstein et al. 1959, in IARC 1977, Vol. 15, p. 127). In the second case, numbness and aching occurred in the fingers and toes 1 week after a second exposure; 6 weeks later, the patient had a well-developed neuropathy (Goldstein et al. 1959, in IARC 1977, Vol. 15, p. 127). In the third case reported, a second exposure to 2,4-D caused severe leg pain and swelling of the metacarpal joints of both hands; 5 months later, flaccid paraparesis was seen (Goldstein et al. 1959, in IARC 1977, Vol. 15, p. 127). In similar cases of workers exposed to 2,4-D during spraying operations, signs and symptoms of muscular weakness, vomiting, diarrhea, fever, hyperthermia, tachycardia, and neurological effects (which continued to occur for 40 days to 2 years after exposure) were reported (Monarca and DiVito 1961; Paggiaro et al. 1974; Todd 1962, in IARC 1977, Vol. 15, p. 128). Of 292 workers involved in the manufacture of 2,4-D compounds for periods ranging from less than 5 years to 6 to 10 years, 63 percent reported experiencing symptoms of weakness, rapid fatigue, headache, and vertigo, while about 20 percent had cardiovascular effects such as hypotension and bradycardia and digestive disturbances such as dyspeptic symptoms and gastritis (Bashirov 1969, in IARC 1977, Vol. 15, p. 128). Workers with longer exposures to 2,4-D compounds showed more pronounced liver dysfunction than workers exposed for shorter periods (Bashirov 1969, in IARC 1977, Vol. 15, p. 128).

Based on this evidence in humans and animals, OSHA preliminarily concludes that 2,4-D causes skin irritation and central nervous system, liver, and kidney effects in humans and animals. OSHA therefore believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. OSHA believes that the proposed 8-hour TWA PEL of 10 mg/m³ for 2,4-D in agriculture is necessary to

significantly reduce the risks of these material health impairments. In addition, promulgation of the proposed PEL will make OSHA's limit for 2,4-D consistent across all OSHA-regulated sectors.

DICHLORODIPHENYL-TRICHLOROETHANE (DDT)
CAS: 50–29–3; Chemical Formula: $C_{14}H_9Cl_5$
H.S. No. 1113

OSHA's existing limit for dichlorodiphenyltrichloroethane (DDT) in general industry, construction, and maritime is 1 mg/m³ as an 8-hour TWA, with a skin notation. There is no limit in agriculture. The ACGIH has the same 8-hour TWA limit for DDT, without a skin notation. NIOSH considers DDT a potential occupational carcinogen and recommends reducing exposure to the lowest feasible concentration, which is the lowest reliably detectable concentration (currently 0.5 mg/m³ TWA). The Agency is proposing an 8-hour TWA limit of 1 mg/m³, and a skin notation, for DDT in agricultural operations.

Although DDT is banned in the United States, this substance is still widely used in other countries (Hayes 1982, p. 182). OSHA is proposing to extend the same limit currently in effect in other sectors to agriculture to achieve the primary goal of this rulemaking, which is consistency in permissible exposure limits across sectors. Workers in this sector would thus be protected in the unlikely event that DDT use is permitted at some time in the future.

DDT is a noncombustible, colorless to white powder with a slightly aromatic odor. Before being banned, DDT was a widely used insecticide.

DDT affects the nervous system, causing convulsions, tremors, and numbness. This substance also has reproductive and teratogenic effects. The oral LD_{50} in rats is 87 mg/kg, and the dermal LD_{50} in rabbits is 300 mg/kg (RTECS 1990), indicating a significant degree of percutaneous absorption. Acutely poisoned animals show incoordination, excitement, muscular weakness, and convulsions before death (Clayton and Clayton 1981, p. 3688). Rats fed large oral doses of DDT show focal and centrilobular necrosis of the liver at autopsy (Hayes 1982, pp. 180–205). The liver damage associated with chronic feeding of DDT is dose-related; rats fed 100 ppm show mild liver damage at autopsy, while those fed at higher levels showed increasingly severe damage (Clayton and Clayton 1981, p. 3689). Some authors (Treon and Cleveland, 1955) have reported liver effects in rats fed 12.5 ppm DDT in the diet for 18 to 24 months. DDT has been tested for reproductive and/or embryotoxic, teratogenic, or developmental effects in rats, mice, dogs, and rabbits using oral, intraperitoneal, or subcutaneous route of administrations; the results of these studies show that DDT has both male and female reproductive effects as well as embryotoxic and developmental effects (RTECS 1990). DDT has been tested for carcinogenicity in rats, mice, hamsters, dogs, and monkeys. In mice and rats, the results of these studies were positive. The principal tumors produced by DDT were benign and malignant liver neoplasms, lymphomas, and lung neoplasms. Based on this evidence, the International Agency for Research on Cancer has concluded that the evidence for the carcinogenicity of DDT in animals is sufficient (IARC 1987, Suppl. 7, p. 187).

In humans, incidents of acute poisoning have generally occurred as a result of accidental ingestion (Hayes 1982, pp. 180–205). If the dose is sufficiently large (i.e., greater than 16 mg/kg), convulsions may occur, followed by respiratory failure and death (Hayes 1982, pp. 180–205). Involvement of the liver, as evidenced by jaundice, has been seen in some cases of accidental ingestion (Hayes 1982, pp. 180–205). After a single dose or small repeated doses, DDT accumulates in adipose tissue, where it remains for long periods of time (Gosselin, Smith, and Hodge 1984, p. III–135). The significance to health of the fat storage of DDT is not known.

Concern that DDT may be carcinogenic in humans has been widespread, but the results of many studies in farmers, forestry workers, pesticide applicators, and other occupationally exposed workers have been inclusive, and interpretation even of the few positive studies are complicated by the concomitant exposure of these workers to other pesticides (IARC 1987, Suppl. 7, p. 186). Based on an evaluation of the available data in humans, IARC has concluded that the evidence for the carcinogenicity of DDT in humans is inadequate (IARC 1987, Suppl. 7, p. 186). A recent prospective follow-up study of cancer mortality in relation to serum DDT levels (Austin, Keil, and Cole 1989) concluded that there is "weak" evidence of a positive relation between respiratory cancer mortality and serum DDT levels.

Based on a review of the evidence of the health effects of exposure to DDT, OSHA preliminarily concludes that the existing PEL of 1.0 mg/m³ is adequately protective in the unlikely event of exposure to this banned substance. The Agency finds that the existing limit, with its skin notation, provides appropriate protection against DDT's systemic effects and proposes to extend this limit to agriculture to make OSHA's PEL for this substance consistent across all regulated sectors.

2-N-DIBUTYLAMINOETHANOL
CAS: 102–81–8; Chemical Formula: $(C_4H_9)_2NCH_2CH_2OH$
H.S. No. 1120

OSHA's current limit for 2-N-dibutylaminoethanol (DBAE) in construction and maritime is 2 ppm as an 8-hour TWA, with a skin notation. There is no limit in agriculture. The ACGIH has a TLV®-TWA of 2 ppm for DBAE, with a skin notation. The Agency is proposing to retain its PEL of 2 ppm as an 8-hour TWA in construction and maritime, to delete the skin notation, and to extend the 2 ppm 8-hour TWA PEL to agriculture. NIOSH concurred (Ex. 8–47, Table N–1) with this limit when it was recently established in general industry.

2-N-Dibutylaminoethanol is a colorless, combustible liquid with a faint amine-like odor. It is used in organic synthesis (ACGIH 1986, p. 390; Hawley's 1987, p. 175).

In rats, 2-N-dibutylaminoethanol has a single-dose oral LD_{50} of 1.7 g/kg and an intraperitoneal LD_{50} of 0.14 g/kg; these values are approximately analogous to the oral and intraperitoneal LD_{50}s for diethanolamine (Hartung and Cornish 1968/Ex. 1–328). The dermal LD_{50} for DBAE in rabbits is 1.68 g/kg (Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1–440). In male rats, the lowest 5-week drinking water dose tolerated without weight loss was 0.13 g/kg/day. Rats that ingested a dose of 0.43 g/kg/day showed elevated kidney-to-body-weight ratios but no histologic changes at autopsy (Cornish, Dambrauskas, and Beatty 1969/Ex. 1–411). In inhalation studies of rats, 6-hour exposures at 70 ppm for 5 days killed one rat; the surviving rats showed a 57-percent average body weight loss, as well as a doubling of kidney-to-body-weight ratios, a tenfold increase in serum bilirubin, a slight increase in clotting time, and an elevated hematocrit. Inhalation of 33 ppm for 1 week caused a 3-percent body weight loss and a slight increase in clotting time, but no significant changes in the other variables observed. Twenty-seven weeks of exposure to 22 ppm resulted in no differences between exposed rats and controls in the variables measured (Cornish, Dambrauskas, and Beatty 1969/Ex. 1–411). 2-N-Dibutylaminoethanol is a more potent inhibitor of acetylcholinesterase

in vitro than is diethylamine (DEA) (Hartung and Cornish 1968/Ex. 1–328).

OSHA is retaining its 8-hour TWA PEL of 2 ppm for 2-N-dibutylaminoethanol in construction and maritime, and deleting the skin notation; in agriculture, OSHA is proposing an 8-hour TWA PEL of 2 ppm for DBAE. The Agency preliminarily concludes that this limit will protect workers in agriculture from the significant risk of metabolic effects associated with inhalation exposure at the levels permitted in the absence of an OSHA limit. OSHA believes that this substance does not present a significant risk of systemic toxicity via percutaneous absorption, and the skin notation is therefore not being retained in construction and maritime. Promulgation of the 8-hour TWA limit in agriculture will make OSHA's PEL for this substance consistent across all regulated sectors.

DIELDRIN

CAS: 60–57–1; Chemical Formula: $C_{12}H_8Cl_6O$

H.S. No. 2061

In general industry, construction, and maritime, OSHA's current TWA–PEL for dieldrin is 0.25 mg/m³, with a skin notation. The NIOSH REL for this substance is the lowest detectable limit; the REL is based on dieldrin's carcinogenicity in animals. The ACGIH TLV®–TWA is 0.25 mg/m³, with a skin notation. OSHA is proposing an 8-hour TWA PEL of 0.25 mg/m³, and a skin notation, for dieldrin in agricultural workplaces. This action will make the PEL consistent across all OSHA-regulated sectors.

Dieldrin, also called HEOD, Octalox®, Panoram®, and Quintox®, is a light brown, nonflammable solid. It is a cyclodiene insecticide that was formerly used extensively in the soil treatment of agricultural fields; it also found widespread use in malaria control (Hayes 1982, pp. 237–238). Dieldrin is stable and highly persistent in the environment (EPA 1987, ODW Health Advisory). All uses of dieldrin have been canceled or suspended in the United States (Farm Chemicals Handbook 1990). OSHA thus anticipates that exposure to this substance will be minimal; the PEL is being proposed to achieve the primary objective of this rulemaking: consistency in limits across all OSHA-regulated sectors.

Dieldrin is a convulsant in humans and animals; it also causes cancer and fetotoxicity in animals. The oral LD_{50}s in rats and monkeys are 38 mg/kg and 3 mg/kg, respectively (EPA 1987, p. 4). The dermal LD_{50} in rabbits is reported to range from 150 to 450 mg/kg (Hayes 1982, p. 238). Acutely poisoned animals convulsed before death (Hayes 1982, pp. 238–240). Rats fed dieldrin over a 2-year period developed hemorrhagic or distended urinary bladders (often associated with nephritis) or increases in liver to body weight ratios and pathological changes in the liver (Fitzhugh et al. 1964; Lehman 1959; in EPA 1987, p. 4). Dogs given dieldrin at dietary doses of between 0.2 and 10 mg/kg 6 days/week for as long as 25 months lost weight and had convulsions (Fitzhugh et al. 1964); other dogs given dieldrin by capsule for 2 years at doses of 0.005 or 0.05 mg/kg/day had increases in liver weights and in liver to body weight ratios (Walker et al. 1969, in EPA 1987, p. 5). Dietary administration of dieldrin to mice at doses between 1.25 and 20 ppm for 128 weeks caused a high rate of mortality in the high-dose group and palpable intra-abdominal masses in animals from the 10, 5, and 2.5-ppm groups (Walker et al. 1972, in EPA 1987, p. 5). No liver enlargement occurred in the 1.25-ppm group (Walker et al. 1972). Two carcinogenicity bioassays in mice were positive; dieldrin-exposed animals in both studies developed hepatocellular carcinomas and other liver tumors (Walker et al. 1972; Lehman 1959, in EPA 1987). Several studies in hamsters and mice have shown dieldrin to be teratogenic and embryocidal when administered to pregnant animals (Ottolenghi et al. 1974; Chernoff et al. 1975, in EPA 1987, pp. 5–6).

There have been several fatal accidental poisonings involving dieldrin in humans; the oral dose estimated to be lethal is 5 grams (Hayes 1982, p. 243; Hodge, Boyce, Deichmann, and Kraybill 1967, in ACGIH 1986, p. 196). Occupational poisonings have been reported in applicators and farmers and among workers manufacturing and formulating this substance (Hayes 1982, p. 243). Many of the reported poisoning episodes (between 47 and 100 percent) involved convulsions (Hayes 1982, p. 244). Other signs and symptoms of dieldrin poisoning are headache, nausea, vomiting, a feeling of malaise, dizziness, and muscle jerking (Hayes 1982, p. 244). Most workers who were poisoned recovered completely after treatment (Hayes 1982, p. 244).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to dieldrin poses a significant occupational risk and that establishment of the proposed PEL is necessary to ensure consistency in the PEL for dieldrin across all OSHA-regulated sectors. Because dieldrin has been banned for use in the United States, OSHA believes that exposure to this substance will be minimal.

DIFLUORODIBROMOMETHANE

CAS: 75–61–6; Chemical Formula: CBr_2F_2

H.S. No. 2063

In general industry, construction, and maritime, OSHA's PEL for difluorodibromomethane is 100 ppm as an 8-hour TWA. The ACGIH TLV®–TWA for this substance is also 100 ppm. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 100 ppm for difluorodibromomethane in agriculture. This action will make the PEL for this substance consistent across all OSHA-regulated sectors.

Difluorodibromomethane (also called Freon 12B2) is a colorless, nonflammable liquid that is used primarily as a fire extinguishant (ACGIH 1986, p. 210). It is also used in the synthesis of dyes, pharmaceuticals, and ammonium compounds (Hawley's 1987, p. 368).

Difluorodibromomethane is a respiratory irritant on acute exposure and a central nervous system and liver toxin on chronic exposure (Cralley and Cralley 1985, p. 166). The 15-minute LC_{50} in rats is 55,000 ppm (ACGIH 1986, p. 201). Rats exposed to a 4000-ppm concentration of difluorodibromomethane for 15 minutes showed signs of pulmonary irritation and developed pulmonary edema (ACGIH 1986, p. 201). Dogs and rats exposed for 6 weeks to a difluorodibromomethane concentration of 2300 ppm showed the following exposure-related effects: mortality in 50 percent of the rats and weakness, incoordination, and signs of increasing intoxication in the dogs after a few days of exposure. At autopsy, neurological and hepatic damage was evident (ACGIH 1986, p. 201).

There are no reports of difluorodibromomethane-induced toxicity from industrial use. The thermal decomposition products generated during the use of difluorodibromomethane as a fire extinguishant, however, have caused tinnitus, paresthesia, anxiety reactions, electroencephalographic changes, slurred speech, and decreased psychological test performance in firefighters and other exposed individuals (Clayton and Clayton 1981, p. 3107).

Based on this evidence, OSHA preliminarily concludes that an 8-hour PEL of 100 ppm is necessary in agriculture both to protect exposed agricultural workers from difluorodibromomethane's irritant and systemic effects and to make OSHA's limit for this substance consistent across

all OSHA-regulated sectors. Accordingly, OSHA is proposing an 8-hour TWA of 100 ppm for difluorodibromomethane in agriculture. The Agency believes that this limit will reduce the significant risk of these material health impairments substantially.

DIGLYCIDYL ETHER (DGE)
CAS: 2238–07–5; Chemical Formula: $C_6H_{10}O_3$
H.S. No. 1139

The current OSHA limit for diglycidyl ether (DGE) in construction and maritime is 0.5 ppm as a ceiling concentration. There is no limit in agriculture. The ACGIH-recommended TLV ® for this substance is 0.1 ppm as an 8-hour TWA. NIOSH considers this substance a potential occupational carcinogen and recommends a limit of 0.2 ppm for DGE as a 15-minute ceiling; however, NIOSH concurred (Exs. 8–47, Table N6A) with the limit being proposed when it was recently established for DGE in general industry. OSHA is proposing an 8-hour TWA PEL of 0.1 ppm for DGE in the construction, maritime, and agriculture industries.

Diglycidyl ether is a colorless liquid with a strong, irritating odor. It is used primarily as a chemical intermediate (ACGIH 1986, p. 202; Hawley's 1987, p. 398).

Both the previous ACGIH 0.5-ppm TLVT4 ® and that organization's current TLVT4 ® are based on the results of an animal study reported by Hine and Rowe (1963b, as cited in ACGIH 1986/Ex. 1–3, p. 202) in which rats were administered repeated 4-hour exposures of 20, 3, or 0.3 ppm DGE. Rats exposed to 20 ppm of DGE showed respiratory irritation, loss of body weight, decreased leukocyte count, involution of the spleen and thymus, and hemorrhagic bone marrow. Residual hematopoietic effects were observed among rats exposed to 3 ppm, and no observed effects were noted among rats exposed to 0.3 ppm, even after as many as 60 exposures. The ACGIH's previous TLVT4 ® of 0.5 ppm as a ceiling value was based on the no-observed-effect level of 0.3 ppm reported in the Hine and Rowe (1963b, as cited in ACGIH 1986/Ex. 1–3, p. 202) study and on industrial experience. In 1979, the ACGIH reconsidered its limit for DGE, noting that, "in view of the seriousness of some of the effects produced [in the rat study], a TLVT4 ® below the no-ill-effect level [of 0.3 ppm] would normally be adopted" (ACGIH 1986/Ex. 1–3). The ACGIH consequently revised the TLVT4 ® to 0.1 ppm as an 8-hour TWA. NIOSH agrees that a PEL of 0.1 ppm is appropriate for DGE but notes that this substance meets the criteria for a potential occupational carcinogen (Ex. 8–47, Table N6A).

OSHA preliminarily concludes that the proposed 8-hour TWA limit of 0.1 ppm will protect workers in construction, maritime, and agriculture from the significant risk of DGE-induced hematopoietic and irritant effects, which constitute material health impairments within the meaning of the Act. The risks of DGE exposure range from respiratory irritation to bone marrow effects, and OSHA believes that the proposed limit for DGE will reduce these risks substantially. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DIISOPROPYLAMINE
CAS: 108–18–9; Chemical Formula: $(CH_3)_2CH\text{-}NH\text{-}(CH_3)_2$
H.S. No. 2064

OSHA's current limit for diisopropylamine in construction, maritime, and general industry is 5 ppm as an 8-hour TWA, with a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing to establish an 8-hour TWA PEL of 5 ppm, and a skin notation, for this substance in agriculture. This action will make OSHA's PEL for diisopropylamine consistent across all OSHA-regulated sectors.

Diisopropylamine is a colorless, volatile liquid with a fishy, amine-like odor (ACGIH 1986, p. 204). This substance is used as an intermediate in the production of pesticides, pharmaceuticals, and other chemicals, as a catalyst, and as an experimental hypertensive drug (HSDB 1986). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Diisopropylamine is an irritant of the eyes and upper respiratory tract in humans and animals. The oral LD_{50} in rats is 770 mg/kg, and the LC_{50} in the same species is 4800 mg/m³ (800 ppm) for 2 hours (RTECS 1990). Instilled into rabbit eyes, this substance caused severe irritation (RTECS 1990). Experimental animals (guinea pigs, rabbits, cats, and rats) exposed to a 2207-ppm concentration of diisopropylamine for 3 hours died; before death, these animals exhibited corneal clouding, tearing of the eyes, and signs of severe respiratory irritation. At autopsy, pulmonary edema and hemorrhage were evident (Proctor, Hughes, and Fischman 1988, p. 205). Exposure of animals of the same species to a 777-ppm concentration of diisopropylamine, however, was not fatal (ACGIH 1986, p. 204).

Workers exposed to concentrations of diisopropylamine of 25 to 50 ppm reported experiencing hazy vision, a symptom indicating corneal edema (Treon, Sigmon, Kitzmiller, and Heyroth 1949, in Proctor, Hughes, and Fischman 1988, p. 205). In addition, some workers experienced nausea and headache (Treon et al. 1949). Because diisopropylamine is highly alkaline, prolonged skin contact with this substance is likely to cause dermatitis (Clayton and Clayton 1981, p. 3155).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to diisopropylamine at the concentrations possible in the absence of an exposure limit poses a significant risk of primary irritation to agricultural workers. The Agency considers this effect a material impairment of health and believes that the proposed limit will substantially reduce this risk. Accordingly, OSHA is proposing an 8-hour TWA of 5 ppm, and a skin notation, for diisopropylamine in agriculture. Promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DIMETHYLFORMAMIDE
CAS: 68–12–2; Chemical Formula: $HCON(CH_3)_2$
H.S. No. 2067

In general industry, construction, and maritime, OSHA's current limit for dimethylformamide is 10 ppm (30 mg/m³) as an 8-hour TWA; there is also a skin notation for this substance. There is no PEL in agriculture. The ACGIH has a TLV®–TWA for dimethylformamide of 10 ppm, with a skin notation. NIOSH has no REL for this substance. OSHA is proposing an 8-hour TWA PEL of 10 ppm, and a skin notation, for dimethylformamide in agriculture. Promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

Dimethylformamide is a colorless to light yellow flammable liquid that has a faint ammonia-like odor. It is used primarily as a solvent for polar polymers. It is commonly found in paint removers and is used in selective gas absorption and solvent extraction operations. Dimethylformamide is also used as a co-solvent or booster in protective coatings, adhesives, films, and printing inks (Merck 1983, p. 473; ACGIH 1986, p. 209).

Dimethylformamide is a liver, kidney, lung, and cardiovascular toxin in humans and animals. Exposure to this substance also has been associated with

testicular cancer in humans and reproductive effects in animals. The oral LD_{50} in rats is 2900 mg/kg, and the LC_{50} in mice is 9400 mg/m³ for 2 hours (RTECS 1990). The dermal LD_{50} in rabbits is 4720 mg/kg (RTECS 1990). Instilled into the eyes of rabbits, dimethylformamide caused a moderate degree of irritation; in contact with the skin, this substance caused mild irritation (Grant 1986, p. 348; RTECS 1990). A single 6-hour exposure to 5000 ppm dimethylformamide, or five 6-hour exposures to 2500 ppm, killed all exposed rats; at autopsy, damage to the liver, kidneys, and lungs was evident (E.I. du Pont de Nemours and Co., Inc. 1976, in ACGIH 1986, p. 289). Repeated oral dosing of rats with dimethylformamide (at doses of 450 mg/kg) caused reversible decreases in the rate of body weight gain and liver injury (Kennedy et al. 1986, in Drug Chem. Toxicol. 9(2):147–170). In a 90-day feeding study in rats, a dietary level of 1000 ppm caused slight anemia and an increase in white blood cell counts, while a dietary level of 200 ppm caused no observable effects. Carcinogenicity bioassays in rats by the oral or subcutaneous routes of administration were negative (Druckry, H. et al. 1967, in ACGIH 1986, p. 289). Although dimethylformamide appears not to be teratogenic, inhalation exposure of pregnant rats caused a substantial decrease in fertility, an increase in intrauterine deaths, and a decrease in embryo weight (Gofonckler 1974, in Gigiena i Sanit. 9:7–10). Injected intratesticularly, 0.1 ml of dimethylformamide had arrested spermatogenesis 2 and 7 days after exposure; the testicular epithelium had also become disorganized and white cell infiltration had occurred (Saxena et al. 1985, in J. Toxicol. Environ. Hlth. 15(3–4):583–587). Dimethylformamide is generally not mutagenic in bacterial assays (Kennedy 1986, in CRC Critical Reviews in Toxicol. 17(2):129–182).

In humans, exposure to dimethylformamide causes mild skin irritation, headaches, gastrointestinal symptoms, and cardiovascular effects, as well as kidney and liver damage; exposure has also been associated with testicular and other cancers. This substance also interacts with alcohol to cause an Antabuse®-like reaction. Symptoms of exposure in a worker splashed with liquid dimethylformamide over 28 percent of his body surface area included skin irritation, abdominal pain and vomiting, and elevated blood pressure, all of which resolved completely within 7 days of exposure (Proctor, Hughes, and Fischman 1988, p. 210). Other workers exposed to dimethylformamide have reported abdominal pain, nausea, and vomiting (ACGIH 1986, p. 209). Ingestion of alcohol up to 4 days following inhalation exposure to dimethylformamide may cause facial flushing and palpitations (Genium MSDS 1982, No. 424). A case-control study of 100 workers exposed to a mean concentration of dimethylformamide of 22 mg/m³ matched with 100 controls showed that reports of headache and dyspepsia and an elevated liver injury enzyme (gamma-glutaryl transferase) were associated specifically with chronic dimethylformamide exposure (Cirla, AM et al. 1984, G. Ital. Med. Lav. 6:149–156). Chronic skin contact with this substance also may cause dermatitis (Proctor, Hughes, and Fischman 1988, p. 210). After a case of symptomatic hepatitis developed in a clothing factory worker, further investigation revealed that 36 of 58 workers tested had elevated liver enzymes. Serologic tests excluded infectious causes in 34 of these 36 workers. The results of liver biopsies in four of these workers demonstrated histological changes characteristic of toxic liver damage. Modification of work hygiene practices was associated with decreased liver enzyme levels (Redlich CA et al. 1988, Annals of Internal Medicine 108:680–686). Three cases of testicular cancer were reported in leather tanners exposed to dimethylformamide; the onset occurred after latency periods of 8 to 14 years (Levin et al. 1987, Lancet 11(8568):1154). A cluster of three cases of testicular cancer among 153 white male aircraft repair employees exposed to dimethylformamide led to the identification of four additional cases among 680 employees at a second, occupationally identical site, a statistically significant increase over national testicular cancer incidence rates. The additional finding that the average age of the identified cases was the average age of all employees and not the peak age for testicular cancer suggests an occupational etiology (Ducatman AM et al. 1986, Journal of Urology 136(4):834–836). Among workers followed from 1954 to 1984 for cancer incidence, dimethylformamide exposure was also associated with excess mouth cancers and malignant melanomas. Combined dimethylformamide and acrylonitrile exposure was associated with increased rates of prostate cancer; no dose-response effects were noted for these cancers, however (Chen JL et al. 1988, Journal of Occupational Medicine 30(10):813–818). Another study showed an excess of several cancers, but not testicular cancer, associated with dimethylformamide exposure. The authors suggested that, as a readily absorbed solvent, dimethylformamide may act to increase the absorption of other potentially carcinogenic compounds such as chromium- and cadmium-based dyes (Ducatman AM 1989, Lancet 1(8643):911).

Based on this evidence in humans and animals, OSHA is proposing a 10-ppm 8-hour TWA PEL, and a skin notation, for dimethylformamide in agriculture. The Agency preliminarily concludes that this limit is necessary to substantially reduce the significant risks of kidney, liver, lung, and cardiovascular damage, as well as cancer, associated with exposure to this substance. OSHA believes that each of these effects constitutes a material impairment of health and that the proposed PEL is necessary to substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for dimethylformamide consistent across all regulated sectors.

1,1-DIMETHYLHYDRAZINE

CAS: 57–14–7; Chemical Formula: $(CH_3)_2NNH_2$

H.S. No. 2068

In construction, maritime, and general industry, OSHA's TWA PEL for 1,1-dimethylhydrazine is 0.5 ppm; there is also a skin notation for this substance. There is no PEL in agriculture. The 1987–1988 ACGIH TLV®–TWA for this substance was 0.5 ppm, with a skin notation. The ACGIH also assigns this substance an A2 designation (suspected human carcinogen). The NIOSH REL for 1,1-dimethylhydrazine is 0.06 ppm as a 2-hour ceiling; this represents the lowest concentration that can be detected analytically. OSHA is proposing to establish a PEL of 0.5 ppm as an 8-hour TWA, with a skin notation, for 1,1-dimethylhydrazine in agriculture. This action will make the PEL for this substance consistent across all OSHA-regulated sectors.

1,1-Dimethylhydrazine, also called asym- or unsymmetrical dimethylhydrazine, is used as an intermediate in chemical synthesis, an ingredient in photographic chemicals, a stabilizer for fuel additives, a plant growth regulator, and a component of jet and rocket fuels (ACGIH 1986, p. 210.1(89); Clayton and Clayton 1981, p. 2792). 1,1-Dimethylhydrazine is a flammable, colorless liquid with an ammonia-like odor; it fumes in air and gradually turns yellow (ACGIH 1986, p. 210.1(89)).

1,1-Dimethylhydrazine causes systemic toxicity in the form of central

nervous system effects, hematological effects, and, in animals, cancer. The 4-hour LC_{50} in rats is 250 ppm, and the occluded dermal LD_{50} in rabbits is 156 mg/kg (ACGIH 1986, p. 210.1(89); RTECS 1990). In monkeys, inhalation of a 162-ppm concentration proved lethal within 1 hour (Clayton and Clayton 1981, p. 2802). Acutely poisoned animals convulsed and vomited before death; autopsy revealed pulmonary edema and hemorrhage (NIOSH Criteria Document (Hydrazines) 1978). Dogs exposed repeatedly to a 25-ppm concentration developed ataxia, diarrhea, vomiting, excessive salivation, depression, convulsions, and hemolytic anemia (Clayton and Clayton 1981, p. 2802). Thirty seconds after application to the skin of dogs, 1,1-dimethylhydrazine was detectable in the blood, and exposure by this route also led to corneal opacities (Smith and Clark 1971; Smith and Castaneda 1970, in ACGIH 1986, p. 210.1(89)). This substance causes embryotoxicity when administered intraperitoneally to rats during pregnancy (Keller et al. 1984, in Proctor, Hughes, and Fischman 1988, p. 212). Administered to mice, rats, and hamsters in the drinking water, 1,1-dimethylhydrazine caused a high incidence of angiosarcomas and tumors of the lung, kidney, and liver in mice; liver carcinomas in rats, and vascular and cecal tumors in hamsters. Inhalation bioassays in mice and rats exposed to 0.5 or 5 ppm 1,1-dimethylhydrazine also were positive (ACGIH 1986, p. 210.1(89); IARC 1974, Vol. 4, pp. 137–143). Based on its tumorigenicity in animals, this substance is considered an animal carcinogen by the International Agency for Research on Cancer, NIOSH, the National Toxicology Program, and the American Conference of Governmental Industrial Hygienists.

In humans, accidental exposure to 1,1-dimethylhydrazine at unknown concentrations has caused eye and skin irritation, nausea, chest pain, difficult breathing, a choking sensation, and lethargy (Shook and Cowart 1957, in Proctor, Hughes, and Fischman 1988, p. 212). Eleven workers accidentally overexposed to 1,1-dimethylhydrazine showed signs of liver involvement in laboratory tests but did not develop clinical signs or symptoms (Shook and Cowart 1957). In another group of 26 workers engaged in the production of rocket fuels (and thus exposed to unspecified concentrations of 1,1-dimethylhydrazine), six cases of fatty liver, evidenced by a rise in SGPT levels, occurred (IARC 1974, Vol. 4, p. 141).

Based on this evidence in humans and animals, OSHA preliminarily concludes that workers in agriculture are at significant risk of experiencing the serious and potentially life threatening effects of exposure to 1,1-dimethylhydrazine. OSHA believes that these effects, which include sensory irritation and liver damage, constitute material health impairments. Accordingly, OSHA is proposing an 8-hour TWA PEL of 0.5 ppm, with a skin notation, for this substance in agriculture. Promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DINITRO-O-CRESOL

CAS: 534–52–1; Chemical Formula: $CH_3C_6H_2OH(NO_2)_2$

H.S. No. 2070

In general industry, construction, and maritime, OSHA's permissible exposure limit for dinitro-o-cresol is 0.2 mg/m³ as an 8-hour TWA; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.2 mg/m³ as an 8-hour TWA, and a skin notation, for dinitro-o-cresol, and NIOSH has a REL of 0.2 mg/m³ as a 10-hour TWA. OSHA is proposing an 8-hour TWA PEL of 0.2 mg/m³, and a skin notation, for dinitro-o-cresol in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Dinitro-o-cresol (DNOC) is an odorless, yellow, crystalline solid. It is used as a selective herbicide and plant growth regulator (Hazardous Substance Fact Sheet 1986, p. 1; Gosselin, Smith, and Hodge 1984, p. II–196). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Exposure to dinitro-o-cresol causes metabolic stimulation and yellow pigmentation of the skin, eyes, and hair, and severe exposure can cause coma and death in humans and animals. In rats, the oral LD_{50} is 31 mg/kg, and in rabbits, it is 23.5 mg/kg (ACGIH 1986, p. 215; RTECS 1990). In rats, the dermal LD_{50} is 200 mg/kg (RTECS 1990). Cats survived one 4-hour exposure to the vapors of dinitro-o-cresol at a concentration of 1.4 mg/m³; however, one of three cats died from a single exposure to a concentration of 40 mg/m³ (Burkatskaya 1965, in ACGIH 1986, p. 215). The signs and symptoms of dinitro-o-cresol intoxication are similar in all species and include listlessness, loss of appetite and activity, deep, rapid respiration, sweating, thirst, oliguria, muscular weakness, prostration, dyspnea, and death with terminal hyperpyrexia; signs may be delayed for several hours (Clarke 1981, in HSDB 1990). When about 70 mg/kg of dinitro-o-cresol was given orally to sheep, toxic effects were observed in the liver and kidneys; moderate methemoglobinemia and hemoconcentration were also seen. Of the nine sheep dosed, three survived; all suffered extensive hemolysis followed by anemia (Nehez et al. 1981, in HSDB 1990). Cats subjected to daily 4-hour exposures of 0.2 mg/m³ DNOC for 3 months suffered from sluggishness, loss of muscle tone, dyspnea, a decrease in hemoglobin and red blood cell counts, and an increase in white blood cell counts and blood sugar levels; three cats died (Burkatskaya 1965, in ACGIH 1986, p. 215).

Dinitro-o-cresol exerts its effects by stimulating the metabolic rate and causing hyperpyrexia; if heat production exceeds heat loss, fatal hyperthermia may occur. In humans, the signs and symptoms of acute exposure to dinitro-o-cresol are nausea, gastric upset, restlessness, a sensation of heat, flushed skin, sweating, rapid respiration, tachycardia, fever, cyanosis, and coma (Klaassen, Amdur, and Doull 1986, p. 556). The lowest toxic oral dose in humans is 7500 μg/kg for 7 days; at this level, the exposed individual experienced somnolence and headache (RTECS 1990). The lowest toxic concentration in humans is 1 mg/m³ (RTECS 1990). A single oral dose of 75 mg caused no adverse effects in five volunteers, but two volunteers who received this dose for 5 or more days experienced headaches, lassitude, and malaise; these symptoms occurred when the volunteers' dinitro-o-cresol blood levels reached 20 μg/g or peaks of 40 and 48 μg/g (Harvey, Bidstrup, and Bonnell 1951, in Proctor, Hughes, and Fischman 1988, p. 217). Application of 50 g of an ointment containing 25 percent dinitro-o-cresol to the skin of a 4-year-old boy produced symptoms of vomiting, headache, yellow stained skin and sclera, elevated pulse and respiratory rate, unconsciousness, and death within 3.5 hours (Buchinskiy 1974, in Hayes 1982, p. 469; NIOSH Dinitro-o-cresol Criteria Document 1978). Acute poisoning from dinitro-o-cresol is usually quick; almost complete recovery or death occurs within 24 to 48 hours (Bidstrup and Payne 1951, in Hayes 1982, p. 469). Eight fatalities have been reported in British agricultural workers spraying this substance; death occurred within 48 hours and was caused by

hyperthermia (Bidstrup and Payne 1951). In fatal cases, autopsy shows yellow staining of the organs, tissues, and fluids, congestion of the lungs, edema and a few petechial hemorrhages, and hemorrhagic changes in the brain and gastric mucosa (Bidstrup and Payne 1951, in Hayes 1982, p. 470). Repeated ingestion of dinitro-o-cresol for therapeutic purposes has caused bilateral cataracts, but workers exposed to this substance in agricultural or industrial applications have not shown this effect (NIOSH Dinitro-o-cresol Criteria Document 1978).

Based on this evidence in humans and animals, OSHA preliminarily concludes that dinitro-o-cresol causes yellow pigmentation, hyperpyrexia, and central nervous system effects in humans and animals. OSHA believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that establishing a permissible exposure limit of 0.2 mg/m^3 as an 8-hour TWA, with a skin notation, for dinitro-o-cresol in agriculture is necessary to substantially reduce the risk of these material health impairments. In addition, establishing the proposed limit in agriculture will make OSHA's PEL for dinitro-o-cresol consistent across all OSHA-regulated sectors.

ETHANOLAMINE

CAS: 141-43-5; Chemical Formula: $NH_2CH_2CH_2OH$

H.S. No. 1159

OSHA currently has an 8-hour TWA limit of 3 ppm for ethanolamine in construction and maritime. There is no limit in agriculture. The ACGIH has the same TLV®-TWA limit of 3 ppm, along with a 15-minute STEL of 6 ppm. In construction and maritime, OSHA is retaining the 8-hour TWA PEL of 3 ppm and proposing to supplement this limit with a 6-ppm STEL. OSHA is also proposing to extend both limits to agriculture. NIOSH has no REL but concurred (Ex. 8-47, Table N1) with the proposed limits when they were recently established for ethanolamine in general industry.

Ethanolamine is a colorless liquid with a mild smell like that of ammonia. Ethanolamine is used in agriculture as a chemical (pesticide) dispersing agent and is also used in the manufacture of antibodies. This substance is also used in chemical synthesis and as an ingredient in emulsifiers, polishes, and waving solutions for hair (ACGIH 1986, p. 235; Hawley's 1987, p. 474). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The health hazards associated with exposures to ethanolamine include irritation and necrosis of the skin and central nervous system depression. The oral LD_{50} in rats is 3.32 g/kg, and the intraperitoneal LD_{50} in rats is 981 mg/kg (Hartung and Cornish 1968/Ex. 1-328). The dermal toxicity of ethanolamine is considerably higher, with an LD_{50} of 1 mg/kg reported in the rabbit. Dermal application of the undiluted liquid also caused redness, swelling, and burns comparable to mild first-degree burns (Union Carbide Corporation, as cited in ACGIH 1986/Ex. 1-3, p. 235). The eye injury potential of ethanolamine is just slightly less than that of undiluted ammonia (Carpenter and Smyth 1946/Ex. 1-859). Rats fed 0.5 percent (320 mg/kg/day) ethanolamine in their food for 90 days (Smyth, Carpenter, and Weil 1951/Ex. 1-439) showed no adverse effects, but at 1.28 g/kg/day, fatalities occurred. Treon, Cleveland, Stemmer, and associates (1957/Ex. 1-1172) reported lung, liver, and kidney damage in various species exposed to high concentrations of the vapor and mist. In tests of various species, Weeks and co-workers (1960/Ex. 1-941) reported marked dermal effects from continuous exposures (24 hours/day, 7 days/week, for from 24 to 90 days) at various concentrations of the vapor; at 12 to 26 ppm, dermal effects were less severe, but at 5 ppm, skin irritation was still evident. After 90 days of exposure to 5 ppm, dogs experienced a slight and temporary weight loss as well as decreased activity and alertness (Weeks, Downing, Musselman et al. 1960/Ex. 1-941). In studies of anesthetized dogs, Priddle (1954, as cited in ACGIH 1986/Ex. 1-3, p. 235) reported that sublethal doses of ethanolamine cause central nervous system stimulation, while lethal doses cause CNS depression.

OSHA is retaining its PEL of 3 ppm as an 8-hour TWA in construction and maritime, is proposing to add a 15-minute STEL of 6 ppm for ethanolamine in these industries, and is also proposing to extend both PELs to agriculture. The Agency preliminarily concludes that both of these limits are required to protect workers against the significant risk of irritation and central nervous system effects, which constitute material health impairments that are potentially associated with exposure to ethanolamine. The Agency believes that these limits will substantially reduce these significant risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

ETHYLENE CHLOROHYDRIN

CAS: 107-07-3; Chemical Formula: $ClCH_2CH_2OH$

H.S. No. 1167

OSHA currently has an 8-hour TWA limit of 5 ppm, with a skin notation, for ethylene chlorohydrin in construction and maritime. There is no limit in agriculture. The ACGIH has a TLV®-ceiling of 1 ppm, also with a skin notation. The Agency is proposing a PEL of 1 ppm as a ceiling, with a skin notation, for this substance in construction, maritime, and agriculture. NIOSH has no REL but concurs (Ex. 8-47, Table N1) with the proposed limit, which is the PEL recently established for ethylene chlorohydrin in general industry.

Ethylene chlorohydrin is a colorless liquid with a faint, ethereal odor. It is used in the manufacture of insecticides, as a solvent in organic synthesis, and to activate sprouting of dormant potatoes (Hawley's 1987, p. 485, ACGIH 1986, p. 248). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

A broad range of serious health hazards are associated with exposure to this substance; these include central nervous system effects, cardiovascular effects, liver damage, kidney damage, gastrointestinal effects, skin irritation, eye irritation, and mutagenic effects. The oral LD_{50} for rats is 72 mg/kg, and the intraperitoneal LD_{50} in the same species is 56 mg/kg (Goldblatt and Chiesman 1944/Ex. 1-980). In guinea pigs, the intraperitoneal LD_{50} is 98 mg/kg, and the percutaneous LD_{50} is 205 mg/kg (Wahlberg and Boman 1978/Ex. 1-938).

The inhalation toxicity of ethylene chlorohydrin is also high. Ambrose (1950/Ex. 1-888) reported that a single 1-hour exposure to 7.5 ppm and repeated 1-hour exposures to 2 ppm can be fatal to rats. Exposures of 15 minutes daily at concentrations of from 900 to 1000 ppm were fatal to rats within a few days (Goldblatt and Chiesman 1944/Ex. 1-980).

In subacute and chronic studies, rats have died from a daily dietary dose of 67.5 mg/kg (Oser, Morgareidge, Cox, and Carson 1975/Ex. 1-923). Semenova and associates (1980, as cited in ACGIH 1986/Ex. 1-3, p. 248) reported a 4-month no-effect inhalation level of 0.0033 ppm; at 0.017 ppm, slight CNS changes and alterations in the urinary excretion of nitrogen were observed after 4 months. These investigators also observed increased chromosomal aberrations in the bone marrow in rats exposed at the

0.22-ppm level for 4 months (Semenova, Kazanina, Fedyanina et al. 1980, as cited in ACGIH 1986/Ex. 1–3, p. 248).

Voogt and Vet (1969/Ex. 1–1205) tested ethylene chlorohydrin in *Klebsiella pneumoniae* and found it strongly mutagenic. This finding was confirmed by the Ames test in *Salmonella typhimurium*; ethylene chlorohydrin reacts with DNA, since it inhibits the growth of DNA-deficient bacteria (Rosenkranz and Wlodkowski 1974/Ex. 1–1201). A dose-related increase of liver protein and depletion in glutathione were observed in rats after a single dose of ethylene chlorohydrin ranging from 10 to 50 mg/kg (Friedman, Scalera, Balazs et al. 1977/Ex. 1–1198).

One fatal and several nonfatal cases of poisoning in industrial workers have been reported from exposure (for unspecified periods of time) to ethylene chlorohydrin at levels of between 300 and 500 ppm. An autopsy of the worker who died revealed severe damage to the liver and brain, as well as effects on other organs. The survivors of this incident experienced nausea, vomiting, and irritation of the eyes, nose, and lungs (Bush, Abrams, and Brown 1949/Ex. 1–1196). Dierker and Brown (1944/Ex. 1–1197) reported that a 2-hour inhalation exposure to 300 ppm was fatal in one accidental exposure.

OSHA is proposing a ceiling limit of 1 ppm for ethylene chlorohydrin in construction, maritime, and agriculture, with a skin notation. The Agency preliminarily concludes that this limit and notation will substantially reduce the significant risks of central nervous system and other systemic effects associated with workplace exposures to ethylene chlorohydrin. The skin notation is necessary because ethylene chlorohydrin is readily absorbed through the skin. Promulgation of this limit also will make OSHA's PEL for this substance consistent across all regulated sectors.

ETHYLENEDIAMINE

CAS: 107–15–3; Chemical Formula: $NH_2CH_2CH_2NH_2$

H.S. No. 2082

In general industry, construction, and maritime, OSHA's permissible exposure limit for ethylenediamine is 10 ppm as an 8-hour TWA. There is no limit for this substance in agriculture. The ACGIH has a TLV®–TWA of 10 ppm for ethylenediamine. NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL of 10 ppm for ethylenediamine in agriculture. Promulgation of this limit will make the PEL for ethylenediamine consistent across all OSHA-regulated sectors.

Ethylenediamine is a thick, colorless, alkaline liquid with an ammonia-like odor. It is used as a fungicide; in the manufacture of chelating agents such as EDTA; as a chemical intermediate, solvent, emulsifying agent, and antifreeze inhibitor; and in textile lubricants (ACGIH 1986, p. 249; Hawley's 1987, p. 486). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Ethylenediamine is an irritant of the eyes, mucous membranes, and respiratory tract and, in humans, a skin and pulmonary sensitizer. Repeated exposure causes kidney and liver damage in animals (Proctor, Hughes, and Fischman 1988, p. 241; NIOSH/OSHA Occupational Health Guideline 1981). The oral LD_{50} in rats is 500 mg/kg; in guinea pigs it is 470 mg/kg (RTECS 1990). The lowest lethal concentration in rats is 4000 ppm for 8 hours (RTECS 1990). The dermal LD_{50} in rabbits is 730 mg/kg (Gosselin, Smith, and Hodge 1984, p. II–206). Rabbits experienced severe irritation when 10 mg of ethylenediamine was kept in contact with their skin for 24 hours (RTECS 1990). Instilled into the eye of a rabbit, ethylenediamine caused extreme irritation and corneal damage; a 5-percent solution caused partial corneal opacity (Smyth et al. 1951, in Proctor, Hughes, and Fischman 1988, p. 242). Ethylenediamine has been shown to be a potent sensitizer in guinea pigs; 10 of 10 albino guinea pigs exposed to ethylenediamine became sensitized (HSDB 1986). Rats exposed to a 4000-ppm concentration of ethylenediamine for 8 hours died; however, at 2000 ppm, no deaths occurred (Smyth et al. 1951, in Proctor, Hughes, and Fischman 1988, p. 242). Daily exposure to a 484-ppm concentration of this substance for 30 days was fatal to rats; at a 132-ppm concentration on the same regimen, lung, liver, and kidney damage were seen in these animals at autopsy, although all animals survived the exposures (Pozzani 1954, in Proctor, Hughes, and Fischman 1988, p. 242). In rats exposed to 0.50 g/kg of ethylenediamine daily for two generations, a reduction in body weight and changes in liver and kidney weights were seen in the F_0 and F_1 parents. Microscopic liver lesions occurred with greater prevalence in the F_1 female rats (Yang et al. 1984, in Proctor, Hughes, and Fischman 1988, p. 242).

In humans, exposure to a 400-ppm concentration for 5 to 10 seconds caused intolerable irritation of the nose; 200 ppm caused tingling of the face and nasal irritation, while 100 ppm had no effect (Pozzani 1954, in Proctor, Hughes, and Fischman 1988, p. 242). The lowest toxic concentration of ethylenediamine in humans is estimated to be 200 ppm (RTECS 1990). The use of ethylenediamine in pharmaceutical preparations such as Mycolog cream has caused many cases of skin sensitization (Fisher 1973; Baer et al. 1973, in Proctor, Hughes, and Fischman 1988, p. 242). Skin patch tests conducted between 1972 and 1974 revealed that 6 percent of 3,216 patients were sensitive to a 1-percent ethylenediamine-HCl solution (North American Contact Dermatitis Group 1975, in Proctor, Hughes, and Fischman 1988, p. 242). A 30-year-old man developed asthma as a result of ethylenediamine exposure in the workplace. Two and one-half years after employment, symptoms of sneezing, nasal discharge, and cough began; these progressed over the next 5 months. Inhalation provocation tests on the worker produced chest tightness, cough, wheezing, and a 26-percent drop in FEV_1 4 hours following exposure to ethylenediamine on two different days (Lam 180, in Proctor, Hughes, and Fischman 1988, p. 242).

Based on this evidence in humans and animals, OSHA preliminarily concludes that ethylenediamine causes irritation of the eyes, mucous membranes, and respiratory tract as well as skin and pulmonary sensitization. The Agency believes that, in the absence of a limit for ethylenediamine, workers in agriculture are at significant risk of experiencing these adverse health effects. OSHA believes that the proposed TWA PEL of 10 ppm will substantially reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

FLUORIDES (as F)

CAS: Varies with compound; Chemical Formula: Varies with compound

H.S. No. 2084

In general industry, construction, and maritime, OSHA's permissible exposure limit for fluorides (measured as F) is 2.5 mg/m^3 as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a 2.5 mg/m^3 8-hour TLV®–TWA for these substances. NIOSH also has a REL of 2.5 mg/m^3 for the fluorides as a 10-hour TWA. OSHA is proposing an 8-hour TWA PEL of 2.5 mg/m^3 for the fluorides (measured as F) in agriculture. Promulgation of this limit will make the PEL for these substances consistent across all OSHA-regulated sectors.

The physical properties of the fluorides vary for specific compounds; for example, sodium fluoride is a colorless or blue odorless solid, and cryolite (sodium hexafluoraluminate) is a colorless to dark, odorless solid. Fluorides are used as pesticides and rodenticides, as electrolytes in aluminum manufacture, as fluxes in the smelting of nickel, copper, gold, and silver, as catalysts for organic reactions, as wood preservatives, as fluoridation agents for drinking water, and to clean graphite, metals, windows, and glassware. Exposure to fluorides can also occur during the preparation of fertilizer from phosphate rock (Sittig 1985, p. 456; NIOSH/OSHA Occupational Health Guideline 1981, pp. 1, 3–4). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Fluoride compounds cause eye, skin, and respiratory tract irritation, and skeletal, gastrointestinal, circulatory, and nervous system effects in humans and animals. The oral LD_{50} in rats is 180 mg/kg for sodium fluoride (Lehman 1951–1952; Smyth et al. 1969, Muehlberger 1930, in Hayes 1982, p. 58). The LD_{50} for sodium fluoroacetate in rats is 2.5 mg/kg (Kalmbach 1945, in Hayes 1982, p. 495). A 2 percent aqueous solution of sodium fluoride instilled into rabbit eyes caused corneal epithelial defects and necrotic areas in the conjunctiva (Grant 1986, p. 435). If fluoride is absorbed in sufficient amounts, the fluoride ion increases capillary permeability and causes a coagulation effect; this can lead to hemorrhagic gastroenteritis, congestion, and edema in various organs, including the brain (Booth and McDonald 1982, in HSDB 1986 "Cryolite"). The signs and symptoms of excess fluoride absorption include excitability, muscle tremors, weakness, urination, defecation, salivation, emesis, sudden collapse, clonic convulsions, coma, and death due to respiratory and cardiac failure (Booth and McDonald 1982, in HSDB 1986, "Cryolite"). Fluoroacetate acts on the central nervous system and heart to cause convulsions in dogs, cardiac effects in rabbits, and fits, severe depression, and death due to ventricular fibrillation or cardiac arrest in rhesus monkeys. All species experience a 0.5 to 2-hour delay between ingestion and onset of illness (Hayes 1982, p. 495). Rats fed sodium fluoride at levels of 7 to 14 ppm for 6 weeks developed dental fluorosis manifested as fine lines of impaired calcification; at a dietary level of 226 ppm, the incisors became chalky and pitted. Dietary levels of 904 ppm caused marked weight loss and death within a few weeks (Smith and Leverton 1934, in Hayes 1982, p. 58). Chronic administration of fluoride in oral doses greater than 3.8 mg/kg caused changes in the teeth, liver, and kidneys of rats (IARC 1982, Vol. 27, p. 272). Intraperitoneal doses of stannous fluoride greater than 10 mg/kg of caused embryolethality and teratogenicity in mice (IARC 1982, Vol. 27, p. 272). A recent study in rats and rabbits (Eisenbrandt and Nitschke 1980, in Fund. Appl. Toxicol. 12(3):540–547) exposed by inhalation to various concentrations of sulfuryl fluoride for periods ranging from 2 weeks to 13 weeks reports the following findings. Nine of ten rats exposed to 600 ppm sulfuryl fluoride 6 hours/day, 5 days/week died between the second and sixth exposures; at autopsy, extensive kidney damage was seen in all rats exposed at this level. Rabbits exposed to 600 ppm on the same regimen were hyperactive and one rabbit convulsed. All rabbits exposed either to 300 or 600 ppm for 2 weeks showed vacuolization and/or malacia of the cerebrum at autopsy. In a 13-week study in rats and rabbits, exposure to 30 ppm sulfuryl fluoride caused no effects. Exposure to 100 ppm for this period caused cerebral vacuolization and/or malacia and inflammation of the nasal tissues in some of the rabbits, and exposure to 300 ppm for 13 weeks caused mottling of the teeth, renal effects, pulmonary histiocytosis, nasal-tissue inflammation, and cerebral vacuolization in rats. Some rats exposed to 100 ppm had dental fluorosis. Various species of animals (mice, rats, cattle) have shown signs of impaired reproductive performance after ingesting large amounts (100 mg/l) of fluoride in their drinking water (IARC 1982, Vol. 27, p. 274; Maurer, Cheng, Boysen, Anderson 1990, in J. Nat. Cancer Institute 82(13):1118–1126). Rats fed 4, 10, or 25 mg/kg/day sodium fluoride for up to 99 weeks showed dose-related effects on the teeth, bones, and stomach; animals in the high-dose groups showed a 30 percent decline in weight gain. No increase in the incidence of preneoplastic or neoplastic lesions was seen at any site in rats of either sex. The International Agency for Research on Cancer has concluded that there is insufficient evidence to evaluate the carcinogenicity of sodium fluoride in animals (IARC 1982, Vol. 27, p. 292).

In humans, the lowest toxic dose of fluoride is reported to be 3 mg/kg; this dose produced pulmonary system, liver, and central nervous system effects (RTECS 1990, "Fluoride"). The acute effects associated with ingestion of a toxic dose of a soluble inorganic fluoride compound are vomiting, abdominal pain, and diarrhea (IARC 1982, Vol. 27, p. 276). Absorption of an estimated 0.2 to 27.5 g of fluoride after ingestion has resulted in convulsions; repeated ventricular fibrillation was observed after the ingestion of 120 g of sodium fluoride (IARC 1982, Vol. 27, p. 276). Death is usually due to respiratory paralysis (IARC 1982, Vol. 27, p. 276). In fatal cases of fluoride poisoning, autopsy reveals acute congestion of the abdominal viscera, swelling of the liver and kidneys, tubular necrosis, hemorrhage of the lungs, and dilation of the right chambers of the heart (IARC 1982, Vol. 27, p. 276). Workers exposed to fumes containing fluoride in concentrations above 10 mg/m^3 fluoride reported experiencing irritation and nosebleeds (Williams 1942, in ACGIH 1986, p. 272). Eye and respiratory irritation and nausea occur in workers exposed to concentrations above 5 mg/m^3 (Elkins 1959, in ACGIH 1986, p. 272). Cryolite workers reported experiencing gastric, intestinal, circulatory, respiratory, and nervous system symptoms, as well as skin rashes and problems with their bones, joints, and muscles. The average workroom air concentrations in the cryolite plant ranged from 22 to 48 mg/m^3 (which corresponds to a fluorine level of between 11 and 24 mg/m^3) (Roholm 1937, in ACGIH 1986, p. 272). A recent study in Danish cryolite workers (Grandjean, Horder, and Thomassen 1990, in J. Occ. Med. 32(1):58–63) showed that exposure to cryolite dust at concentrations ranging from 0.16 to 21.2 mg/m^3 for 4 days caused evidence of skeletal fluorosis. Workers in a Yugoslav aluminum processing plant had a significantly increased incidence of telangiectases (enlarged blood vessels); the authors attribute this to exposure to hydrogen fluoride and other fluorides (Balic and Kansky 1988, in Derma. Beruf. Umwelt 36(1):20–22). Repeated contact of the skin with fluoride dust results in dermatitis; several cryolite workers exposed to fluoride concentrations of 15 to 20 mg/m^3 developed an intermittent skin rash (AIHA Hygienic Guide Series 1978). Chronic exposure to fluoride can lead to effects on the teeth, bones, kidneys, and the reproductive and circulatory systems (IARC 1982, Vol. 27, p. 276). Aluminum workers, who are exposed to inorganic fluorides during the production process, showed evidence of bone and joint pathology believed to be attributable to the fluoride exposure (Czerwinski, Nowak, Dabrowska,

Klolarczyk, and Ksiezyk 1988, in Arch. Environ. Hlth. 43(5):340–343). A group of 2258 aluminum workers with an average exposure to fluoride of 17.6 years showed the following prevalence of joint and bone pathology on radiological examination: In those exposed up to 15 years, 135 cases; from 6 to 32 years, 1,463 cases, and in retired workers, 660 cases. A study of potroom workers exposed to fluorides during aluminum smelting showed that the majority had developed fluorosis after 10 years of exposure; after 15 years, moderate to severe osteosclerosis, causing limited mobility of the dorsal spine, developed in many workers (Kaltreider et al. 1972, in ACGIH 1986, pp. 272–273). A recent study (Tokar, Voroshinin, Zhovtak, and Shcherbakov 1989, in Gig. Sanit. 12:85–86) suggests an association between chronic occupational exposure to inorganic fluorides and adverse effects on the parathyroid gland and on the C-cells of the thyroid. The International Agency for Research on Cancer found no evidence of an association between fluoride ingestion and mortality from cancer in humans (IARC 1982, Vol. 27, p. 292).

Based on this evidence in humans and animals, OSHA preliminarily concludes that fluoride causes eye, skin, and respiratory irritation, and skeletal, gastrointestinal, circulatory, and nervous system effects in exposed individuals. OSHA therefore believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that extending the proposed limit of 2.5 mg/m^3 as an 8-hour TWA to agriculture is necessary to significantly reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for fluorides consistent across all regulated sectors.

GLYCIDOL (2,3–EPOXY–1–PROPANOL)
CAS: 556–52–5; Chemical Formula: $C_3H_6O_2$
H.S. No. 1189

Currently, OSHA has an 8-hour TWA limit for glycidol of 50 ppm in construction and maritime. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 25 ppm for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA limit of 25 ppm for glycidol in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Glycidol is a colorless, combustible liquid. It is used as a stabilizer, a dye-leveling agent, and a demulsifier (Hawley's 1987, p. 569; ACGIH 1986, p. 287).

Glycidol causes eye, respiratory, and pulmonary irritation. Hine and associates (1956/Ex. 1–331) conducted a study of animal toxicity caused by glycidol exposure and reported that glycidol is irritating to the lungs: mice and rats both exhibited pneumonitis and emphysema as a result of vapor inhalation. The LC_{50} in mice is 450 ppm for a 4-hour exposure; the 8-hour LC_{50} in rats is 580 ppm (Hine, Kodama, Wellington et al. 1956/Ex. 1–331). A single dermal application was only mildly irritating (Draize score 4.5); however, repeated daily skin applications were severely irritating after 4 days. One drop of pure glycidol in the rabbit eye caused severe but reversible corneal injury (Hine, Kodama, Wellington et al. 1956/Ex.1–331). In rats, chronic exposures to 400 ppm (7 hours/day for 50 days) did not cause systemic toxicity, but eye irritation and respiratory distress were observed after the first few exposures (Hine, Kodama, Wellington et al. 1956/Ex. 1–331). A study to determine glycidol's tumorigenic potential on the skin of mice showed negative results (Van Duuren, Langseth, Goldschmidt, and Orris 1967/Ex. 1–1203).

OSHA is proposing to establish an 8-hour TWA limit of 25 ppm for glycidol in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors from the significant risk of eye, respiratory, and pulmonary irritation potentially associated with exposure to this substance. The Agency believes that this limit will substantially reduce these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

HEPTACHLOR
CAS: 76–44–8; Chemical Formula: $C_{10}H_5Cl_7$
H.S. No. 2088

In general industry, construction, and maritime, OSHA's current permissible exposure limit for heptachlor is 0.5 mg/m^3 as an 8-hour TWA, with a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit for heptachlor in agriculture. The ACGIH's current TLV®–TWA for this substance is 0.5 mg/m^3, with a skin notation. NIOSH has no REL for heptachlor but concurs (Ex. 8–47, Table N3A) with the limit being proposed. The Agency is proposing to establish an 8-hour TWA PEL of 0.5 mg/m^3, and a skin notation, for heptachlor in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Heptachlor is a white to light tan waxy solid with a mild camphor-like odor. Heptachlor has been used as an insecticide, although its use has been restricted by the U.S. Environmental Protection Agency to subsurface soil treatment for termite control and the dipping of non-food plants (Hayes 1982, p. 233; ACGIH 1986, p. 296). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Heptachlor is a convulsant in humans and animals and a liver toxin and carcinogen in animals. In humans, exposure to heptachlor has also caused blood dyscrasias (EPA 1987 (ODW Health Advisory)). The oral LD_{50} in rats is 40 mg/kg; in mice, it is 68 mg/kg (RTECS 1990). The dermal LD_{50} in rabbits is 2000 mg/kg; rabbits dermally exposed convulsed before death (RTECS 1990). Rats given a single oral 90-mg/kg dose of heptachlor exhibited tremors and convulsions within 30 to 60 minutes of administration; liver necrosis was observed in these animals at autopsy (von Oettingen 1955, in Proctor, Hughes, and Fischman 1988, p. 268). Rats fed 7 to 12 mg/kg heptachlor per day for as long as 14 days showed evidence of severe liver damage and changes in liver function (Krampl 1971); these effects were also seen at a dietary level of 10 mg/kg for 5 to 7 days (Enan et al. 1982, both cites in ODW Health Advisory). In oral carcinogenicity bioassays involving heptachlor (contaminated with 20 percent chlordane), mice of both sexes developed liver carcinomas and female rats exhibited signs suggestive of a carcinogenic effect on the thyroid (IARC 1979, Vol. 20, p. 148). The International Agency for Research on Cancer has concluded that the evidence of heptachlor's carcinogenicity in animals is limited (IARC 1982, Suppl. 4, p. 81).

Humans exposed to chlordane that contained up to 20 percent heptachlor exhibited the same nervous system effects as those seen in heptachlor-poisoned animals: dyspnea, tremors, excessive salivation, irritability, and convulsions (EPA 1987, p. 5). These effects occurred regardless of the route of exposure: Dermal, ingestion, or inhalation. There are several reports of blood dyscrasias and aplastic anemia in workers exposed to heptachlor and chlordane (IARC 1982, Suppl. 4, p. 81). Epidemiological studies in chlordane and heptachlor applicators and of cyclodiene pesticide formulators have

yielded inconclusive results (IARC 1982, Suppl. 4, p. 81). Other studies suggest a relationship between heptachlor or chlordane exposure (alone or with other compounds) and blood dyscrasias, acute leukemia, and the development of neuroblastomas in children (IARC 1979, Vol. 20, p. 146).

Based on this evidence in humans and animals, OSHA preliminarily concludes that heptachlor is a convulsant, liver toxin, and, based on effects seen in animals, a potential occupational carcinogen. OSHA therefore preliminarily concludes that, in the absence of a limit for heptachlor, workers in agriculture are at significant risk of experiencing these exposure-related health effects. The Agency believes that the proposed TWA PEL of 0.5 mg/m^3, with a skin notation, is necessary to substantially reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

HEXAFLUOROACETONE

CAS: 684–16–2; Chemical Formula: C_3F_6O

H.S. No. 1198

Currently, OSHA has no limit for hexafluoroacetone in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 0.1 ppm, with a skin notation, for this substance. OSHA is proposing a PEL of 0.1 ppm as an 8-hour TWA, with a skin notation, for this substance in construction, maritime, and agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the proposed limit, which was recently established for this substance in general industry.

Hexafluoroacetone is a colorless, nonflammable, highly reactive gas. It is used primarily in organic synthesis (ACGIH 1986, p. 303).

Inhalation studies of hexafluoroacetone in animals have shown varied systemic toxicities, including injury to the liver, kidney, testes, thymus, and bone marrow. In rats and dogs exposed 6 hours/day, 5 days/week for 13 weeks at concentrations of about 0.1, 1.0, or 12 ppm, no effects (other than increased lung weights in dogs) were observed in either species at 0.1 ppm. However, the 12-ppm exposures produced severe effects in both species, including marked but reversible testicular damage and slight hypoplasia of the spleen, thymus, and lymph nodes (E.I. du Pont de Nemours & Company, Inc. 1971, as cited in ACGIH 1986/Ex. 1–3, p. 303). Reversible kidney damage in rats and increased lung weights in dogs occurred during the 1.0-ppm exposures. An earlier 4-hour acute exposure of rats demonstrated that 300 ppm was a lethal concentration (E.I. du Pont de Nemours & Co., Inc. 1971, as cited in ACGIH 1986/Ex. 1–3, p. 303).

In rats, 2-week dermal exposures of 65, 130, or 250 mg/kg resulted in numerous adverse effects, including testicular damage and corresponding changes in lipid metabolism (Kennedy, Henry, Chen, and Dashiell 1982/Ex. 1–1038). A dermal dose of 13 mg/kg produced no adverse effects in rats (Lee and Gillies 1984/Ex. 1–561). An injected dose of radiolabeled hexafluoroacetone was, for the most part, rapidly excreted in the urine in unmetabolized form; this material also did not accumulate in rat testes (Gillies and Rickard 1984/Ex. 1–322). Brittelli and co-workers (1979/Ex. 1–300) reported that hexafluoroacetone was fetotoxic in rats. Dermal application of 90 mg/kg/day to pregnant rats resulted in maternal toxicity. Fetal toxicity occurred at maternal doses of 25 mg/kg, and fetal size was reduced at maternal doses of 5 and 25 mg/kg; however, 1 mg/kg produced no fetal effect. Although soft-tissue damage and external abnormalities were observed, teratogenicity could not be demonstrated definitively (Brittelli, Culik, Dashiell, and Fayerweather 1979/Ex. 1–300).

OSHA is proposing an 8-hour TWA PEL of 0.1 ppm TWA and a skin notation for hexafluoroacetone in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors from the significant risk of systemic injuries, reproductive effects, kidney damage, and fetotoxic effects, all of which constitute material health impairments that are associated with exposure to hexafluoroacetone at levels above the proposed PEL. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

HYDROGEN CYANIDE

CAS: 74–90–8; Chemical Formula: HCN

H.S. No. 1207

The OSHA limit for hydrogen cyanide in construction and maritime is a 10-ppm 8-hour TWA, with a skin notation. There is no limit in agriculture. The ACGIH has a 10-ppm ceiling limit, also with a skin notation. NIOSH (1976e/Ex. 1–240) has recommended that workplace exposures to hydrogen cyanide not exceed 4.7 ppm (5mg/m^3) as a 10-minute ceiling; however, NIOSH concurs (Ex. 8–47, Table N1) with the selection of the proposed PEL. OSHA is proposing a 15-minute STEL of 4.7 ppm, and a skin notation, for hydrogen cyanide in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Hydrogen cyanide is a colorless gas at room temperature; it has a faint odor of bitter almonds. If contaminated with impurities or not adequately stabilized, hydrogen cyanide will polymerize spontaneously and violently. The commercially available product is usually 96 to 99 percent pure and contains a stabilizer. It is used in the manufacture of pesticides, rodenticides, chelates, dyes, and other substances including acrylonitrile, acrylates, adiponitrile, and cyanide salts (Hawley's 1987, p. 615). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The ACGIH (1986/Ex. 1–3) has summarized the extensive body of human evidence on the adverse effects resulting from exposure to hydrogen cyanide. The Documentation notes that exposure to levels of 45 to 54 ppm hydrogen cyanide can be tolerated for 1 hour with no immediate or delayed effects, but that 18 to 36 ppm produces "slight" symptoms after several hours of exposure. The ACGIH also cites Grabois (1954/Ex. 1–1150), who reported that workers in apricot kernel processing plants experienced no ill effects when exposed to hydrogen cyanide at a concentration of approximately 10 ppm.

The NIOSH recommendation of 4.7 ppm as a 10-minute ceiling limit is based largely on an epidemiologic study by El Ghawabi et al. (1975/Ex. 1–632) that showed an increase in symptoms of headache, weakness, throat irritation, vomiting, dyspnea, lacrimation, colic, and nervousness among workers exposed to cyanide for an average of 7.5 years. The 36 male workers who were studied were employed in three electroplating factories. Breathing zone samples (15 minutes in duration) were collected, and results ranged from 4.2 to 12.4 ppm. Cyanide levels at two of the three plants did not exceed 9.6 ppm. El Ghawabi et al. (1975/Ex. 1–632) also reported that two workers in one plant suffered from psychotic episodes; these conditions were reported to be similar to cases that occurred during the therapeutic use of thiocyanate. Mean values of urinary thiocyanate in the 36 workers correlated well with air concentrations of cyanide (El Ghawabi, Gaafar, El-Saharti et al. 1975/Ex. 1–632).

Symptoms resulting from chronic exposure to cyanide were also reported by Radojicic (1973, as cited in NIOSH 1976e/Ex. 1–240) among workers exposed to HCN levels between 5.4 and

12.3 ppm, and by Saia, DeRosa, and Galzigna (1970, as cited in NIOSH 1976e/Ex. 1–240). NIOSH (1976e/Ex. 1–240) interpreted the significance of these studies as follows:

Colle (1972) * * * advanced the belief that these symptoms of headache, dyspnea, epigastric burning, vertigo, tinnitus, nausea, vomiting, tremor, and precordial pain represent a true clinical entity and that they are sufficiently documented and characteristic of chronic cyanide exposure to be grouped into a true syndrome. * * *

Chaumont (1960) * * * also stated that there is no clinical evidence to deny that cyanides can cause this type of occupational intoxication. He apparently found the debate on whether this intoxication is truly chronic or whether it involves repeated subacute symptoms to be semantic in nature and opted for the admission that chronic intoxication caused by HCN and the cyanide salts is a true occupational disease. * * *

Thus, one might describe chronic cyanide poisoning as a slow deterioration of resistance, and, therefore, an intensified sensitivity, due to inadequate time between exposures for replacement of damaged tissues, enzyme systems and metabolic stores, the elimination of detoxication products, and the regeneration of homeostatic mechanisms (NIOSH 1976e/Ex. 1–240, pp. 90–91).

Based on this evidence, OSHA preliminarily concludes that a variety of symptoms are associated with exposure to hydrogen cyanide at concentrations below 10 ppm. OSHA is therefore proposing a 4.7-ppm 15-minute STEL, and a skin notation, for hydrogen cyanide in construction, maritime, and agriculture. The Agency believes that the short-term limit will protect workers in these sectors from the significant risk of headache, weakness, colic, and nervousness, which constitute material impairments of health; these effects have been observed in individuals exposed at the 10-ppm level over a full working shift. OSHA preliminarily concludes that this limit will substantially reduce these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

HYDROGEN SELENIDE (as Se)
CAS: 7783–07–5; Chemical Formula: H_2Se
H.S. No. 2093

In general industry, construction, and maritime, OSHA's current permissible exposure limit for hydrogen selenide (measured as selenium) is 0.05 ppm. There is no limit in agriculture. The ACGIH's TLV®–TWA for this substance is 0.05 ppm as an 8-hour TWA. NIOSH has no REL for this substance. The Agency is proposing to establish an 8-hour TWA PEL of 0.05 ppm for hydrogen selenide in agriculture. Promulgation of this limit will make the PEL for hydrogen selenide consistent across all OSHA-regulated sectors.

Hydrogen selenide is a colorless gas with a disagreeable odor like that of decaying horseradish. Hydrogen selenide is used to prepare metallic selenides and organoselenium compounds and in doping gas mixtures to prepare semiconductor materials (ACGIH 1986, p. 317; Braker and Mossman 1980, p. 404).

Hydrogen selenide is an irritant of the eyes, mucous membranes, and respiratory tract in humans and animals. The lowest lethal concentration in rats is 20 mg/m³ for 1 hour (RTECS 1990). The LC_{50} in guinea pigs is 0.3 ppm (RTECS 1990). Signs of mucous membrane irritation, pulmonary edema, bronchitis, and bronchial pneumonia were seen in rats exposed to hydrogen selenide concentrations of 1 to 1.2 ppm (Buchan 1947, in Clayton and Clayton 1982, p. 2133). At a concentration of 10 ppm for 2 hours, guinea pigs exhibited signs of eye and nose irritation, and many of these animals died from pneumonitis (AIHA Hygienic Guide Series 1959, pp. 514–515).

The lowest toxic concentration of hydrogen selenide in humans is estimated to be 0.2 ppm (HSDB 1985). Exposure to a hydrogen selenide concentration of 1.5 ppm was judged intolerable by human subjects because of eye and nasal irritation. Exposure for a few minutes to a 0.3 ppm-concentration caused no irritation (Grant 1974, p. 560). Five workers exposed to hydrogen selenide at concentrations of less than 0.2 ppm for 1 month experienced nausea, vomiting, diarrhea, a metallic taste in the mouth, garlic odor of the breath, dizziness, lassitude, and fatigability; these symptoms gradually subsided during the months following exposure (Buchan 1974, in Proctor, Hughes, and Fischman 1988, p. 283). Other signs and symptoms caused by exposure to hydrogen selenide include runny nose and eyes, cough, sneeze, tightness of the chest, and pulmonary edema (Glover 1970, in ACGIH 1986, p. 317).

Based on this evidence in humans and animals, OSHA preliminarily concludes that hydrogen selenide's irritant effects pose a significant risk to workers in agriculture. The Agency believes that the proposed 8-hour TWA limit of 0.05 ppm is necessary to reduce the significant risk of material health impairment potentially associated with exposure to this substance. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

HYDROGENATED TERPHENYLS
CAS: 61788–32–7; Chemical Formula: None
H.S. No. 1210

Currently, OSHA does not regulate the hydrogenated terphenyls in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 0.5 ppm (approximately 5 mg/m³) for these complex mixtures of ortho-, meta-, and para-terphenyls in various stages of hydrogenation. OSHA is proposing a PEL of 0.5 ppm as an 8-hour TWA for hydrogenated terphenyls in construction, maritime, and agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the proposed limit, which was the PEL recently established for these substances in general industry.

The hydrogenated terphenyls are mixtures of ortho-, meta-, and para-terphenyls in various stages of hydrogenation. Hydrogenated terphenyls are used as heat-transfer media and as plasticizers (ACGIH 1986, p. 311).

Acute exposure to the hydrogenated terphenyls poses a risk of potential lung, eye, and skin damage. Chronic exposure presents a risk of systemic toxicity involving injury to the liver, kidneys, and blood-forming organs, as well as possible metabolic disturbances and cancer (ACGIH 1986/Ex. 1–3, p. 311).

Early studies of unhydrogenated terphenyl isomers determined that the LD_{50} in rats is low, i.e., 1900 mg/kg for the ortho isomer, 2400 mg/kg for the meta isomer, and 10,000 mg/kg for the para isomer (Cornish, Bahor, and Ryan 1962/Ex. 1–410). Thirty-day oral administration of 500 mg/kg/day in the diet of rats indicated possible liver and kidney damage, which was suggested by increases in the liver- and kidney-to-body-weight ratios and decreases in the rate of weight gain (Cornish, Bahor, and Ryan 1962/Ex. 1–410). Other studies have demonstrated nephrotoxicity and liver damage in rats fed 33 mg/kg or more of unirradiated terphenyl isomers (Petkau and Hoogstraaten 1965/Ex. 1–432; Young, Petkau, and Hoogstraaten 1969/Ex. 1–459). Inhalation studies showed that bronchopneumonia is associated with exposure to 88 to 356 ppm of the ortho and meta isomers, but not to the para isomer at 103 ppm (Haley, Detrick, Komesu et al. 1959/Ex. 1–326). The work of Cornish, Bahor, and Ryan (1962/Ex. 1–410) showed that none of the isomers caused skin irritation in rabbits following a 24-hour dermal application. For terphenyls that are approximately 40-percent hydrogenated, the acute oral LD_{50} in rats is reported as 17,500 mg/kg; in mice, it is 12,500 mg/kg (Adamson and Weeks 1973/Ex. 1–295).

This study also demonstrated that an irradiated hydrogenated terphenyl mixture is three times more acutely toxic by ingestion than is a nonirradiated mixture. This finding was confirmed in 16-week chronic ingestion studies (Adamson, Bowden, and Wyatt 1969/Ex. 1–293); these authors found that 1200 mg/kg of an irradiated mixture was lethal to mice, while the same dose in nonirradiated form produced only an irreversible interstitial nephritis. In the same study, no effects were observed for either mixture at a dose level of 250 mg/kg.

Eight-day inhalation studies in mice showed some pathologic changes in lung tissue after 500 mg/m³ (50 ppm) exposures to nonirradiated hydrogenated terphenyls; 8-week exposures at 2000 mg/m³ (200 ppm) resulted in the same lung damage, as well as in some proliferation of the smooth endoplastic reticulum in the liver (Adamson, Bowden, and Wyatt 1969/Ex. 1–293; Adamson and Weeks 1973/Ex. 1–295). Carcinogenesis in mice has been reported from 8-week skin exposures to the irradiated mixture (Henderson and Weeks 1973/Ex. 1–784). The significance of the changes observed by Adamson and Furlong (1974/Ex. 1–294) in the mouse lung after 8 weeks of inhalation exposure to the irradiated mixture is difficult to interpret in terms of the potential of the hydrogenated terphenyls to cause pulmonary cancer; particles were found to clear the lungs rapidly but to accumulate and clear more slowly in the intestine, kidney, and liver.

OSHA is proposing a 0.5-ppm 8-hour TWA PEL in construction, maritime, and agriculture for the complex mixtures of ortho-, meta-, and para-terphenyls (either irradiated or nonirradiated) in various stages of hydrogenation. The Agency preliminarily concludes that this limit will protect workers in these sectors from the significant risks of eye, skin, and lung damage and of systemic toxicity to the liver, kidneys, and blood-forming organs, all material health impairments that are potentially associated with exposure to these substances at levels above the proposed PEL. In addition, promulgation of this limit will make OSHA's PEL for these substances consistent across all regulated sectors.

2-ISOPROPOXYETHANOL

CAS: 109–59–1; Chemical Formula: $(CH_3)_2CHOCH_2CH_2OH$

H.S. No. 1223

OSHA has no limit for 2-isopropoxy-ethanol in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 25 ppm for this substance. NIOSH has no REL. OSHA is proposing a PEL of 25 ppm as an 8-hour TWA for this substance in construction, maritime, and agriculture. This is the limit recently established for 2-isopropoxyethanol in general industry.

2-Isopropoxyethanol is a mobile, combustible liquid. It is used as a solvent for resins, a dye for textiles, and as a component of lacquers and other coatings (ACGIH 1986, p. 335; Hawley's 1987, p. 660).

2-Isopropoxyethanol has been demonstrated to produce systemic toxicity in laboratory animals. In studies of rats, 15 6-hour exposures to 1000 ppm caused hemoglobinuria, anemia, and lung congestion, but no fatalities (Gage 1970/Ex. 1–318). At 300 ppm, Gage reported blood changes and lung congestion after 15 exposures. Exposure at the 100-ppm level produced no effect (Gage 1970/Ex. 1–318). Another study reported a significant increase in the osmotic fragility of erythrocytes in female rats after a 4-hour inhalation exposure to 62 ppm, but no effect was observed at 32 ppm (Carpenter, Pozzani, Weil et al. 1956/ Ex. 1–303). Studies of four species of experimental animals exposed at concentrations of 200, 50, or 25 ppm for 6 hours/day for 26 weeks reported hematologic changes only in rats; increased osmotic fragility of erythrocytes was marked at 200 ppm, slight at 50 ppm, and minimal at 25 ppm (Moffett, Linnett, and Blair 1976, as cited in ACGIH 1986/Ex. 1–3, p. 235).

In the prior rulemaking, NIOSH (Ex. 8–47) did not concur with OSHA's proposed limit of 25 ppm, noting that 25 ppm represented an effect level. Although "slight" increases in osmotic fragility were reported in animals subchronically exposed (Moffett, Linnett, and Blair 1976, as cited in ACGIH 1986/Ex. 1–3, p. 235), OSHA notes that a marked reaction did not occur until exposure was increased eightfold. Therefore, at this time, OSHA judges the 25-ppm PEL to be sufficiently protective.

OSHA is proposing to establish an 8-hour TWA PEL of 25 ppm for 2-isopropoxyethanol in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that this limit will substantially reduce the significant risk of hemolytic effects, which are material health impairments, that are associated with exposure to this substance. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ISOPROPYL GLYCIDYL ETHER (IGE)

CAS: 4016–14–2; Chemical Formula: $C_6H_{12}O_2$

H.S. No. 1227

OSHA's current limit for isopropyl glycidyl ether (IGE) in construction and maritime is 50 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 50 ppm and a 15-minute TLV®–STEL of 75 ppm for IGE. NIOSH recommends a limit of 50 ppm as a 15-minute ceiling. OSHA is retaining the 8-hour TWA of 50 ppm and proposing to add a 15-minute STEL of 75 ppm for IGE in construction and maritime; the Agency is also proposing to extend both limits to agriculture. These are the limits recently established for this substance in general industry.

IGE is a colorless, volatile liquid. It is used as a stabilizer for chlorinated solvents and as a viscosity reducer for epoxy resins (Hawley's 1987, p. 662).

The 4-hour LC_{50} for IGE in mice is 1500 ppm, and the 8-hour LC_{50} in rats is 1100 ppm (Hine, Kodama, Wellington et al. 1956/Ex. 1–331). The intragastric LD_{50}s in mice and rats are 1.30 and 4.2 g/kg, respectively; in rabbits, the dermal LD_{50} is 9.65 g/kg (Hine, Kodama, Wellington et al. 1956/Ex. 1–331). Fifty daily 7-hour exposures of rats to 400 ppm caused a reduced rate of weight gain, an increase in hemoglobin, a decrease in peritoneal fat, and, in some animals, emphysematous lungs and mottling of the liver (Hine, Kodama, Wellington et al. 1956/Ex. 1–331). Animals in this study also exhibited signs of ocular irritation and respiratory distress.

In humans, eye, nose, and upper respiratory tract irritation occurred in the technicians handling the animals in the Hine and co-workers' study (1956/Ex. 1–331); exposure levels were not specified. Dermatitis has also been reported in workers exposed to other glycidyl ethers during manufacture, and one such case involved IGE exposure (ACGIH 1986/Ex. 1–3, p. 340).

In construction and maritime, OSHA is retaining the 8-hour TWA of 50 ppm and proposing to add a 15-minute STEL of 75 ppm for IGE; the Agency is also proposing to extend both limits to agriculture. The Agency preliminarily concludes that both the TWA and STEL are necessary to reduce the risk to these workers of chronic organ effects, such as those demonstrated to occur in animals (Hine, Kodama, Wellington et al. 1956/Ex. 1–331), and the significant risk of eye, skin, and upper respiratory tract irritation associated with short-term IGE exposures at the levels permitted in the absence of a STEL. OSHA considers sensory irritation, dermatitis, and chronic organ effects to be material impairments of health. In addition, promulgation of these limits

will make OSHA's PELs for this substance consistent across all regulated sectors.

LEAD, INORGANIC (as Pb)
CAS: 7439–92–1; Chemical Formula: Pb
H.S. No. 2098

The current OSHA limit for inorganic lead in the construction and maritime sectors is 0.2 mg/m³ (200 μg/m³), and in general industry it is 50 μg/m³. There is no PEL for lead in agriculture. The ACGIH has a TLV®–TWA of 0.15 mg/m³ for lead. NIOSH has a REL of <100 μg/m³ as a 10-hour TWA. OSHA is proposing an 8-hour TWA PEL of 50 μg/m³ for lead in the construction, maritime, and agriculture sectors. This is the limit established by OSHA in 1978 for lead in general industry workplaces (see 29 CFR 1910.1025).

Lead is a ductile, soft, heavy, gray, metallic element. It is used in batteries, radiation shielding, ammunition, solder, and in cable coverings. It has many other industrial uses (Hawley's 1987, p. 687).

Lead has profound and adverse effects on the health of exposed workers. Inhalation, the most important source of lead intake, and ingestion result in damage to the nervous, urinary, and reproductive systems and additionally inhibit synthesis of the heme molecule, which is responsible for oxygen transport in living systems. The adverse health effects associated with exposure to lead range from acute and perhaps reversible effects such as inhibition of enzyme activity, reduction in motor nerve conduction velocity, behavioral changes, and mild central nervous system (CNS) symptoms, to chronic disease and death.

The signs and symptoms of severe lead intoxication associated with blood lead levels of 80 μg/100 g and above are well documented. In recent years, however, there have been numerous research findings indicating that adverse health effects are associated with lead concentrations previously thought to be inconsequential. The following subsections will address the health effects of low level lead exposure in each system: heme synthesis inhibition and damage to the nervous, urinary, and reproductive systems. Prior to this, however, the air to blood lead relationship will be addressed.

Air to blood relationship. OSHA believes that a relationship between air lead levels and population-average blood lead levels unquestionably exists. In order to accurately predict the effects on blood lead levels that are produced over time by changes in air lead levels, it was necessary to construct a model that takes into account the important factors that affect blood lead levels. The adaptation of the physiological model originally developed by S.R. Bernard by the Center for Policy Alternatives (CPA) (Doc. H–004, Ex. 439) combines experimentally observed properties of mammalian lead transport and metabolism, including considerations of the dynamics of blood lead response to long-term exposure with observed physical properties of airborne particulates encountered in the workplace to produce a complete and accurate picture of the response of blood lead levels to particulate lead exposure. The Bernard model is an example of one of the most common types of models used to describe the transport and metabolism of drugs or foreign substances in the body; models of this type are known as multicompartment mammalian models. OSHA believes that the CPA's application of the Bernard model accurately predicts the effects on blood lead levels that are produced over time by changes in air lead levels.

Based on calculations using the CPA adaptation of the Bernard model, OSHA predicts that, assuming compliance with the proposed PEL of 50 μg/m³, 0.5 percent of all blood lead levels (PbB) collected from exposed workers will exceed 60 μg/100 g; 5.5 percent of workers will have a PbB between 50 and 60 μg/100 g; 23.3 percent will have levels between 40 and 50 μg/100 g; and overall, 29.3 percent will have PbBs above 40 μg/100 g at any one time. These blood lead levels would represent a substantial improvement in the blood lead levels for workers in these sectors under current conditions. The current blood lead level distribution, assuming compliance with a 200 μg PEL, is approximately (1) 22.4 percent of workers with PbBs greater than 60 μg/100 g; (2) 32.6 percent with PbBs between 50 and 60 μg/100 g; (3) 28.7 percent of workers with PbBs between 40 and 50 μg/100 g; and (4) overall, 83.3 percent with PbBs above 40 μg/100 g.

Heme Synthesis Inhibition. The earliest demonstrated effect of lead involves its ability to inhibit the formation of heme. Scientific evidence has established that lead inhibits at least two enzymes of the heme synthesis pathway at very low blood lead (PbB) levels. Inhibition of delta aminolevulinic acid dehydrogenase (ALAD), an enzyme responsible for the synthesis of a precursor to heme, is observed at PbB levels below 20 μg/100 g (Hernberg et al., Doc. H–004, Ex. 6–20). At a PbB level of 40 μg/100 g, more than 20 percent of the population would have a 70 percent inhibition of ALAD. Another product of impairment that builds up and that indicates inhibition of another enzyme, ferrochelatase, also occurs at low PbB levels. At a PbB level of 50 μg/100 g, a larger proportion of the population would suffer a more extreme version of these effects. At a PbB level of 50 μg/100 g, 70 percent of the population would have a 70 percent inhibition of ALAD and 80 percent of men and 100 percent of women would have increased free erythrocyte protoporphyrin, which is the product of inhibition of ferrochelatase (Zeilhuis, Doc. H–004, Ex. 294E).

The depression of heme synthesis in all cells of the body is a potentially far-reaching effect, and the prevention of enzyme effects is thus the key to the prevention of more serious clinical effects of lead toxicity, which become more obvious as the exposure continues. These measurable effects are a direct result of lead exposure and are considered by the Agency to indicate the occurrence of disruptions of a fundamental and vital subcellular process, heme synthesis.

OSHA believes that the toxicological evidence indicates a progression of health effects caused by lead exposure that starts with the inhibition of enzymes, continues through effects demonstrating a measurable disruption of subcellular processes (such as the buildup of the products of impaired heme synthesis), and eventually develops into overt signs and symptoms of lead poisoning that are manifested as disorders of the nervous, renal, and blood-forming systems. Given this understanding of the progressive stages of lead toxicity, OSHA has preliminarily concluded that enzyme effects indicative of the disruption of heme synthesis are early stages of a disease process that eventually results in the clinical symptoms of lead poisoning. Whether or not the effects have proceeded to the later stages of clinical disease, disruption of these processes over a working lifetime must be considered a material impairment of health. As was previously discussed, at a PbB level of 40 μg/100 g and above, a significant proportion of the population would manifest extensive inhibition of ALAD as well as elevations of protoporphyrin levels. The Agency believes that PbB levels should ideally be kept below 40 μg/100 g to minimize these effects.

Neurological effects. Lead exposure may adversely affect both the central and peripheral nervous systems. In particular, Lilis et al. (Doc. H–004, Ex. 24–10) have demonstrated central nervous system symptoms in 56 percent of workers with blood lead levels below 80 μg/100 ml. The mean blood level was approximately 60 μg/100 ml. This same

study reported symptoms of muscle and joint pain and/or soreness in 39 percent of the workers, many of whom had been exposed for less than a year. The authors cautioned that blood lead levels should not be allowed to exceed 60 μg/100 ml and should be maintained around 40 μg/100 ml. OSHA is of the opinion that the earliest stages of lead-induced central nervous system disease first manifest themselves in the form of behavioral disorders and CNS symptoms. OSHA preliminarily concludes that a blood lead level of 40 μg/100 g must be considered a threshold level for behavioral changes and mild CNS symptoms in adults and that, to protect against long-term neurological effects, blood lead levels should never exceed 50 μg/100 g.

The earliest sign of neurological disease of the peripheral nerves is a slowing of motor nerve conduction velocity (NCV). Slowing of NCV is seen in workers with no obvious clinical signs of lead poisoning. OSHA believes that prevention of this stage is necessary to prevent further development of the disease and its associated forms, which are likely to be irreversible. The work of Catton et al. (Doc. H–004, Ex. 5–15, Ref. 2), Feldman (Doc. H–004, Ex. 24–19, pp. 15), Behse (Doc. H–004, Ex. 24–19, pp. 15), Gerard (Doc. H–004, Ex. 24–19, pp. 15), Guardriglia (Doc. H–004, Ex. 51–B), Araki and Honma (Doc. H–004, Ex. 51–B), Repko et al. (Doc. H–004, Ex. 5–14), and Seppalainen et al. (Doc. H–004, Ex.5–12) all demonstrates statistically significant loss of motor NCV in lead-exposed workers. Seppalainen and his co-workers were able to determine a dose-response relationship between blood lead levels and the slowing of NCV. It is apparent that slowing occurs in workers whose PbB levels are 50 μg/100 g and above. Therefore, a necessary goal of a standard for occupational lead exposure must be to ensure that blood lead levels are maintained below 50 μg/100 g.

Renal system. The research of Wedeen and co-workers, the Health Hazard Evaluation done by NIOSH at Eagle Picher Industries, Inc. (Doc. H–004, Ex. 38C), and the research in secondary smelters done by Lilis, Fishbein, et al. (Doc. H–004, Ex. 23) all demonstrate that lead exposure is a key etiologic agent in the development of kidney disease among occupationally exposed workers. Wedeen et al. (Doc. H–004, Tr. 1765–1766) found that very few workers in a group of lead workers with kidney disease had blood lead levels over 60 μg/100 g. The authors concluded, therefore, that 40 μg/100 g was the upper acceptable PbB limit (Doc. H–004, Tr. 1771). OSHA believes that maintenance of PbB levels at or below 40 μg/100 g will reduce the overall lead dose to the worker, decrease the body burden of lead, and prevent sufficient buildup of lead in the kidney to produce renal damage.

Reproductive effects. Exposure to lead has been associated with adverse reproductive effects in both males and females. In male workers exposed to lead, there is evidence of decreased sexual drive, impotence, decreased ability to produce healthy sperm, and sterility. Lancranjan and co-workers (Doc. H–004, Tr. 577) have demonstrated that the reproductive ability of men occupationally exposed to lead is altered. These authors reported a significant increase in malformed sperm (teratospermia) among workers with mean PbB levels of 74.5 μg/100 ml or 52.8 μg/100 ml. A decreased number of sperm (hypospermia) and decreased sperm motility (athenospermia) were observed not only in the preceding groups but also in those with only slightly increased absorption, i.e., in those with a mean PbB level of 41 μg/100 ml. The authors concluded that these alterations were produced by a direct toxic effect on the male gonads, and that a dose response relationship exists with respect to teratospermia. In OSHA's view, altered spermatogenesis, which reflects impaired reproductive capacity in the male, is a material impairment of health.

Lead may cause genetic damage in the egg or sperm cells before conception, and this damage may be passed on to the developing fetus. The record (Doc. H–004) indicates that genetic damage from lead may occur prior to conception in either males or females. The result of genetic damage could be failure to implant, miscarriage, stillbirth, or birth defects.

There is conclusive evidence that lead passes through the placental barrier. The lead levels in the mother's blood are comparable to concentrations of lead in the umbilical cord blood at birth. The record (Doc. H–004) indicates that, as a result, the exposure of women to lead is associated with abnormal ovarian cycles, premature birth, menstrual disorders, sterility, miscarriage, and stillbirth. Maternal lead poisoning may also result in a short gestational period, low birth weight, postnatal growth retardation, impeded neurobehavioral development, and infant mortality.

There is little direct data on damage to the fetus from exposure to lead but there are extensive studies that demonstrate neurobehavioral effects at blood leads of about 30 μg/100 ml and above in children. OSHA believes that the fetus and newborn are likely to be at least as susceptible to neurological damage as older children and therefore that data on children are also relevant to the fetus. Exposure to lead would be expected to adversely affect heme biosynthesis and the nervous system earliest and most profoundly in the fetus. Damage to the fetus reflects impairment of the reproductive capacity of the parent and is considered by OSHA to constitute a material impairment of functional capacity within the meaning of the Act.

In children, behavioral disturbances such as hyperactivity have been associated with blood lead levels of between 25 and 55 μg/100 ml. Beattie (1975, Doc. H–004, Ex. 6–6) demonstrated an increased probability of mental retardation in children exposed to lead via maternal ingestion of lead in water. Elevated blood lead levels were found in the retarded children when their blood leads were compared with those of children in the control group. There appeared to be a significant relationship between blood lead concentration and mental retardation. The mean blood lead level for the retarded children was 25.5 μg/100 ml.

Motor nerve conduction velocity (NCV) decrements indicating early peripheral neuropathy have also been reported in children. Early studies showed NCV decrements in children whose blood lead levels were 40 μg/100 g and above.

Given the available data, OSHA preliminarily concludes that, to protect the fetus and newborn from the effects of lead on the nervous system, airborne lead levels in workplaces where their maternal parents are employed must be kept below 50 μg/m³. OSHA believes that this evidence overwhelmingly indicates that the lead exposure levels for workers who may wish to have children should be maintained below 50 μg/m³ in order to minimize adverse effects from lead on the worker's reproductive abilities. To minimize the risk of genetic damage, menstrual disorders, interference with sexual function, lowered fertility, difficulties in conception, damage to the fetus during pregnancy, miscarriage, stillbirth, toxic effects on the newborn, and problems with the healthy development of the newborn or developing child, lead exposure levels should be kept below 50 μg/m³ for both male and female workers who are exposed to lead and who may wish to have children. Although there is no evidence for a "no effect" level for lead, OSHA believes

that the risk of reproductive effects would be minimized at this concentration.

OSHA believes that this evidence preliminarily shows that workers exposed to lead suffer material impairments of health at blood lead levels far below those previously considered hazardous. Inhibition of the heme biosynthesis pathway, early stages of peripheral and central nervous system disease, reduced renal function, and adverse reproductive effects are all evidence of adverse health effects from occupational exposure to lead in workers whose blood lead levels are at 40 μg/100 g and above. OSHA has preliminarily concluded that blood lead levels should be maintained at or below 40 μg/100 g and even lower for workers who wish to have children. Therefore, OSHA is proposing an 8-hour TWA PEL for lead of 50 $\mu g/m^3$ to protect workers in the construction, maritime, and agriculture sectors from the significant risk of these adverse health effects. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

METHYL HYDRAZINE

CAS: 60–34–4; Chemical Formula: CH_3NHNH_2

H.S. No. 2112

OSHA's current permissible exposure limit for methyl hydrazine in general industry, construction, and maritime workplaces is 0.2 ppm as a ceiling; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. OSHA has no PEL for methyl hydrazine in agriculture. The ACGIH's current TLV® for this substance is 0.2 ppm as a ceiling, with a skin notation. The ACGIH considers this substance a potential human carcinogen and assigns it an A2 classification, and NIOSH also designates methyl hydrazine a potential occupational carcinogen; the REL for methyl hydrazine of 0.04 ppm as a 2-hour ceiling. OSHA is proposing a PEL of 0.2 ppm as a ceiling, with a skin notation, for methyl hydrazine in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Methyl hydrazine, also called monomethyl hydrazine, is a clear, colorless liquid with an ammonia-like odor. This substance is used in missile propellants and as a solvent and chemical intermediate (ACGIH 1986, p. 398(89)).

Methyl hydrazine causes central nervous system depression, liver damage, and blood dyscrasias in humans and animals. This substance is a carcinogen and teratogen in experimental animals and is also a suspected human carcinogen (ACGIH 1986, p. 398(89)); Proctor, Hughes, and Fischman 1988, p. 334). The oral LD_{50}s in rats, mice, and hamsters are 32 mg/kg, 29 mg/kg, and 22 mg/kg, respectively (RTECS 1990). The LC_{50} for rats is 34 ppm for 4 hours, and for monkeys the LC_{50} is 82 ppm for 1 hour; before death, monkeys showed signs of conjunctival irritation and nausea and then convulsed (RTECS 1990). The dermal LD_{50} in rabbits is 95 mg/kg (RTECS 1990). Acute toxicity in animals poisoned by methyl hydrazine is characterized by convulsions, neurological effects, hypoglycemia, anemia, vomiting, and irritation of the nose and eyes (NIOSH Criteria Document 1978). Undiluted methyl hydrazine that has been applied to the skin of dogs at doses ranging from 14.7 to 264.5 mg/kg was detected in the bloodstream of these animals within 30 seconds (Smith and Clark 1969, in ACGIH 1986, p. 398(89)). Methyl hydrazine caused corneal edema in dogs after it was applied to their skin and carried to the eyes by the bloodstream (Takahashi and Dasher 1969, in ACGIH 1986, p. 398(89)). Dogs exposed to a 21-ppm concentration of dimethyl hydrazine for 4 hours convulsed and died; autopsy revealed pulmonary hemorrhage and edema in these animals. At a 15-ppm concentration, however, the dogs convulsed but survived (Jacobson et al. 1970, in Proctor, Hughes, and Fischman 1988, p. 334). Hemolysis and Heinz bodies in the erythrocytes were seen in monkeys exposed to a 5-ppm concentration of methyl hydrazine for 6 hours/day for 6 months; no effects were observed at lower doses (MacEwen and Haun 1971; Kroe 1971, in ACGIH 1986, p. 398(89)). Pregnant female rats receiving intraperitoneal injections of 2.5, 5.0, or 10 mg/kg of methyl hydrazine on days 6 through 15 of gestation showed a reduction in body weight; embryotoxic effects and an equivocal increase in eye abnormalities were observed in the offspring (Keller et al. 1984, in ACGIH 1986, p. 398(89)). The offspring of rats given intraperitoneal doses of 50 mg/kg methyl hydrazine during days 6 through 15 of pregnancy had an increased incidence of developmental abnormalities of the eyes and ears (RTECS 1990). Mice developed lung tumors and hamsters developed malignant histiocytomas of the liver and cecal tumors when methyl hydrazine was administered to these animals in the drinking water at concentrations of 0.01 percent (Toth 1972, Toth and Shimizu 1972, in ACGIH 1986, p. 398.1(89)). The potential carcinogenicity of methyl hydrazine vapor has also been investigated in several species; studies involving exposures to methyl hydrazine at concentrations ranging from 0.02 ppm to 5 ppm were conducted for 6 hours/day, 5 days/week for 1 year (Kinkeah et al. 1985, in ACGIH 1986, p. 398.1(89)). Rats exposed to methyl hydrazine concentrations of 0.02 ppm and higher had a decrease in their rate of growth, and mice exposed to a 2-ppm concentration developed lung tumors, nasal adenomas, nasal polyps, nasal osteomas, hemangiomas, and liver adenomas and carcinomas (Kinkeah et al. 1985, in ACGIH 1986, p. 398.1(89)). Dogs exposed on this regimen to concentrations of 0.2 ppm and above showed a reversible decrease in red blood cell count, hematocrit, and hemoglobin; at 2 ppm, they exhibited a reversible increase in methemoglobin and signs of liver pathology (Kinkeah et al. 1985, in ACGIH 1986, p. 398(89)). Hamsters in these studies showed lower body weights and an increased incidence of nasal adenomas when exposed to a 5-ppm concentration of methyl hydrazine (Kinkeah 1985, in ACGIH 1986, p. 398.1(89)).

Methyl hydrazine is the strongest convulsant of the methyl-substituted hydrazines (Clayton and Clayton 1981, p. 2292). In high doses, this substance also can cause extensive kidney damage. Liver changes are primarily of the fatty degeneration type and seldom progress to necrosis (Parmeggiani 1983, p. 1069). Methyl hydrazine poisoning can result in tremors, increased central nervous system excitability at high doses, and convulsions that can be fatal (HSDB 1985). Methyl hydrazine causes oxidative damage to human erythrocytes in vitro; effects include Heinz body formation and methemoglobin production (NIOSH Criteria Document 1978). Human volunteers exposed to a 90-ppm concentration of methyl hydrazine for 10 minutes developed eye redness and a tickling sensation in the nose; Heinz body formation in 3 to 5 percent of the volunteers' erythrocytes became apparent on the seventh day after exposure (NIOSH Criteria Document 1978).

Based on this evidence, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing the serious exposure-related effects associated with methyl hydrazine. The Agency believes that establishing a 0.2 ppm ceiling limit, with a skin notation, is necessary to substantially reduce these risks of

material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

4,4'-METHYLENE BIS(2-CHLOROANILINE)

CAS: 101–14–4; Chemical Formula: $CH_2(C_6H_4ClNH_2)_2$

H.S. No. 1273

OSHA has no limit in construction, maritime, or agriculture for 4,4'-methylene bis(2-chloroaniline), or MBOCA, although in 1974, OSHA did issue a standard for MBOCA as part of the Agency's "14 Carcinogens" rulemaking; however, the reviewing court set the MBOCA standard aside on procedural grounds. The ACGIH has a TLV®–TWA of 0.02 ppm (0.22 mg/m³), with a skin notation, and classifies MBOCA as a suspected human carcinogen (A2). NIOSH recommends a TWA limit of 3 μg/m³ for MBOCA, which NIOSH considers a potential occupational carcinogen. OSHA is proposing an 8-hour TWA PEL of 0.02 ppm (0.22 mg/m³) TWA for MBOCA, with a skin notation, in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

MBOCA is a tan-colored solid. It is used as a curing agent for polyurethanes and epoxy resins (Hawley's 1987, p. 767).

MBOCA is highly toxic, causing cyanosis, kidney irritation, methemoglobinemia, and cancer. It is similar in many of its effects to the other aromatic amines (Hosein and van Roosmalen 1978/Ex. 1–1054; Mastromatteo 1965/Ex. 1–146). Steinhoff and Grundmann (1969/Ex. 1–762) demonstrated that feeding MBOCA at unspecified levels to rats on a protein-deficient diet caused a high incidence of liver cancer. Russfield, Homburger, Boger and associates (1975/Ex. 1–929) reported liver and lung tumors in rats fed MBOCA while on a standard diet. Dogs fed MBOCA at a dose of 100 mg/day, 5 days/week showed no hepatic cancer, but malignant nodules in the bladder occurred in a dog fed MBOCA for 9 years (Stula et al. 1977, as cited in ACGIH 1986/Ex. 1–3, p. 392.4).

In industry, reversible hematuria has been reported among MBOCA-exposed workers, but data on the associated exposure levels are lacking (Mastromatteo 1965/Ex. 1–146). An early study of workers exposed for as long as 18 years to MBOCA showed no adverse effects, although the substance and its metabolites were detected in the urine of these subjects (Linch, O'Connor, Barnes et al. 1971/Ex. 1–791). Hosein and van Roosmalen (1978/ Ex. 1–1054) reported an industrial accident in which molten MBOCA was splashed in a worker's face; urinary levels of 3.6 mg/L MBOCA, as well as protein, were detected in the urine, and the subject experienced nausea. However, this worker recovered quickly.

A recent NIOSH retrospective study involving 370 workers employed in a MBOCA-manufacturing plant evaluated the carcinogenicity of this substance, which is structurally similar to benzidine. This study found two cases of bladder cancer in very young workers (less than 30 years of age), both of whom were nonsmokers.

In the prior rulemaking, the Polyurethane Manufacturers Association (PMA) expressed its support for establishing a 0.02-ppm TWA PEL for MBOCA, stating that the proposal "will significantly assist in assuring that any exposure to the chemical is appropriately controlled while imposing a regulation which can be feasibly complied with by employers" (Ex. 3–683, p. 4). The PMA also supported the addition of a skin notation for MBOCA, identifying dermal contact as a "principal potential route for employee exposure" (Ex. 3–683, p. 7).

Other commenters in the earlier rulemaking (Ex. 8–47, 194) urged OSHA to undertake a separate 6(b) rulemaking for MBOCA. OSHA shares these commenters' concerns about MBOCA's toxicity; however, the primary goal of this rulemaking is to achieve consistency in OSHA's PELs across sectors. In the first PEL update, OSHA will evaluate the toxicologic evidence on MBOCA to determine whether the evidence warrants a further reduction in the exposure limit.

Based on this evidence in humans and animals, OSHA is proposing to establish an 8-hour TWA limit of 0.02 ppm, with a skin notation, for MBOCA in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risks of cyanosis, methemoglobinemia, kidney irritation, and bladder cancer, all material health impairments potentially associated with exposure to this substance. A skin notation is being proposed to protect workers against the percutaneous absorption and systemic toxicity demonstrated to occur with this substance in industrial accidents. Promulgation of the proposed limit of 0.02 ppm will also make OSHA's PEL for this substance consistent across all regulated sectors.

MOLYBDENUM, SOLUBLE COMPOUNDS (as Mo)

CAS: 7439–98–7; Chemical Formula: Mo

H.S. No. 2111

OSHA's current permissible exposure limit for the soluble molybdenum compounds in construction and maritime is 5 mg/m³ (measured as molybdenum) as an 8-hour TWA. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA limit of 5 mg/m³ for these substances. There is no NIOSH REL. OSHA is proposing an 8-hour TWA of 5 mg/m³ for the soluble compounds of molybdenum in agriculture. Promulgation of this limit will make the PEL for these substances consistent across all OSHA-regulated sectors.

The soluble compounds of molybdenum include molybdenum trioxide, ammonium molybdate, ammonium paramolybdate, and calcium molybdate, all of which are yellow to white, odorless solids. These substances are used as electrodes, chemical reagents, intermediates in the manufacture of corrosion inhibitors, coloring agents, agricultural chemicals, pigments for paints, lacquers, dyes, and catalysts, and in electroplating and enameling (NIOSH/OSHA Occupational Health Guideline 1981, p. 4; Hawley's 1987, pp. 68, 205–206, 795, and 1065–1066).

Molybdenum's soluble compounds cause eye, nose, and respiratory irritation, central nervous system effects, anemia, and liver and kidney damage in humans and animals. The oral LD_{50} for molybdenum trioxide in rats is 125 mg/kg; the lowest lethal concentration in the same species is 1? g/m³ for 1 hour (RTECS 1990). Ammonium molybdate has an oral LD_{50} of 333 mg/kg in rats (RTECS 1990). There is a subcutaneous LD_{50} of 570 mg/kg for sodium molybdate in mice (RTECS 1990). Guinea pigs and rats fed 1200 to 6000 mg/kg doses of molybdenum trioxide, calcium molybdate, or ammonium molybdate died; there were fewer fatalities among animals fed 120 to 600 mg/kg doses (ACGIH 1986, p. 415). In rats, the inhalation of estimated 100 mg/kg/day doses of molybdenum trioxide by inhalation irritated the eyes and mucous membranes and was lethal (Klaassen, Amdur, and Doull 1986, p. 615). Molybdenum trioxide was extremely irritating to guinea pigs after repeated 1-hour exposures to 250 mg/m³; symptoms included appetite and weight loss, diarrhea, muscular incoordination, and hair loss. Twenty-six of the 51 animals exposed died after the tenth exposure (Fairhall, Dunn, Sharpless, and Pritchard 1951, in Clayton and Clayton 1981, p. 1813). Cattle, rabbits, and chicks fed high levels (dose not specified) of

molybdenum displayed deformities of the joints of the extremities (Ferguson et al. 1943; Maltoni 1973, in Clayton and Clayton 1981, p. 1814). In rats and guinea pigs, ammonium molybdate at oral doses of 1.2 g/kg caused anorexia, colic, trembling, incoordination, and dyspnea; rabbits showed signs of anemia and deformity of the forelegs (Browning 1969, in Proctor, Hughes, and Fischman 1978, p. 359). Repeated oral doses (amount unspecified) induced fatty degeneration of the liver and kidney in rats (Klaassen, Amdur, and Doull 1986, p. 615).

In humans, the lowest toxic concentration of molybdenum trioxide is estimated to be 6 mg/m³ over 4 years; coughing and signs of focal fibrosis were observed in the individual involved in this incident (RTECS 1990). Exposure to molybdenum trioxide causes irritation of the eyes, nose, and throat (HSDB 1990). Anemia, a characteristic symptom of molybdenum intoxication, is manifested by low hemoglobin concentrations and reduced red-cell counts (HSDB 1989). In workers at a Soviet molybdenum-copper plant, chronic exposure led to liver dysfunction and hyperbilirubinemia (Avakyau 1968, in Clayton and Clayton 1981, p. 1814). Gout, with signs and symptoms that included joint pain, articular deformities, erythema, and edema of the joints, has been seen among factory workers and inhabitants of the molybdenum-rich areas of Armenia (Kovalskii 1961; Akopyan 1966, in Clayton and Clayton 1981, p. 1818). Three of 19 workers exposed to molybdenum and molybdenum trioxide at concentrations ranging from 1 to 19 mg/m³ for 4 to 7 years showed evidence of pneumoconiosis (HSDB 1990).

Based on this evidence, OSHA preliminarily concludes that exposure to soluble molybdenum compounds causes eye, nose, and respiratory tract irritation, anemia, and joint pain. OSHA therefore believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed PEL of 5 mg/m³ as an 8-hour TWA limit for soluble molybdenum compounds in agriculture is necessary to significantly reduce the risks of these material health impairments. Promulgation of this limit will also make the PEL for these substances consistent across all OSHA-regulated sectors.

NICKEL, METAL AND INSOLUBLE COMPOUNDS (as Ni)

CAS: 7440–02–0; Chemical Formula: Varies with compound

H.S. No. 2114

In general industry, construction, and maritime, OSHA's current PEL for nickel and its insoluble compounds (measured as nickel) is 1 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 1 mg/m³; NIOSH has a REL of 0.015 mg/m³ (measured as nickel) as a 10-hour TWA; this limit is based on nickel's carcinogenicity. OSHA is proposing an 8-hour TWA PEL of 1 mg/m³ in the agricultural sector for nickel and its insoluble compounds (measured as nickel). This action will make the PEL for these substances consistent across all regulated sectors.

Nickel is a hard, silver-white, magnetic metal. The principal insoluble compounds of nickel in industrial use are nickel oxides, nickel-containing alloys, nickel hydroxide, nickel sulfide, nickel carbonate, and nickel phosphate (ACGIH 1986, p. 422; AIHA 1978). Metallic nickel is used in the manufacture of stainless steel, various other alloys, and in electroplating.

Contact of the skin with metallic nickel and some of its compounds causes dermal sensitization, and exposure to nickel subsulfides or nickel oxide has been determined by the International Agency for Research on Cancer to cause cancer in humans (IARC 1982, Suppl. 4, pp. 167–170). Nickel is also carcinogenic in experimental animals. The lowest lethal dose of nickel in rats is 5 g/kg (RTECS 1990). Rats exposed to a 1 mg/m³ concentration of nickel sulfide for 14 months developed a significant excess of lung cancer (Hueper 1958, in AMA Arch. Pathol. 65:600). Exposing hamsters to nickel oxide at a concentration of 53 mg/m³ caused pneumoconiosis described by the authors of the study as "massive" (Wehner, Busch, Olson, and Craig 1975, in Am. Ind. Hyg. Assoc. J. 36:801). Rats and mice were exposed to nickel subsulfide 6 hours/day for 12 days at concentrations ranging from 0.7 to 13.5 mg/m³ (measured as nickel); mice and rats developed degeneration of the epithelium of the respiratory tract at the 0.9 mg/m³ Ni exposure, and the rats additionally developed pulmonary inflammation (National Academy of Sciences 1975, in ACGIH 1986, p. 422(89)). Nickel oxide exposure (at a nickel concentration of 0.112 mg/m³) for 12 hours/day, 6 days/week for 2 weeks caused increases in alveolar macrophages and thickened alveolar walls (Bingham, Barkley, Zerwas, et al. 1972, in Arch. Environ. Hlth. 25:406). Nickel subsulfide exposure at a concentration of 0.97 mg/m³ nickel for 78 weeks caused lung cancers in 14 of 208 rats (Ottolenghi, Haseman, Payne, et al. 1958, in J. National Cancer Inst. 54(5):1165). A recent in-vitro study of the morphological and neoplastic transformation induced by insoluble nickel compounds in mouse embryo cells showed that these substances induced type II foci, some of which were tumorigenic, in these cells (Miura, Patierno, Sakuramoto, and Landolph 1984, in Environ. Mol. Mutagen. 14(2):65–78). Reproductive effects in the form of testicular degeneration were seen in mice and rats exposed to the subsulfide by inhalation at a nickel concentration of 0.9 mg/m³ (Benson, Carpenter, Hahn et al. 1987, in Fund. Appl. Toxicol. 9(2):251).

Many nickel compounds cause dermal sensitivity; the prevalence of nickel sensitivity in the general population is estimated at 2.5 to 5.0 percent (Peltonen 1979, in Contact Dermatitis 5:27). A recent review (Christensen 1990, in Dermatol. Clin. 8(1):37–40), however, reports that the prevalence of nickel sensitivity among women in the general population may range as high as 10 percent. The symptoms of "nickel itch" begin with itching and may progress to skin lesions, discrete ulcers, and eczema (Fisher 1973, Contact Dermatitis; Phila: Lea & Febiger). Several reports of asthma among nickel-exposed workers also appear in the literature (Tolot, Bordeur, Neulat in Arch. Mal. Prof. Med. Travail. Secur. Soc. 1956:18, p. 291; McConnell, Fink, Schlueter, Schmidt et al. 1973, Ann. Intern. Med 78:888). A recent review of the toxicity of nickel compounds in humans (Coogan, Lalta, Snow, and Costa 1989, in Crit. Rev. Toxicol. 19(4):341–384) addresses the systemic and molecular toxicology, as well as the carcinogenicity, of these substances. This paper notes that the major target organs of systemic toxicity are the kidney and the immune system. Nickel subsulfide roasting in nickel refining is generally accepted as being a carcinogenic process on the basis of studies in refinery workers (Grandjean et al. 1988; Agency for Toxic Substances and Disease Registry 1987). Nickel-exposed workers in many other industries (electroplating, alloy manufacture) have also shown a statistically significant increase among these workers (Grandjean et al. 1988, ATSDR 1987). Based on this evidence in humans, the International Agency for Research on Cancer, NIOSH, and the ACGIH have all concluded that nickel is a probable or confirmed human carcinogen. The mechanisms of nickel carcinogenesis are reviewed in a 1989 article by Sunderman (Scand. J. Work. Environ. 15(1):1–12); this author hypothesizes that differences in the carcinogenic activities of nickel

compounds may reflect variations in their capacities to provide nickel ions at critical sites within target cells.

Based on this evidence, OSHA is proposing a limit of 1 mg/m³ as an 8-hour TWA for nickel and its insoluble compounds (measured as nickel) in agricultural operations. The Agency preliminarily concludes that this limit is necessary to protect workers in agriculture from the significant risk of material health impairments in the form of nickel-induced cancer and skin and pulmonary sensitization. In addition, promulgation of this PEL will make OSHA's limit for these substances consistent across all regulated sectors.

NICOTINE

CAS: 54–11–5; Chemical Formula: $C_{10}H_{14}N_2$

H.S. No. 2115

OSHA's current permissible exposure limit for nicotine in general industry, construction, and maritime workplaces is an 8-hour TWA of 0.5 mg/m³; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. OSHA has no PEL for nicotine in agriculture. The ACGIH's current TLV®–TWA for this substance is 0.5 mg/m³, with a skin notation. NIOSH has no REL for nicotine but concurs (Ex. 8–47) with the limit being proposed. OSHA is proposing a TWA PEL of 0.5 mg/m³, with a skin notation, for nicotine in agriculture. Promulgation of this limit will make the PEL for nicotine consistent across all OSHA-regulated sectors.

Nicotine is a thick, colorless, pale yellow oil that turns brown on exposure to air and has a slight fishy odor when warm. Nicotine is used as an insecticide and fumigant. It is also used in human medicine and in tanning (ACGIH 1986, p. 427). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Nicotine causes stimulation, followed by depression, of the central and autonomic nervous systems in humans and animals. Nicotine also causes fetotoxic, developmental, and reproductive effects (NIOSH/OSHA Occupational Health Guideline 1981, p. 4; Proctor, Hughes, and Fischman 1988, p. 363). The oral LD_{50} in rats is 50 mg/kg; in mice it is 3.34 mg/kg (RTECS 1990). The dermal LD_{50} in rabbits is 50 mg/kg (RTECS 1990). Nicotine exerts its effect by transiently stimulating, and then severely depressing, the central nervous system; death is due to respiratory paralysis (HSDB 1988). Dogs showed signs of restlessness, apnea followed by hyperpnea, and a rapid decrease in the strength of respiration terminating in apnea (Franke and Thomas 1933, in Hayes 1982, p. 86). The effects of nicotine poisoning in dogs include fluctuations in blood pressure, convulsions, respiratory failure, and increase in heart rate; these symptoms can result in death (Franke and Thomas 1933, in Hayes 1982, p. 86). Nicotine was teratogenic in mice receiving injections of 25 mg/kg on days 9 through 11 of gestation; skeletal malformations and cleft palates were observed (National Research Council 1977, in HSDB 1988). Deformities were also found in the fetuses of rabbits administered 20 mg/kg of nicotine five times during pregnancy (Crowe 1972, in HSDB 1988). Administered to inbred Fischer and Buffalo rats, nicotine resulted in greatly reduced fertility (Riesenfeld and Oliva 1987, in HSDB 1988).

In small doses, nicotine causes symptoms of vomiting, dizziness, tachycardia, hypertension, sweating, and salivation in humans (Baselt 1980, p. 201). Symptoms of toxicity have been observed in workers harvesting tobacco, probably as a result of dermal absorption of nicotine (Gehlbach et al. 1975, in Baselt 1980, p. 201). Nicotine poisoning in humans is similar to that in animals; signs of poisoning include respiratory failure and circulatory collapse (Beeman and Hunter 1937, in Hayes 1982, p. 90). The estimated minimal lethal dose of nicotine in humans is 40 to 60 mg; this dose causes prostration, convulsions, respiratory paralysis, and death after ingestion (Baselt and Cravey 1977, in Baselt 1980, p. 201). Nicotine splashed in a patient's eye caused severe pain, conjunctival irritation, and corneal infiltration (Villard 1927, in Grant 1974, p. 747). Intravenous injection of 1.0 mg of nicotine alkaloid caused approximately the same increase in blood pressure and heart rate and decrease in skin temperature of the fingers and toes as did smoking a cigarette (Moddock and Collier 1933, in Hayes 1982, p. 90). Nicotine crosses the placental barrier and has been found in the amniotic fluid of smokers (Mosier and Jansons 1972; Van Vunakis et al. 1974, in Clayton and Clayton 1981, p. 2813).

Based on this evidence in humans and animals, OSHA preliminarily concludes that nicotine causes central and autonomic nervous system effects and affects reproduction and fertility. In the absence of a limit, OSHA therefore preliminarily finds that workers in agriculture are at significant risk of experiencing these effects. The Agency believes that establishing an 8-hour TWA PEL of 0.5 mg/m³, with a skin notation, will substantially reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PERCHLOROMETHYL MERCAPTAN

CAS: 594–42–3; Chemical Formula: CCl_3SCl

H.S. No. 2125

In general industry, construction, and maritime, OSHA's current permissible exposure limit for perchloromethyl mercaptan is 0.1 ppm as an 8-hour TWA. There is no limit for this substance in agriculture. The ACGIH has a TLV®–TWA of 0.1 ppm. NIOSH has no REL for this substance but concurs (Ex. 8–47) with the limit being proposed. OSHA is proposing a PEL of 0.1 ppm as an 8-hour TWA for perchloromethyl mercaptan in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Perchloromethyl mercaptan is a pale yellow, oily liquid with a disagreeable odor. This substance is used in the production of fungicides, as a fumigant, as a dye intermediate, and in organic synthesis (ACGIH 1986, p. 466; Proctor, Hughes, and Fischman 1988, p. 401). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Perchloromethyl mercaptan is a severe eye, mucous membrane, and pulmonary irritant in humans and animals; accidental overexposure has caused liver and kidney damage and death in humans. The oral LD_{50} in rats is 83 mg/kg (RTECS 1990). In mice, the LC_{50} is 296 mg/m³ (37 ppm) for 2 hours (RTECS 1990), and the dermal LD_{50} in rabbits is 1410 mg/kg (Vernot et al. 1977, in ACGIH 1986, p. 466.1(88)). Rabbits showed signs of severe eye irritation after application of 0.05 mg; applied to the skin, 560 mg also caused severe irritation in these animals (RTECS 1990). Cats and mice exposed to perchloromethyl mercaptan for 15 minutes at a concentration of 45 ppm developed pulmonary edema and died within 1 to 2 days (Althoff 1973, in Proctor, Hughes, and Fischman 1988, p. 401; ACGIH 1986, p. 466). Rats exposed to the vapors of perchloromethyl mercaptan for 6 hours/day, 5 days/week for 2 weeks at concentrations of 0.13, 1.0, or 8.7 mg/m³ (0.01, 0.1, or 1.1 ppm) had labored breathing, tremors, nasal irritation, and pulmonary edema at the highest concentration; no symptoms or effects were observed in rats exposed to lower concentrations (Knapp et al. (no

date), in ACGIH 1986, p. 466.2(88)). Male rats exposed to perchloromethyl mercaptan vapors at a concentration of 2 ppm for 6 hours/day, 5 days/week for 4 weeks exhibited signs of respiratory distress; at autopsy, lung congestion was observed. Exposure to a 0.5-ppm concentration on the same regimen caused no adverse effects (Gage 1970, in ACGIH 1986, p. 466.1(88)).

Exposure to 10 mg/m^3 (1.3 ppm) perchloromethyl mercaptan causes eye irritation in humans (Althoff 1973, in Proctor, Hughes, and Fischman 1988, p. 401). Acute exposures cause the following signs and symptoms: coughing, dyspnea, lacrimation, pallor, vomiting, tachycardia, convulsions, and cyanosis; death due to lung edema also may occur (Althoff 1973; Ruth 1986; Spacilova 1971, in ACGIH 1986, p. 466.1(88)). Three chemical workers were accidentally overexposed to an unspecified concentration of perchloromethyl mercaptan vapors; two survived subsequent episodes of pulmonary edema, but the third died 36 hours later. At autopsy, necrotizing tracheitis, massive hemorrhagic pulmonary edema, toxic nephrosis, and vacuolization of centrilobular hepatic cells were seen (Althoff 1973, in Proctor, Hughes, and Fischman 1988, p. 401).

Based on this evidence in humans and animals, OSHA preliminarily concludes that perchloromethyl mercaptan causes severe eye and pulmonary irritation and liver and kidney damage. In the absence of a limit, OSHA believes that workers in agriculture are at significant risk of experiencing these effects, which are material health impairments. The Agency preliminarily finds that the proposed PEL of 0.1 ppm as an 8-hour TWA is necessary to substantially reduce these significant risks. This action would also make the PEL for perchloromethyl mercaptan the same for workplaces in all OSHA-regulated sectors.

PHENOL

CAS: 108–95–2; Chemical Formula: C_6H_5OH

H.S. No. 2126

OSHA's current permissible exposure limit for phenol in general industry, construction, and maritime is 5 ppm as an 8-hour TWA; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit in agriculture. The ACGIH has a TLV®–TWA limit of 5 ppm, with a skin notation, for phenol. NIOSH has RELs for phenol of 5.2 ppm as a 10-hour TWA and 15.6 ppm as a 15-minute ceiling. OSHA is proposing an 8-hour TWA PEL of 5 ppm, with a skin notation, for phenol in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Phenol consists of white or clear crystals that turn pinkish or reddish in color on exposure to air or light. This substance is used as a disinfectant, in germicidal paints and slimicides, and in the manufacture of phenolic resins, bis-phenol-A, caprolactam, and a variety of other chemicals and drugs (ACGIH 1986, p. 469; Hawley's 1987, p. 897). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Phenol causes irritation of the eyes, mucous membranes, and skin and central nervous system effects in humans and animals. Phenol is also corrosive in contact with the skin or eyes. In rats and mice, oral LD_{50} values are 317 mg/kg and 270 mg/kg, respectively, and the LC_{50}s in rats and mice are 316 mg/kg and 177 mg/kg, respectively (RTECS 1990). The dermal LD_{50} in rabbits is 850 mg/kg; in rats it is 669 mg/kg (RTECS 1990). In contact with the skin of rabbits, 500 mg of phenol caused severe irritation; instilled into the eyes of rabbits, 5 mg also caused a severe degree of irritation (RTECS 1990). In a study by Pullin et al. (1978, in ACGIH 1986, p. 469), swine were given skin applications of 500 mg/kg over 35 to 40 percent of the body area for 1.0 and 2.5 minute periods to study the percutaneous absorption effects of phenol. The phenol was absorbed rapidly through the skin and caused twitching and tremors within 3 to 5 minutes. After 5 minutes, signs of excessive salivation, nasal discharge, and respiratory distress were observed; 1.75 hours after exposure, animals were found to have a peak plasma phenol concentration of 52.6 ppm (Pullin et al. 1978, in ACGIH 1986, p. 469). Guinea pigs exposed to phenol concentrations of 25 to 50 ppm for 20 days died; autopsy revealed severe injury of the heart, lungs, liver, and kidneys (Deichmann 1944, in ACGIH 1986, p. 469). Mice treated weekly for 42 weeks by application of one drop of phenol in benzene to shaved skin developed papillomas (5 of 14 mice) after 52 weeks (NIOSH Criteria Document 1976). Although phenol is considered noncarcinogenic in rats or mice, increased incidences of leukemia and lymphoma were detected in male rats given 2500 or 5000 ppm in their drinking water for 103 weeks (National Cancer Institute 1980, in Proctor, Hughes, and Fischman 1988, p. 404).

The lowest lethal oral dose in adult humans is 14 mg/kg (RTECS 1990). Brief industrial exposures to phenol concentrations of 48 ppm (in combination with 8 ppm formaldehyde) caused marked eye, nose, and throat irritation (NIOSH Criteria Document 1976). The signs and symptoms of human overexposure to phenol include loss of appetite, darkened urine, muscle pain, rapid and weak pulse, difficult breathing, cyanosis, convulsions, and coma; death may occur if the overexposure is severe (Merliss 1972; Stajduhar-Caric 1968, in Rom 1983, p. 528). Phenol is highly corrosive to the skin and causes a whitening of the contacted area (Key, Henschel, Butler et al. 1977, in Rom 1983, p. 528). A recent NIOSH study (O'Malley, Mathias, Priddy, Molina, Grote, and Halperin 1988, J. Occup. Med. 30(6):512–516) reports that workers handling rubber that contained phenol developed chemically induced vitiligo (skin depigmentation). In contact with the eyes, phenol causes severe damage and may lead to permanent loss of vision (Grant 1986, p. 720). Ingestion of as little as 10 g causes severe mouth and throat burns, tremor, convulsions, and muscle twitching (NIOSH Criteria Document 1976; U.S. EPA Summary 1986; in Proctor, Hughes, and Fischman 1988, p. 404). A laboratory technician exposed to vapors and to liquid phenol spilled on the skin experienced symptoms of anorexia, weight loss, weakness, muscle pain, and dark urine; after brief improvement, the patient suffered a worsening of symptoms, including tenderness and enlargement of the liver (Merliss 1972, in Proctor, Hughes, and Fischman 1988, p. 404). A 32-year old male who spilled a strong solution of phenol over his upper body died 10 minutes later; the victim suffered coagulation necrosis of the skin and left eye, acute dermatitis veneta, acute passive congestion of the lungs, liver, spleen, and kidneys, and "acute phenol toxicosis" (Gottlieb and Storey 1936, in ACGIH 1986, p. 470).

Based on this evidence in humans and animals, OSHA preliminarily concludes that phenol causes eye, skin, and mucous membrane irritation and central nervous system effects in exposed workers. This substance is also corrosive on contact with the skin or eyes and is rapidly absorbed through the skin. The Agency preliminarily finds that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. OSHA believes that establishing a PEL of 5 ppm as an 8-hour TWA, and a skin notation, is necessary to

substantially reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PHENYLHYDRAZINE

CAS: 100–63–0; Chemical Formula: $C_6H_5NHNH_2$

H.S. No. 1317

OSHA's current limit for phenylhydrazine in construction and maritime is 5 ppm as an 8-hour TWA, with a skin notation. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 5 ppm and a TLV®–STEL of 10 ppm, and a skin notation, for this substance. The ACGIH considers this substance a potential human carcinogen and assigns it an A2 classification. NIOSH considers this substance a potential occupational carcinogen and (1978e/Ex. 1–263) recommends that workplace exposures not exceed 0.14 ppm, measured over a 2-hour period. In construction and maritime, OSHA is proposing to retain the 8-hour TWA PEL of 5 ppm and the skin notation and to add a STEL of 10 ppm; OSHA is also proposing to extend these limits to agriculture. These are the limits recently established for phenylhydrazine in general industry.

Phenylhydrazine may take the form either of yellow crystals or an oily liquid that darkens on exposure to air and light. It is used as a reagent in analytical chemistry and in organic synthesis. The hydrochloride is a strong reducing agent (Hawley's 1987, p. 903).

No data are available on the effects of phenylhydrazine resulting from inhalation. OSHA's limits are based on the high acute toxicity of the compound when administered orally or subcutaneously to animals; single doses on the order of 20 mg/kg have resulted in the death of dogs within 22 days (Hesse, Franke, and Hering 1935/Ex. 1–785) and produced a marked decrease in erythrocyte count in rodents (von Oettingen and Deichmann-Greubler 1936/Ex. 1–771). Anemia and hemolysis are the characteristic responses seen in animals fed or injected with phenylhydrazine.

In its criteria document on the hydrazines, NIOSH (1978e/Ex. 1–263) reviewed four studies on the carcinogenicity of phenylhydrazine in mice. One study (Toth and Shimizu 1976/Ex. 1–675) found significant increases in blood vessel tumors. Another study (Clayson, Biancifiori, Milia, and Santilli 1966, as cited in ACGIH 1986/Ex. 1–3, p. 477) reported increased incidences of lung adenomas and adenocarcinomas. Two other studies (Roe, Grant, and Millican 1967/Ex. 1–659; Kelly, O'Gara, Yancy et al. 1969/Ex. 1–703) were negative. NIOSH concluded that phenylhydrazine should be considered a potential human carcinogen and recommended that exposures not exceed 0.14 ppm over a 2-hour sampling period, which represents the lowest level that can be detected reliably. The ACGIH (1986/Ex. 1–3) has placed phenylhydrazine on its A2 (suspected human carcinogens) list.

In the prior rulemaking, several commenters (Exs. 8–47; Tr. 3–97 to 3–98, 116, 195, Tr. 9–218, 8–16) were of the opinion that a lower PEL was warranted for phenylhydrazine. OSHA is aware of the developing literature on phenylhydrazine. However, the primary objective of the present rulemaking is to ensure consistency in OSHA's limits across sectors. At the time of the first PEL update, OSHA will evaluate the toxicologic literature on phenylhydrazine to determine whether there is a need at that time for a further reduction in the occupational exposure limit.

However, at the present time, OSHA is retaining the 5-ppm 8-hour TWA limit and skin notation and is proposing to add a 10-ppm STEL for phenylhydrazine in construction and maritime; OSHA is also proposing to extend these limits to agriculture. The Agency preliminarily concludes that these limits will reduce the significant health risks associated with exposure to this substance among workers in these sectors. These risks include acute blood-related toxicity and may also include cancer; these effects clearly constitute material impairments of health. Promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

PHENYLPHOSPHINE

CAS: 638–21–1; Chemical Formula: $C_6H_5PH_2$

H.S. No. 1318

OSHA has no PEL for phenylphosphine in construction, maritime, or agriculture; NIOSH also has no REL for this substance. The ACGIH has a TLV®-ceiling of 0.05 ppm for phenylphosphine. The proposed PEL is a ceiling of 0.05 ppm; NIOSH concurred (Ex. 8–47, Table N1) with this limit when it was recently established in general industry.

Phenylphosphine is a crystalline solid that is used in catalysts and antioxidants (Sittig 1985, p. 712).

A 90-day inhalation study conducted by the du Pont Company, in which rats and beagle dogs were exposed to average concentrations of 0.6 ppm or 2.2 ppm phenylphosphine for 6 hours per day, 5 days per week, showed that rats exposed to 2.2 ppm had significant hematologic changes and testicular degeneration (E.I. du Pont de Nemours & Co., Inc. 1970, as cited in ACGIH 1986/Ex. 1–3, p. 479). These effects were not noted among rats exposed to 0.6 ppm, but rats exposed at the lower level did show hypersensitivity to sound and touch and mild hyperemia. The dogs tolerated the higher exposure level better than the rats in that some regeneration of testicular damage occurred in dogs during a 1-month recovery period. Dogs exposed to 0.6 ppm exhibited intermittent nausea, diarrhea, lacrimation, and hind leg tremor (ACGIH 1986/Ex. 1–3). The ACGIH considered 0.6 ppm to be an NOE level for severe effects in animals and recommended a 0.05-ppm ceiling TLV® to provide a tenfold safety margin to protect workers against the changes exhibited by the test animals at the 0.6-ppm level.

OSHA preliminarily concludes that workers in construction, maritime, and agriculture who are exposed to uncontrolled levels of phenylphosphine are at significant risk of experiencing the nausea, irritation, and CNS effects found to be associated with such exposures. OSHA preliminarily finds that these effects constitute material health impairments. The Agency believes that the proposed ceiling of 0.05 ppm for phenylphosphine in construction, maritime, and agriculture will reduce these significant risks substantially. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PHOSPHINE

CAS: 7803–51–2; Chemical Formula: PH_3

H.S. No. 1321

OSHA currently has a PEL of 0.3 ppm TWA for phosphine in construction and maritime. There is no limit in agriculture. The ACGIH recommends a TLV®–TWA of 0.3 ppm and a TLV®–STEL of 1 ppm. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limits being proposed. OSHA is retaining the 8-hour TWA limit of 0.3 ppm and proposing to add a 1 ppm STEL for phosphine in construction and maritime; the Agency is also proposing to extend these limits to agriculture. These are the limits recently established for this substance in general industry.

Phosphine is a colorless gas with a disagreeable, garlic-like odor. Phosphine is used primarily as a fumigant. It is also used in chemical synthesis and as a doping agent for electrical components (ACGIH 1986, p. 482). When used in pesticidal applications and in

accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Early studies reported that laboratory animals could tolerate phosphine in 4-hour daily exposures of 5 ppm for 2 months, but fatalities were observed from seven similar exposures at 10 ppm (Muller 1940/Ex. 1–819). In 1975, Waritz and Brown (Ex. 1–451) reported a 4-hour LC_{50} of 11 ppm in rats; these lethal exposures caused effects typical of respiratory irritation.

Prior to 1958, numerous cases of phosphine-related occupational poisonings and deaths were reported, including a fatality caused by pulmonary edema that was attributed to an exposure of 8 ppm for 2 hours daily (Harger and Spolyar 1958/Ex. 1–327). Sublethal symptoms (without chronic effects) occurred at phosphine exposures averaging 10 ppm or less, with excursions of up to 35 ppm; recorded symptoms included diarrhea, nausea, vomiting, respiratory distress, and dizziness (Jones, Jones, and Longley 1964/Ex. 1–420). The literature contains no documented reports of chronic poisoning caused by prolonged exposure to phosphine, although several authorities have asserted that this is a possibility (Henderson and Haggard 1943e/Ex. 1–1066; Fairhall 1957h, as cited in ACGIH 1986/Ex. 1–3, p. 883; Johnstone and Miller 1960/Ex. 1–1114; Patty 1963f, as cited in ACGIH 1986/Ex. 1–3, p. 883; American Industrial Hygiene Association (AIHA) 1964/Ex. 1–407).

In the earlier rulemaking, Joel Carr, Health and Safety Research Director for the American Federation of Grain Millers Union, testified on the toxicology of and employee exposures to phosphine in grain elevators and flour mills (Ex. 8–1; Tr. pp. 7–240 to 7–259). Mr. Carr described a report of a group of industrial hygiene studies published by NIOSH (Zaebst 1986; Zaebst, Blade, Morelli-Schroth et al. 1987; Zaebst, Blade, Burroughs et al. 1988), in which applicators of phosphine were found to be exposed above the proposed TWA PEL and STEL; nonapplicator workers also become exposed while working near fumigated grain, while loading or transferring fumigated grain, or while working in elevators and mills. OSHA believes that workers handling treated grain in agricultural settings and in marine terminal and longshoring operations would be likely to have exposures similar to those described by Mr. Carr.

Based on this evidence, OSHA is retaining its 8-hour TWA PEL of 0.3 ppm and proposing to add a 15-minute STEL of 1 ppm for phosphine in the construction and maritime sectors and is additionally proposing to extend these limits to agriculture. The Agency preliminarily concludes that both of the proposed limits are necessary to substantially reduce the significant risk of lung damage, diarrhea, and nausea, all material health impairments associated with elevated short-term and long-term exposure to this toxic gas. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

PHOSPHORUS, YELLOW

CAS: 7723–14–0; Chemical Formula: P_4

H.S. No. 2130

OSHA's permissible exposure limit for yellow phosphorus in general industry, construction, and maritime is 0.1 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 0.1 mg/m³ for yellow phosphorus. NIOSH has no REL for this substance. OSHA is proposing an 8-hour TWA limit of 0.1 mg/m³ for yellow phosphorus in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Yellow phosphorus is a yellow, soft, waxy, crystalline solid that darkens on exposure to light and has a distinct garlic-like odor. It is used in the manufacture of rat poisons, fertilizers, and fireworks, in gas analysis, and to produce smoke screens (ACGIH 1986, p. 484; Gosselin, Smith, and Hodge 1984, p. III–348; Proctor, Hughes, and Fischman 1988, p. 412). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Yellow phosphorus causes eye, skin, and respiratory tract irritation and burns, bone changes, and kidney and liver injury in humans and animals. The oral LD_{50} in rats is 3030 mg/kg, and in mice it is 4820 mg/kg (RTECS 1991). Inhalation of phosphorus vapors at concentrations of greater than 20 ppm for 7 hours/day, 5 days/week caused severe respiratory irritation and a high mortality rate in rats as a result of lung edema and bronchopneumonia (ACGIH 1986, p. 484). Subcutaneous injection of rats with 0.05 mg/kg of yellow phosphorus per day caused the development of bone changes; 0.8 mg/kg/day resulted in an increased death rate (Fleming et al. 1942, in J. Ind. Hyg. Tox. 24:154). Dogs subcutaneously injected with 0.2 to 0.4 mg/kg/day yellow phosphorus died within a few days (Buchanan et al. 1954, in Arch. Ind. Hyg. Occup. Med. 9:1). Experimental animals exposed to 13 to 16 ppm of phosphorus for 7 hours a day, 5 days a week for 4 months showed no difference in growth patterns as compared with controls (unpublished TVA Report 1947, cited in ACGIH 1986, p. 484). Repeated doses of 0.1 mg/kg/day of yellow phosphorus caused chronic poisoning and liver damage in dogs (Buchanan et al. 1954, in Arch. Ind. Hyg. Occup. Med. 9:1).

In humans, yellow phosphorus has caused death from a single 1-mg/kg dose (Smyth 1956, Am. Ind. Hyg. Assoc. Q. 17:129), and as little as 0.2 mg/kg can produce adverse effects (Diaz-Rivera et al. 1950, in Medicine 29:269, as cited in Clayton and Clayton 1981, p. 2122). Yellow phosphorus fumes are irritating to the respiratory tract and eyes; exposure causes severe irritation, with blepharospasm, photophobia, and lacrimation (Scherling and Blondi's 1945, in Mil. Surg. 96:70–78, as cited in Grant 1986, p. 735). Yellow phosphorus causes deep, painful burns of the skin, producing a firm eschar surrounded by vesiculation (Summerlin et al. 1967, in J. Trauma 7:476–484, as cited in Proctor, Hughes, and Fischman 1988, p. 412). Signs and symptoms of yellow phosphorus intoxication include abdominal pain, jaundice, and a garlic odor of the breath (Proctor, Hughes, and Fischman 1988, p. 412). Severe acute yellow atrophy of the liver is a delayed symptom of yellow phosphorus poisoning that may prove fatal (Klaassen, Amdur, and Doull 1986, p. 567). A Russian study (Beloskurskaia, Aitbembetov, Balmakhaeva, Korolćhuk 1989, in Vrach Delo 7:104–106) of 600 yellow phosphorus production workers showed that the liver toxicity associated with chronic phosphorus poisoning was a result of impaired fat metabolism. Chronic poisoning from yellow phosphorus can cause anemia, cachexia, and necrosis of the bone, and particularly of facial bones, including the maxilla and mandible (Proctor, Hughes, and Fischman 1988, p. 412). Phosphorus necrosis of the jaw ("phossy jaw") was common among match workers early in this century and has more recently been reported among chemical and fireworks industry workers (Heimann 1946, in J. Ind. Hyg. Tox. 28:142; Hughes et al. 1962, in Brit. J. Ind. Med. 19:83; Nomura 1956, in J. Sci. Labour (Japan) p. 109). Phossy jaw is the result of the oral absorption of small quantities of phosphorus over a long period of time. A recent Japanese study (Horiguchi, Endo, Nakano, Shinagawa, and Harima 1988, in Sumitomo Bull. Ind. Hlth. 24:27–32) reports several cases of phosphorus necrosis among workers exposed before 1974 to phosphorus in

phosphoric acid, phosphoric acid fertilizer, and yellow phosphorus production plants.

Based on this evidence in humans and animals, OSHA preliminarily concludes that yellow phosphorus causes eye, skin, and respiratory tract irritation, bone degeneration, and kidney and liver injury in exposed workers. OSHA therefore believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency preliminarily finds that a limit of 0.1 mg/m³ as an 8-hour TWA is necessary to significantly reduce these risks of material health impairment. In addition, promulgation of this limit will make the PEL for yellow phosphorus consistent across all OSHA-regulated sectors.

PIPERAZINE DIHYDROCHLORIDE

CAS: 142–64–3; Chemical Formula: $C_4H_{10}N_2 \cdot 2HCl$

H.S. No. 1330

OSHA has no limit for piperazine dihydrochloride in construction, maritime, or agriculture. The ACGIH recommends a TLV®–TWA limit of 5 mg/m³. There is no NIOSH REL; however, NIOSH concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing a 5 mg/m³ 8-hour TWA limit for this substance in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Piperazine dihydrochloride is a white, water-soluble solid in the form of needles. This substance is used in the manufacture of insecticides, pharmaceuticals, and fibers (Hawley's 1987, p. 921). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

This substance has low systemic toxicity and mild irritant properties; the compound is biologically active. The oral LD_{50} for rats has been reported as 4.9 g/kg (NIOSH 1984, as cited in ACGIH 1986/Ex. 1–3, p. 491). Eye and skin irritation have been reported as a result of human exposures to high (not further specified) levels of piperazine dihydrochloride; subjects experienced mild to moderate skin burns and sensitization. Inhalation of the dust has been associated with asthmatic reactions (Dow Chemical Company 1977h, as cited in ACGIH 1986/Ex. 1–3, p. 491).

OSHA is proposing to establish a limit of 5 mg/m³ as an 8-hour TWA for piperazine dihydrochloride in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit is necessary to reduce the significant risks of sensitization and eye and skin irritation, material health impairments that are potentially associated with exposure to this substance at levels above the proposed limit. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

N-PROPYL NITRATE

CAS: 627–13–4; Chemical Formula: $CH_3CH_2CH_2ONO_2$

H.S. No. 1340

OSHA currently has an 8-hour TWA limit of 25 ppm for n-propyl nitrate in the construction and maritime industries. There is no limit in agriculture. The ACGIH has a 25-ppm TLV®–TWA and a 15-minute TLV®–STEL of 40 ppm. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limits being proposed. OSHA is retaining the 25 ppm 8-hour TWA limit and proposing to add a 40 ppm STEL for this substance in construction and maritime; the Agency is also proposing to extend these limits to agriculture. These are the limits recently established for this substance in general industry.

N-Propyl nitrate is a pale yellow liquid with a sickly sweet odor. It is used as an intermediate in chemical synthesis and as a fuel ignition promoter (ACGIH 1986, p. 505).

Rats inhaling propyl nitrate vapor for 4 hours at a concentration of 10,000 ppm exhibited cyanosis and methemoglobinemia before they died (Hood 1953, as cited in ACGIH 1986/Ex. 1–3, p. 505). The intravenous LD_{50} in unanesthetized rabbits has been reported to be between 200 and 250 mg/kg; in anesthetized dogs and cats, intravenous doses of between 100 and 200 mg/kg were usually fatal (Murtha, Stabile, and Wills 1956/Ex. 1–649). The authors of these studies (1956/Ex. 1–649) concluded that n-propyl nitrate exerts a direct action on the vascular smooth muscle and that the ensuing cardiac effects and respiratory depression contribute to the compound's hypotensive action (Murtha, Stabile, and Wills 1956/Ex. 1–649). Inhalation trials in mice, rats, hamsters, guinea pigs, and dogs have established 4-hour LC_{50} values ranging from 9000 to 10,000 ppm for rats, 6000 to 7000 ppm for mice, and 2000 to 2500 ppm for dogs. Dogs survived repeated exposures (6 hours/day, 5 days/week) at 260 ppm for 6 months, although slight clinical signs were observed during the first 2 weeks of exposure (Rinehart, Garbers, Greene, and Stoufer 1958/Ex. 1–524). The percutaneous toxicity of n-propyl nitrate is low, but this substance may cause inflammation and thickening of the skin after repeated exposures; these effects are sometimes transient (ACGIH 1986/Ex. 1–3, p. 505).

In construction and maritime, OSHA is retaining the PEL of 25 ppm as an 8-hour TWA and proposing to add a STEL of 40 ppm for n-propyl nitrate; the Agency is also proposing to extend these limits to agriculture. The Agency preliminarily concludes that these limits will protect workers in these sectors against the significant risk of cyanosis, methemoglobinemia, and hypotension, all material health impairments that are potentially associated with exposure to n-propyl nitrate. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

PYRETHRUM

CAS: 8003–34–7; Chemical Formula: $C_{21}H_{28}O_3$ or $C_{22}H_{28}O_5$

H.S. No. 2136

In general industry, construction, and maritime, OSHA's current permissible exposure limit for pyrethrum is 5 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 5 mg/m³ for pyrethrum. NIOSH has no REL for this substance but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA limit of 5 mg/m³ for pyrethrum in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Pyrethrum is an insecticide extracted from chrysanthemums; it is a viscous brown resin or solid composed of six active principles: Pyrethrins I and II, cinerins I and II, and jasmolin I and II. Pyrethrum is used as a botanical insecticide (ACGIH 1986, p. 506; New Jersey Fact Sheet 1986, p. 1). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Pyrethrum causes dermatitis, sensitization, and central nervous system effects in humans and animals. The oral LD_{50}s in rats and mice are 200 mg/kg and 370 mg/kg, respectively (RTECS 1990). The dermal LD_{50} in rats is greater than 1800 mg/kg (ACGIH 1986, p. 506). An intravenous pyrethrum dose of 5 mg/kg caused behavioral symptoms and convulsions in rats (RTECS 1990). The acute effects of pyrethrum poisoning in animals include excitation, convulsions, tetanic paralysis, and muscular fibrillation (Chevalier and Ripert 1927, in Hayes 1982, p. 76). Death is usually due to respiratory failure (Chevalier 1930, Shimkin and Anderson

1936, Leonard 1942, Chevalier and Ripert 1927, in Hayes 1982, p. 76). Rats exposed once to 6000 mg/m³ of pyrethrum in peanut oil for 30 minutes showed moderate lung congestion at autopsy (Carpenter, Weil, Pozzani, and Smyth 1950, in ACGIH 1986, p. 506). A pyrethrum concentration of 16 mg/m³ inhaled by rats and dogs for 30-minute periods over 31 days caused only slight lung irritation (Carpenter, Weil, Pozzani, and Smyth 1950, in ACGIH 1986, p. 506). Dogs showed signs of tremor, ataxia, labored respiration, and excess salivation when fed pyrethrins at a dietary level of 5000 ppm for 90 days (Griffin 1973, in Hayes 1982, p. 77). The only significant effects seen in male and female rats fed dietary levels of pyrethrin (equivalent to 10, 50, or 250 mg/kg) for 2 years was slight but definite liver damage in animals exposed at the higher dose levels (Lehman 1965, in Hayes 1982, p. 77). Rats given daily gavage doses of 50, 100, or 150 mg/kg on days 6 to 15 of pregnancy had a higher incidence of resorptions compared with controls, and rats fed a dietary pyrethrum level of 5000 ppm beginning 3 weeks before first mating had weanlings whose weights were significantly lower than those of controls (Khera et al. 1982, in Proctor, Hughes, and Fischman 1988, p. 430; Griffin 1973, in Hayes 1982, p. 78).

The most common effect of exposure to pyrethrum in humans is dermatitis, which is usually manifested as a mild erythematous dermatitis with vesicles, papules in moist areas, and intense pruritus; a bullous dermatitis may also develop (Hayes 1963, in Proctor, Hughes, and Fischman 1988, p. 430). The lowest lethal oral dose of pyrethrum in humans is 1 g/kg (RTECS 1990). In pure form, pyrethrum constituents pyrethrin I and II are reported to be irritating to the eyes and mucous membranes (Grant 1986, p. 774). In a study of workers processing pyrethrum powder, 30 percent were found to have erythema, skin roughening, and pruritus; these effects cleared after cessation of exposure (Casida 1973, in Proctor, Hughes, and Fischman 1988, p. 430). A sensitization reaction has been reported in a worker exposed to pyrethrum dust; the signs and symptoms associated with this episode included reddening, burning, itching, and swelling of the cheeks, eyes, and face and severe pruritus. These effects disappeared within 2 days after the cessation of exposure (Casida 1973, in Proctor, Hughes, and Fischman 1988, p. 430). The symptoms of sensitivity to pyrethrum may resemble those seen in pollinosis: sneezing, nasal discharge, and nasal stuffiness (Feinberg 1934; Ramirez 1930, in Hayes 1982, p. 79).

Based on this evidence, OSHA preliminarily concludes that pyrethrum causes dermatitis, skin sensitization, and eye and nose irritation in exposed workers. OSHA therefore believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that establishing a PEL of 5 mg/m³ as an 8-hour TWA for pyrethrum in agriculture is necessary to significantly reduce these risks of material health impairment. In addition, promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

PYRIDINE

CAS: 110–86–1; Chemical Formula: C_5H_5N

H.S. No. 2137

In general industry, construction, and maritime, OSHA's current permissible exposure limit for pyridine is 5 ppm as an 8-hour TWA. There is no limit for this substance in agriculture. The ACGIH has a TLV®–TWA of 5 ppm for pyridine. NIOSH has no REL for this substance but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA limit of 5 ppm for pyridine in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Pyridine is a flammable, colorless to yellow liquid with a strong and disagreeable odor. Pyridine is used as a fungicide, in the synthesis of vitamins and drugs, as a solvent, an alcohol and antifreeze denaturant, and a dyeing assist (ACGIH 1986, p. 507; Hawley's 1987, p. 982). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Pyridine causes eye, skin, and mucous membrane irritation, central nervous system effects, bone marrow changes, and liver and kidney damage in humans and animals. The oral LD_{50}s in rats and mice are 891 mg/kg and 1500 mg/kg, respectively (RTECS 1990). The dermal LD_{50} in rabbits is 1121 mg/kg (RTECS 1990). The mean lethal concentration in rats is 4000 ppm for 4 hours (RTECS 1990). Severe eye irritation (graded 7 on an ascending severity scale of 1 to 10) developed in rabbits when 2 mg of pyridine was put into their eyes; in contact with the skin for 24 hours, pyridine caused mild irritation in rabbits (RTECS 1990). In animals, the major effects from exposure to large doses of pyridine by any route include irritation and narcosis; liver and kidney damage occur after repeated feedings (Clayton and Clayton 1981, p. 2728).

The vapor of pyridine causes eye and nasal irritation, and prolonged or repeated contact of pyridine with the skin results in skin irritation (ACGIH 1986, p. 507; Proctor, Hughes, and Fischman 1988, p. 431). Exposure to 10 ppm pyridine caused a mild degree of irritation in some volunteers (Smyth 1956). A 29-year-old woman developed symptoms of nervous system inhibition, speech disorders, and diffuse cortical affliction after breathing pyridine vapors at an unspecified concentration for 15 to 20 minutes; symptoms were not detected until 10 hours after exposure (Kuzelova et al. 1975, in HSDB 1988). Symptoms of nausea, headache, insomnia, nervousness, low back pain, and frequent urination have occurred in workers exposed to concentrations of pyridine averaging 125 ppm for 4 hours/day for 1 to 2 weeks (Reinhardt 1981, in ACGIH 1986, p. 507). An epileptic patient being treated with 1.8 to 2.5 ml pyridine daily for 2 months developed serious liver and kidney damage (Pollack et al. 1943, in Proctor, Hughes, and Fischman 1988, pp. 430–431). The most important effects of chronic pyridine inhalation are on the liver, kidneys, and bone marrow (Smyth 1956, in ACGIH 1986, p. 507). Chronic exposure of chemical plant workers to pyridine concentrations ranging from 6 to 12 ppm caused symptoms of central nervous system depression such as headache, vertigo, nausea, and vomiting (Teisinger 1948, in Proctor, Hughes, and Fischman 1988, p. 430).

Based on this evidence in humans and animals, OSHA preliminarily concludes that pyridine causes irritation, central nervous system effects, and liver and kidney damage. OSHA believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these exposure-related effects. The Agency preliminarily concludes that establishing a PEL of 5 ppm as an 8-hour TWA for pyridine in agriculture is necessary to substantially reduce the risk of these material health impairments. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

SELENIUM AND COMPOUNDS (as Se)

CAS: 7782–49–2; Chemical Formula: Se

H.S. No. 2140

In construction and maritime, OSHA's permissible exposure limit for selenium and its compounds (measured as selenium) is 0.2 mg/m³ as an 8-hour TWA. There is no limit in agriculture.

The ACGIH has a TLV®–TWA of 0.2 mg/m³ for selenium and compounds. NIOSH has no REL. OSHA is proposing an 8-hour TWA PEL of 0.2 mg/m³ for selenium and its compounds in agriculture. Promulgation of this limit will make the PEL for these substances consistent across all OSHA-regulated sectors.

Selenium is a non-metallic dark red to blush black solid; it may also take the form of dark red, gray, or black crystals. Selenium and its compounds are used as insecticides and in the manufacture of glass, pigments, xerography, rectifiers, steel, photography, catalysts, and rubber; selenium compounds are also used to treat various human and animal diseases (ACGIH 1986, p. 517; Clayton and Clayton 1981, p. 2130). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Selenium and its compounds cause eye, skin, and mucous membrane irritation, central nervous system effects, gastrointestinal disturbances, and loss of hair and fingernails. The oral LD_{50} in rats is 6700 mg/m³; rats given this dose showed somnolence, dyspnea, and nutritional and metabolic changes (RTECS 1990). The lowest lethal inhalation dose in rats is 33 mg/m³ for 8 hours, at which level hemorrhage, emphysema, and acute pulmonary edema were seen (RTECS 1990). Of the selenium compounds, sodium selenate has the lowest oral LD_{50}, which is 2.3 mg/kg in rabbits (ACGIH 1986, p. 517). Selenium sulfide has an oral LD_{50} of 370 mg/kg in mice (ACGIH 1986, p. 517). Animals exposed to selenium anhydride at a concentration of 150 mg/m³ for 4 hours showed signs of conjunctivitis, pulmonary edema, and convulsions prior to death; degenerative changes in the liver, kidneys, spleen, and heart were seen at autopsy (NIOSH/OSHA Occupational Health Guideline 1981, p. 2). Animals fed diets containing 5 to 15 ppm of selenium developed hepatic necrosis, hemorrhage, and cirrhosis; some species also developed marked progressive anemia (National Research Council 1980, in Proctor, Hughes, and Fischman 1988, p. 439). Chronic exposure can cause a condition called "alkali disease," which is characterized by emaciation, lack of vitality, loss of hair, and deformities of the bones that cause pain and lameness; animals may also be anemic, have congestive heart failure, cirrhosis of the liver, scarring of the kidneys, and joint erosion of the long bones (Radeleff 1964, in Hayes 1982, p. 54). Eleven of 53 rats developed liver-cell adenomas or low-grade carcinomas; 43 of 53 rats developed cirrhotic livers, and four rats developed advanced adenomatoid hyperplasia after surviving on diets of 5, 7, or 10 mg selenium per kg of diet for 18 to 24 months (Nelson et al. 1943, in IARC 1975, Vol. 9, p. 251). Rats fed selenium at concentrations of 4, 6, 8, or 16 mg/kg of diet died of toxic hepatitis within 100 days (Harr et al. 1967, in IARC 1975, Vol. 9, p. 252). Selenium also causes reproductive effects in mice and premature death in young mice and rats (Schroeder and Mitchener 1971b, in IARC 1975, Vol. 9, p. 253).

The lowest toxic dermal dose for a selenium compound in humans is 2 mg/kg for selenium oxychloride (RTECS 1990). Acute industrial exposure to selenium causes symptoms of mucous membrane irritation, sneezing, coughing, eye redness, difficult breathing, frontal headache, and dyspnea in exposed workers (Clinton 1947, in Clayton and Clayton 1981, p. 2131). Garlic breath is a classic symptom of selenium exposure (Rom 1983, p. 499). Selenium dioxide splashed into the eye can cause the development of a pink allergic-type reaction of the eyelids; there may also be conjunctivitis of the palpebral conjunctiva (Glover 1970, in Clayton and Clayton 1981, p. 2131). After being accidentally sprayed in the eyes with selenium dioxide, a chemist experienced burns of the skin, eye irritation, blurred vision, and dulling of the cornea; the eyes had returned to normal 16 days later (Middleton 1947, in Proctor, Hughes, and Fischman 1988, p. 438). Symptoms of mucous membrane irritation, pulmonary edema, bronchitis, and bronchial pneumonia occurred after an accidental poisoning from hydrogen selenate (Olson 1986, in Proctor, Hughes, and Fischman 1988, p. 437). Workers exposed to unspecified concentrations of selenium oxide developed bronchospasm and dyspnea; after 12 hours, they developed metal fume fever, with symptoms such as chills, fever, and headache. A few of these workers developed bronchitis and pneumonitis, but all of the workers were asymptomatic within a week (Wilson 1962, in Proctor, Hughes, and Fischman 1988, p. 438). In China, a disease that was characterized by symptoms of hair and nail loss, skin lesions, nervous system abnormalities, paralysis, and hemiplegia was attributed to chronic selenium poisoning (Yang et al. 1983, in PH&F 1988, p. 438). Unaffected people had an average daily selenium intake of 0.1 mg, while six sick individuals had an average daily intake of 5.0 mg; there were no reports of disabling chronic disease or death from these exposures (Yang 1983, in Proctor, Hughes, and Fischman 1988, p. 438). In a plant producing selenium rectifiers, 35 of 62 workers reported experiencing various symptoms including headache; nine workers were found to have conjunctivitis and slight tracheobronchitis (Kinningkeit 1962, in ACGIH 1986, p. 517). A group of 200 to 300 workers in a similar plant experienced symptoms of garlic odor of breath, skin rashes, indigestion, metallic taste, and socio-psychological effects; urinary selenium concentrations among these workers ranged from 0.25 mg/ml to 0.43 mg/ml, and air concentrations ranged from 0.2 mg/m³ to as high as 3.6 mg/m³ (Glover 1970; Glover 1967, in ACGIH 1986, p. 517). According to the International Agency for Research on Cancer, there is inadequate evidence to evaluate the carcinogenicity of selenium and its compounds in humans and animals (IARC 1975, Vol. 9).

Based on this evidence, OSHA preliminarily concludes that exposure to selenium and its compounds causes irritation of the eyes, skin, and mucous membranes, central nervous system effects, gastrointestinal disturbances, and liver and kidney effects. OSHA preliminarily finds that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed limit for selenium and compounds of 0.2 mg/m³ as an 8-hour TWA is necessary to significantly reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for these substances consistent across all regulated sectors.

SILVER, METAL AND SOLUBLE COMPOUNDS (as Ag)

CAS: 7440–22–4; Chemical Formula: Ag (metal); varies (compounds)

H.S. No. 2143

In general industry, construction, and maritime, OSHA's permissible exposure limit for silver metal and the soluble silver compounds (measured as silver), is 0.01 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 0.01 mg/m³ for these substances. NIOSH has no REL for these substances but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing to establish an 8-hour TWA limit of 0.01 mg/m³ for silver metal and the soluble silver compounds in agriculture. Promulgation of this limit will make the PEL for these substances consistent across all OSHA-regulated sectors.

Silver is a hard, brilliant white, lustrous, ductile metal. Silver nitrate is a colorless, odorless solid that may turn gray, and silver fluoride takes the form of a yellow to white, odorless solid. The principal soluble compounds of silver are silver nitrate, silver chloride, silver fluoride, and silver sulfide. Soluble silver compounds are used in photographic films, indelible inks, silver salts, medicines, and hair dyes and are also used to silver mirrors, plate silver, and catalyze ethylene oxide. These compounds are also used as pesticides, laboratory reagents, and as substitutes in organic compounds (Hawley's 1987, p. 1043; ACGIH 1986, p. 529). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In humans, industrial exposure to the dust or fume of metallic silver or to the mists of soluble silver compounds is associated with the development of argyria, a bluish-grey discoloration of the eyes, mucous membranes, or skin; argyria is caused by the impregnation of the tissues with silver. In addition, several of the soluble silver compounds, such as silver nitrate, cause eye, nose, and upper respiratory tract irritation on acute exposure and kidney, liver, and lung damage on chronic exposure (Klaassen, Amdur, and Doull 1986, p. 625). The acute toxicity of all of the soluble silver compounds is high, regardless of the route of administration (Clayton and Clayton 1981, p. 1886). The oral LD_{50} for silver nitrate in mice is 50 mg/kg; in guinea pigs, the oral LD_{50} for silver fluoride is 300 mg/kg (Clayton and Clayton 1981, p. 1886). Administered orally to experimental animals in repeated doses, metallic silver caused pigmentation of the eyes (Grant 1986, p. 815). Instilled into the eyes of rabbits, a 6-percent solution of silver nitrate caused scarring and a 12-percent solution caused blindness (HSDB 1989). Oral dosing with silver nitrate caused vascular hypertension in experimental animals; at autopsy, kidney changes were seen in these animals (HSDB 1989). Intravenous administration of silver nitrate caused hemolysis and death in animals of several species (HSDB 1989).

In humans, overexposure to the dust or fume of silver metal or to the soluble compounds of silver causes argyria, evidenced as a bluish-gray discoloration of the eyes, mucous membranes, or skin. Argyria may be either localized or systemic. Localized argyria is caused by the embedding of silver particles in the skin or by the absorption of silver salts through the sweat glands (Clayton and Clayton 1981, p. 1887). If the dust, fume, or mist of the soluble compounds of silver is inhaled in sufficient quantities, argyria of the upper respiratory tract may occur. Two workers engaged in the manufacture of silver nitrate developed argyria of the respiratory tract, which was associated with mild chronic bronchitis (Browning 1969, pp. 296–301, in Proctor, Hughes, and Fischman 1988, p. 442). Generalized argyria is manifested as widespread pigmentation of the skin, eyes, and nails (Clayton and Clayton 1981, p. 1887–1888). A worker "massively" overexposed to the heated vapor of metallic silver for 4 hours subsequently developed pulmonary edema and lung damage (Forycki et al., Bull. Inst. Maritime Tropical Medicine in Gydnia 34:199–202, 1963). Twelve cases of argyria were reported in silver workers exposed to concentrations of 1 to 2 mg/m³ of silver during varnish spraying (Jindrichova 1963, in ACGIH 1986, p. 529). Eleven of these workers had upper respiratory tract argyrosis and nine showed argyria of the conjunctiva or cornea (Jindrichova 1963, in ACGIH 1986, p. 529). Four cases of eye and skin argyrosis also were reported in silver polishers (Perrone et al. 1977, in ACGIH 1986, p. 529).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to silver metal and the soluble silver compounds causes eye, skin, and mucous membrane irritation, as well as respiratory, gastrointestinal, central nervous system, liver, and kidney effects. OSHA therefore believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse exposure-related effects. The Agency believes that the proposed TWA limit of 0.01 mg/m³ for silver and its soluble compounds (measured as silver) is necessary to substantially reduce the significant risk that workers in agriculture will experience these material health impairments. Promulgation of this limit will also make the PEL for silver and its soluble compounds consistent across all regulated sectors.

SODIUM FLUOROACETATE

CAS: 62–74–8; Chemical Formula: $CH_2FCOONa$

H.S. No. 1366

In construction and maritime, the current OSHA standard for sodium fluoroacetate is 0.05 mg/m³ as an 8-hour TWA, with a skin notation. There is no limit in agriculture. The ACGIH has exposure limits of 0.05 mg/m³ TLV®–TWA and 0.15 mg/m³ TLV®–STEL, with a skin notation. In construction and maritime, OSHA is retaining its 8-hour TWA PEL and skin notation and is proposing to add a STEL of 0.15 mg/m³; the Agency is also proposing to extend these limits to agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limits being proposed, which were recently established for this substance in general industry.

Sodium fluoroacetate is a fine white powder, which is sometimes dyed black for commercial use. It is used as a rodenticide (ACGIH 1986, p. 534). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Sodium fluoroacetate causes vomiting, convulsions, and ventricular fibrillation. It is highly toxic by inhalation, ingestion, or via absorption through the skin (NIOSH/OSHA 1981). The ACGIH calculated and set the threshold limit of 0.05 mg/m³ based on studies of rats indicating an oral LD_{50} of 1.7 mg/kg (Lehman 1951/Ex. 1–790). Tissue changes in rats were noted in a later study by the same author in which the animals were fed 0.25 mg sodium fluoroacetate/kg/day (Lehman 1952, as cited in ACGIH 1986/Ex. 1–3, p. 534); the equivalent level in humans would be 17 mg/person/day. A further study by Miller and Phillips (1955, as cited in ACGIH 1986/Ex. 1–3, p. 534) examined growth rates in rats fed various dosages of sodium fluoroacetate. Rats who received 10 ppm in their diet experienced a transient fluctuation in growth rate. At 20 ppm (approximately 2 mg/kg in young rats), the growth rate declined markedly the first week; the rats survived and resumed growth at the normal rate in 3 to 4 weeks. Tolerance for the chemical lasted less than 2 weeks, and those rats who had adjusted to sodium fluoroacetate showed a second retardation of growth when returned to a dietary level of 20 ppm after a 2-week interval of eating a normal diet. Miller and Phillips (1955, as cited in ACGIH 1986/Ex. 1–3, p. 534) noted that rats conditioned to a dietary level of 20 ppm were then able to adjust to a level of 40 ppm (a dose that is greater than the single LD_{50} dose per day).

Based on this evidence, OSHA is retaining the 8-hour TWA limit of 0.05 mg/m³ and the skin notation and is proposing a STEL of 0.15 mg/m³ for sodium fluoroacetate in construction and maritime; the Agency is also proposing these limits in agriculture. OSHA preliminarily concludes that the 8-hour TWA and short-term exposure limits, with a skin notation, will reduce the risk of systemic effects posed to workers in these sectors who are

exposed to sodium fluoroacetate. OSHA considers these effects material health impairments. Promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

STRYCHNINE

CAS: 57–24–9; Chemical Formula: $C_{21}H_{22}N_2O_2$

H.S. No. 2145

OSHA's PEL for strychnine in general industry, construction, and maritime is 0.15 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The 1987–1988 ACGIH TLV®–TWA is also 0.15 mg/m³. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed for strychnine. OSHA is proposing an 8-hour TWA of 0.15 mg/m³ for strychnine in agriculture. Promulgation of this limit would make OSHA's PEL for this substance consistent across all industry sectors.

Strychnine is an odorless, colorless or white crystalline solid (HSDB 1989). This substance is used to control rodents in agriculture and also finds limited use in medicine (ACGIH 1986, p. 538). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Strychnine is a powerful convulsant in both humans and animals. The oral LD_{50} in rats is 2350 μg/kg, and the oral LD_{50} in dogs is 500 μg/kg (RTECS 1990). Acutely poisoned animals develop severe convulsions and paralysis before death (RTECS 1990). Strychnine's stimulant properties act principally on the spinal cord, but the medullary centers of the brain are also involved (Hayes 1982, p. 98).

Strychnine's effects on humans are similar to those in animals. The mean lethal dose in humans is estimated to lie between 100 and 120 mg (or 1.5 to 2 mg/kg body weight). However, a total dose as small as 16 mg has killed an adult (Gosselin, Smith, and Hodge 1984, p. III–375). Doses of 5 to 7 mg cause muscle tightening and twitching; the neck, jaws, and little fingers are particularly affected (Hayes 1982, pp. 96–101). After ingestion, any stimulus (light, sound, touch) is likely to set off a violent convulsion (Franz 1985). As many as 10 to 15 convulsions may occur before death, although cardiac or respiratory arrest may occur between the second and third convulsions (Hayes 1982, pp. 96–101).

Based on this evidence in humans and animals, OSHA preliminarily concludes that workers in agriculture are at significant risk of experiencing strychnine's convulsant effects in the absence of an OSHA PEL. Accordingly, OSHA is proposing a PEL of 0.15 mg/m³ as an 8-hour TWA for strychnine in agriculture. The Agency believes that this limit is necessary to substantially reduce the significant risk of these adverse health effects, which constitute material health impairments. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

TELLURIUM AND COMPOUNDS (as Te)

CAS: 13494–80–9; Chemical Formula: Te

H.S. No. 2150

In general industry, construction, and maritime, OSHA's permissible exposure limit for tellurium and its compounds (measured as tellurium) is 0.1 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 0.1 mg/m³ for tellurium and its compounds. NIOSH has no REL for these substances but concurs (Ex. 8–47) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 0.1 mg/m³ for tellurium and compounds (measured as tellurium) in agriculture. Promulgation of this limit will make the PEL for these substances consistent across all OSHA-regulated sectors.

Tellurium is a non-metallic silvery-white lustrous solid. This substance is used as an additive for copper, iron, and steel, in the vulcanization of rubber, as a coloring agent in ceramics, and as an ingredient in thermoelectric devices and in storage batteries. Some telluride compounds are used as semiconductors (ACGIH 1986, p. 555; Hawley's 1987, p. 1124). Examples of tellurium compounds are hydrogen telluride, potassium tellurite, and sodium tellurate. (Tellurium hexafluoride has a separate PEL.)

Tellurium causes garlic odor of the breath, central nervous system effects, and kidney and liver damage in humans; in animals, this substance causes neurotoxic and teratogenic effects. The oral LD_{50}s for tellurium in rats, mice, rabbits, and guinea pigs are 83 mg/kg, 20 mg/kg, 67 mg/kg, and 45 mg/kg, respectively (RTECS 1990). Acute exposure to tellurium can cause restlessness, tremor, diminished reflexes, paralysis, convulsions, somnolence, coma, and death (Cooper 1971, in Proctor, Hughes, and Fischman 1988, p. 458). Inhalation of hydrogen telluride has produced pulmonary irritation and hemolysis of red blood cells in animals (AIHA Hygienic Guide Series 1964; Cerwenka and Cooper 1961, in Proctor, Hughes, and Fischman 1988, p. 458). A dietary concentration of 1500 ppm had only a slight effect on the growth in rats (DeMeio 1946, in ACGIH 1986, p. 555). Chronic tellurium poisoning (by subcutaneous or intramuscular injection) caused degenerative changes in the retinal ganglion cells and brain of cats after 3 months (Pentschew 1958, in HSDB 1989). Tellurium poisoning (route, dose, and duration not specified) caused encephalopathy and changes in the retinal ganglion cells, optic nerves, and optic tract (Grant 1986, p. 887). Given 3300 ppm in the diet for 5 months, rats showed a markedly impaired ability to learn sequential behavioral tasks (Dru et al. 1972, in HSDB 1989). Rats chronically exposed to high doses of tellurium dioxide had decreased growth and developed necrosis of the liver and kidney (Cerwenka and Cooper 1961; Browning 1969, in Klaassen, Amdur, and Doull 1986, p. 625). Pregnant rats fed 300 to 500 ppm tellurium had a high incidence of hydroencephalic offspring (Duckett 1970, in Proctor, Hughes, and Fischman 1988, p. 458). Pregnant rats and rabbits fed a diet containing 3000 or 15,000 ppm (rats) or 5250 ppm (rabbits) tellurium during pregnancy give birth to offspring with skeletal and soft-tissue malformations (Johnson, Christian, Hoberman, DeMurco, Kilpper, and Mermelstein 1989, in Fund. Appl. Toxicol. 11(4):691–702). Weanling rats fed 10,000 ppm tellurium developed segmental demyelination; however, remyelination occurred even after continued exposure to tellurium (Lampert, Garro, and Pentschew 1970, in Proctor, Hughes, and Fischman 1988, p. 458). Neonatal rats exposed to tellurium in mothers' milk from birth until sacrifice at 7, 14, 21, or 28 days of age showed Schwann cell and myelin degeneration in the sciatic nerves and hypomyelination of the optic nerves at autopsy (Jackson, Hammurg, Worth, and Duncan 1989, in Acta. Neuropathol. Berlin 78(3):301–309).

No serious cases of industrial poisoning caused by tellurium have been reported in humans. A garlic odor of the breath and sweat, dryness of the mouth, a metallic taste in the mouth, somnolence, anorexia, and nausea were reported among iron foundry workers exposed to tellurium concentrations between 0.01 and 0.1 mg/m³ for 22 months; somnolence and metallic taste did not occur until these workers' urinary tellurium levels were at least 0.01 mg/l (Steinberg, Massari, Miner, and Rink 1942; Cerwenka and Cooper 1961, in AIHA Hygienic Guide Series 1964). Mild neurologic effects have occurred in workers exposed to tellurium salts during industrial manufacturing processes (Klaassen, Amdur, and Doull 1986, p. 374). In workers in an electrolytic lead refinery,

exposure to tellurium dioxide resulted in skin lesions in the form of scaly, itching patches, and loss of sweat function (Browning 1969, in Proctor, Hughes, and Fischman 1988, p. 458). Accidental administration of about 2 gm of sodium tellurite caused death in two adults within 6 hours. The signs of toxicity were vomiting, renal pain, stupor, loss of consciousness, irregular breathing, and cyanosis; autopsy revealed fatty degeneration of the liver (Gosselin 1984, p. II–129).

Based on this evidence in humans and animals, OSHA preliminarily concludes that tellurium and its compounds impart a garlic odor to the breath and sweat and cause central nervous system effects, cardiac effects, and kidney and liver changes. OSHA preliminary finds that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. Accordingly, the Agency proposes to establish an 8-hour TWA limit of 0.1 mg/m³ for tellurium and its compounds in agriculture. OSHA believes that the proposed limit is necessary to substantially reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for these substances consistent across all regulated sectors.

TETRAETHYL LEAD (as Pb)
CAS: 78–00–2; Chemical Formula: $(C_2H_5)_4Pb$
H.S. No. 1386

OSHA's 8-hour TWA limit for tetraethyl lead (TEL) in construction and maritime is 0.1 mg/m³, measured as lead, with a skin notation. There is no limit in agriculture. The ACGIH recommends that worker exposures to TEL not exceed 0.1 mg/m³ TWA; the ACGIH also recommends a skin notation. NIOSH has no REL, but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA limit of 0.075 mg/m³, and a skin notation, for tetraethyl lead in construction, maritime, and agriculture. This is the limit recently established for TEL in general industry.

Tetraethyl lead is a colorless liquid that may be dyed red, orange, or blue and that has a slightly musty odor. It is used as an anti-knock additive in gasoline (ACGIH 1986, p. 562).

Tetraethyl lead is a central nervous system toxin in humans and animals. The oral LD_{50} in rats is 12.3 mg/kg, and the LC_{50} in the same species is 850 mg/m³ for 1 hour (RTECS 1990). The lowest lethal dermal dose in rabbits is 830 mg/kg (RTECS 1990). Instilled into the eyes of rabbits, TEL caused redness, blepharospasm, and discharge, but these effects were reversible (Grant 1986, pp. 890–893). Acutely poisoned animals showed tremors, irritability, and spasticity after a single dose of approximately 17 mg/kg; repeated doses at one-tenth this level, however, caused behavioral changes, peripheral hyperemia, cardiac hypertrophy, brain edema, and changes in the liver, pancreas, thyroid, lung, and thymus (IARC 1973, Vol. 2, p. 156). Administered to rhesus monkeys orally (at a dose of 6 mg/kg/day) for 6 months, TEL caused no clinical manifestations of toxicity, although elevated blood and tissue levels of lead were seen in these animals (Heywood, R. et al. 1979, Toxicol Letter 4(2): 119–125). Carcinogenicity bioassays have been negative.

In humans, TEL poisoning causes psychosis, convulsions, and if the exposure is sufficiently severe, death. More than 150 deaths have been attributed to TEL poisoning, although many of these have involved accidental or suicidal ingestion rather than occupational exposure. However, several workers have died while cleaning leaded gasoline storage tanks without proper personal protective equipment (Proctor, Hughes, and Fischman 1988, p. 461). TEL poisoning can occur after a single one-hour exposure to 100 mg/m³ or after repeated exposures to lower levels, and the signs and symptoms may be so subtle as to go undetected (Kehoe, in Parmeggiani 1983, pp. 1197–1199). The signs and symptoms of TEL poisoning include headache, fatigue, weakness, nausea, vomiting, diarrhea, and anorexia and may progress to ataxia, tremor, bradycardia, and hypothermia (Grandjean 1984). Chronic exposure to TEL causes cumulative liver, kidney, and central nervous system damage (Cralley and Cralley 1985, p. 182).

Based on this evidence in humans and animals, OSHA preliminarily concludes that workers in construction, maritime, and agriculture exposed to this substance are at significant risk of experiencing central nervous system damage as well as liver and kidney damage. The Agency believes that the proposed PEL of 0.075 mg/m³ is necessary to substantially reduce this significant risk. OSHA is therefore proposing a PEL of 0.075 mg/m³ as an 8-hour TWA (measured as Pb), and a skin notation, for tetraethyl lead in construction, maritime, and agriculture. Promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

TETRAMETHYL LEAD (as Pb)
CAS: 75–74–1; Chemical Formula: $(CH_3)_4Pb$
H.S. No. 1388

The OSHA limit for tetramethyl lead (TML) in construction and maritime is 0.15 mg/m³ (measured as lead) as an 8-hour TWA, with a skin notation. The ACGIH has a TLV®–TWA of 0.15 mg/m³, with a skin notation, for this substance. There is no limit in agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA of 0.075 mg/m³, with a skin notation, for this substance in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Tetramethyl lead is a colorless liquid, which may be dyed blue, orange, or red in commercial use; it has a slight musty odor. It is used as an anti-knock additive in gasoline (ACGIH 1986, p. 565).

Tetramethyl lead is toxic to the central nervous system of humans and animals. The oral LD_{50} in rats is 90 mg/kg, the LC_{50} in mice is 8500 mg/m³ for 30 minutes, and the lowest lethal dermal dose in rabbits is 3391 mg/kg (RTECS 1990). Acutely poisoned animals develop tremors, hyperexcitability, and convulsions before death (Schepers 1964). If inhaled, TML has only one-tenth the toxicity of tetraethyl lead in rats; however, in dogs and mice, TML is more toxic than TEL (Cremer and Calloway 1961). Rats exposed repeatedly to 10.8 mg/kg developed behavioral changes and peripheral hyperemia; at autopsy, cardiac hypertrophy, hyperemia, and edema of the brain and lungs were seen (IARC 1973, Vol. 2, p. 157). Monkeys given 6 mg/kg/day intravenous doses of TML showed gastrointestinal disturbances, tremor, abnormal reflexes, and depressed brain cholinesterase levels (Heywood et al. 1978). Carcinogenicity bioassays with TML have been negative. No teratogenic effects were seen in the offspring of rats given 40, 80, 112, or 160 mg/kg TML by oral administration (McClain and Becker 1972, in IARC 1980, Vol. 23, p. 370).

In humans, several cases of acute toxicity have been reported; the signs and symptoms were indicative of encephalopathy (IARC 1973, Vol. 2, p. 158). In humans, TEL is believed to be three times as toxic as TML (IARC 1973, Vol. 2, p. 158). However, there are few data on the toxicity of TML in humans.

OSHA preliminarily concludes, based on this evidence in humans and animals, that workers in construction, maritime, and agriculture are at significant risk of experiencing lead poisoning in the form of encephalopathy. The Agency believes that the proposed PEL of 0.075 mg/m³ is

necessary to substantially reduce this risk among these workers. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

TETRAMETHYL SUCCINONITRILE
CAS: 3333–52–6; Chemical Formula: $C_6H_{12}N_2$
H.S. No.: 2156

The OSHA limit for tetramethyl succinonitrile in general industry, construction, and maritime is 0.5 ppm as an 8-hour TWA, with a skin notation. The ACGIH TLV®–TWA is also 0.5 ppm as an 8-hour TWA, with a skin notation. NIOSH has a REL of 1 ppm as a 15-minute ceiling but concurs with the limit being proposed (Ex. 8–47, Table N3A). OSHA is proposing a PEL of 0.5 ppm as an 8-hour TWA, with a skin notation, in agriculture, an action that would make the PEL for this substance consistent across all OSHA-regulated workplaces.

Tetramethyl succinonitrile is a colorless and odorless solid. This substance is a decomposition product that is released when azobisisobutyronitrile, a foam blowing agent, is heated (ACGIH 1986, p. 466).

Tetramethyl succinonitrile is a systemic toxin that produces convulsions in exposed humans and animals. The oral LD_{50} is 60 mg/kg (Sax and Lewis 1989, p. 3228). Rats exposed to a 60-ppm concentration of this substance for 2 or 3 hours died, as did rats exposed for 30 hours to a 6-ppm concentration (Oetel 1958, in ACGIH 1986, p. 566). Ingestion of a 25-mg/kg dose of tetramethyl succinonitrile also caused 100 percent mortality in rats (Oetel 1958). Rats, guinea pigs, rabbits, and dogs dosed with this substance by various routes of administration developed convulsions and subsequently died of asphyxiation (NIOSH Criteria Document (Nitriles) 1978, p. 155).

In a personal communication to the ACGIH's TLV® committee, Dr. Ernest Mastromatteo reported that several workers at a vinyl foam plant in Ontario, Canada, reported experiencing headaches and nausea after exposure to tetramethyl succinonitrile (ACGIH 1986, p. 566). There are reports of similar symptoms, and of convulsions and comas, in European workers exposed to unspecified concentrations of tetramethyl succinonitrile (Reinl 1957; Quoss 1959, both cited in ACGIH 1986, p. 566). No other toxicity information has been reported for this substance.

Based on this evidence in humans and animals, OSHA preliminarily concludes that tetramethyl succinonitrile is a systemic poison that potentially causes headache, nausea, and convulsions in exposed workers. Accordingly, the Agency is proposing a PEL of 0.5 ppm as an 8-hour TWA, with a skin notation, for this substance in agriculture. OSHA believes that this action will substantially reduce these risks of material health impairment among agricultural workers. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

TETRYL (2,4,6-Trinitrophenylmethylnitramine)
CAS: 479–45–8; Chemical Formula: $C_7H_5N_5O_8$
H.S. No. 2158

OSHA's current limit for tetryl in construction, maritime, and general industry is 1.5 mg/m³ as an 8-hour TWA, with a skin notation. There is no limit in agriculture, and NIOSH has no REL for this substance but concurs (Ex. 8–47, Table N3A) with the limit being proposed. The ACGIH TLV®–TWA is 1.5 mg/m³, with a skin notation, for this substance. OSHA is proposing an 8-hour TWA PEL of 1.5 mg/m³, with a skin notation, for tetryl in agriculture; adoption of this limit would make OSHA's PEL for this substance consistent across all OSHA-regulated industry sectors.

Tetryl (2,4,6-trinitrophenylmethylnitramine) is used as a booster explosive, a component in binary explosives, a primer for other explosives, and as a chemical indicator in the laboratory (HSDB 1988). Tetryl is a colorless to yellow crystalline solid with a bitter taste and no odor (ACGIH 1986, p. 568.1(88)).

Tetryl is an irritant and sensitizer of the skin and respiratory tract; exposure to tetryl has also been associated with systemic poisoning in the form of blood changes and liver toxicity. Little acute toxicity information is available. The lowest lethal dose of tetryl in dogs by subcutaneous administration is 5000 mg/kg (RTECS 1990). Rabbits given 1-g/kg doses of tetryl by stomach tube died after no more than three doses; at autopsy, degeneration of the kidneys was seen in these animals (Wells, Lewis, Sansum et al. 1920, in ACGIH 1986, p. 568.3(88)).

In humans, exposure to tetryl dust has caused eye, skin, and respiratory irritation; skin and pulmonary sensitization; liver damage; and anemia. Tetryl dust also stains the hair and skin of exposed workers yellow. Workers exposed to concentrations of tetryl below 1.5 mg/m³ for 10 years did not show signs of systemic toxicity but did report gastrointestinal and respiratory symptoms and skin sensitization (Bergman 1952). The dermal sensitization effects in these workers progressed from itching of the skin and eyes to erythema and edema (Bergman 1952). Eleven workers accidentally overexposed to tetryl developed yellow pigmentation of the skin, dermatitis, sensitization, respiratory tract irritation, and clinical signs of liver damage; two of these workers died and one became disabled (Hardy and Maloof 1950). Several of the surviving workers were subsequently forced to leave employment because of respiratory sensitization (Hardy and Maloof 1950). A study of a group of 1,258 tetryl-exposed workers revealed dermatitis and clinical evidence of secondary anemia in many of these workers. The symptoms of systemic illness reported by workers in this group include headache, lassitude, sleeplessness, and irritability (Hardy and Maloof 1950). In workers severely overexposed, irreversible liver damage has also occurred (Hardy and Maloof 1950).

Based on this evidence in humans and animals, OSHA preliminarily concludes that an 8-hour TWA limit of 1.5 mg/m³, and a skin notation, is necessary to protect agricultural workers from the skin and respiratory tract irritation and sensitization, blood effects, and liver damage potentially associated with exposure to tetryl. The Agency believes that the proposed limit will substantially reduce these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

THIRAM
CAS: 137–26–8; Chemical Formula: $C_6H_{12}N_2S_4$
H.S. No. 2160

OSHA's limit for thiram in general industry, construction, and maritime is 5 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH TLV®–TWA for this substance is 5 mg/m³ as an 8-hour TWA. NIOSH has no REL for this substance but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ for thiram in agriculture, which would make OSHA's PEL for this substance consistent across all industry sectors.

Thiram is a white or pale yellow powder with a characteristic odor. Thiram is used in many products and processes, including the compounding of natural and synthetic rubbers, the production of fungicides, in animal repellents, as a bacteriostat in soaps, and as an ingredient in antiseptic sprays (IARC 1976, Vol. 12, p. 228). Thiram is the methyl analog of Antabuse® (disulfiram), the drug used to control

alcoholism by interfering with the metabolism of alcohol (Clayton and Clayton 1981, p. 2104). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Thiram is an irritant of the eyes, mucous membranes, and skin; a skin sensitizer; and a metabolic poison. This substance is also a teratogen in experimental animals. The oral LD_{50} in rats is 560 mg/kg, and the lowest lethal dermal dose in rabbits is 1 g/kg (RTECS 1990). Acutely poisoned animals show hyperemia and focal ulceration of the gastrointestinal tract, necrosis of the liver and kidney, and demyelinization of the cerebellum and medulla (Child and Cramp 1952, in Gosselin, Smith, and Hodge 1984, p. III–383). Before death, acutely poisoned animals lose coordination, develop tremors and difficult breathing, and convulse (Ben-Dyke, Sunderson, and Ninakes 1970, in ACGIH 1986, p. 573.3(87)). In contact with the skin or eyes of rabbits, thiram causes irritation, and repeated contact of this substance with the skin of rabbits and guinea pigs has caused dermal sensitization (RTECS 1990; Griepentrop 1960; Brusilovsky and Fialikovsky 1973, in ACGIH 1986, p. 573.3(87)). Rats fed 100, 300, or 500 mg/kg thiram for 2 years showed reductions in growth rate; at the highest dose, the rats convulsed and, at autopsy, had thyroid hyperplasia and calcification of the brain (IARC 1976, Vol. 12, p. 231). Administered to rats, mice, and hamsters by the oral and/or subcutaneous route, thiram caused teratogenic effects in the offspring (RTECS 1990).

Ingestion of alcohol and concomitant exposure to thiram causes the signs and symptoms of systemic Antabuse®-alcohol syndrome: Flushing of the skin, palpitations, headache, nausea, vomiting, and difficult breathing (Hayes 1982, pp. 603–606). One worker died after treating seeds with thiram over a 10-hour period; it is not known whether this worker also ingested alcohol (Hayes 1982, pp. 603–606). Workers chronically exposed to low levels of thiram may develop facial flushing after they ingest alcohol, and thiram can produce dermatitis and sensitization in some workers even in the absence of alcohol ingestion (Shelley 1964). A study of 223 workers exposed to thiram for more than 3 years revealed an increased incidence of eye irritation, chest pain, tachycardia, nose bleeds, skin lesions, liver dysfunction, myocardiodystrophia, and asthenia in these workers compared with the incidence for a group of non-thiram-exposed workers. The thiram-exposed group also showed an excess incidence of enlarged thyroid and other thyroid abnormalities, as well as one case of thyroid adenocarcinoma (Cherpak et al. 1971; Kaskevich and Bezugly 1973, in IARC 1976, Vol. 12, p. 232).

Based on this evidence in humans and animals, OSHA preliminarily concludes that thiram poses a potentially significant risk of systemic toxicity to agricultural workers. The Agency believes that the proposed 5 mg/m^3 8-hour TWA PEL is necessary to substantially reduce this risk of material health impairment among these workers. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

TIN, METAL AND INORGANIC COMPOUNDS (as Sn)

CAS: 7440–31–5; Chemical Formula: Sn
H.S. No. 2161

OSHA's permissible exposure limit for tin and its inorganic compounds (measured as tin) in general industry, construction, and maritime is 2 mg/m^3 as an 8-hour TWA. There is no limit in agriculture. The ACGIH has an 8-hour TLV®-TWA of 2 mg/m^3 for these substances, and NIOSH has no REL. OSHA is proposing an 8-hour TWA limit of 2 mg/m^3 for tin and inorganic tin compounds (measured as tin) in agriculture. Promulgation of this limit will make the PEL for these substances consistent across all OSHA-regulated sectors.

Tin is an odorless, soft, white, silvery metal. Among its inorganic compounds are stannous chloride, an odorless, colorless to brown solid; stannic chloride, which is a colorless to yellow fuming liquid with an acrid odor; and stannous sulfate, a colorless to brown, odorless solid. Tin and its inorganic compounds are used in tin plating, solder and alloy production, and in the manufacture of toothpaste, ceramics, drill-glass, porcelain, enamels, textiles, and ink (Rom 1983, p. 501; NIOSH/OSHA Occupational Health Guideline 1981, p. 1).

Exposure to metallic tin dust or to inorganic tin compounds causes eye, nose, skin, and respiratory tract irritation, gastrointestinal effects, and anemia in humans and animals. The oral LD_{50} values for tin sulfate in rats and mice are 2207 mg/kg and 2152 mg/kg, respectively (RTECS 1990). In rats, mice, and rabbits, the oral LD_{50}s for stannous chloride are 700 mg/kg, 250 mg/kg, and 10 g/kg, respectively (RTECS 1990). Tin tetrachloride has an LC_{50} in rats of 2300 mg/m^3 for 10 minutes (RTECS 1990). The intraperitoneal injection of tin dust resulted in a non-specific, vascularized chronic granulomatous reaction in guinea pigs (Oyanguren, Haddad, and Maass 1958, in ACGIH 1986, p. 574). The soluble acid salts of tin produce dermal effects in rabbits, including intraepidermal polymorphonuclear leukocyte pustules when large amounts are held in contact with the skin for 18 hours; the application of lower concentrations causes polymorphonuclear infiltrates (Stone and Willis; Connie et al. 1975 and 1976; Yum et al. 1976, in Clayton and Clayton 1981, p. 1946). The signs and symptoms of low-level subacute tin toxicity are diarrhea, paralysis, twitching, hyperemia, and vascular changes, which in turn cause internal bleeding and death (Barnes and Stoner 1959, in Clayton and Clayton 1981, p. 1946). Rats fed tin oxides and various tin salts for 4 or 13 weeks at levels of 0.03, 0.1, 0.3, or 1.0 percent showed signs of growth retardation, decreased food efficiency, slight anemia, and liver changes at the 0.3 percent level or higher; the no-effect level was estimated to be 0.1 percent or 22 to 23 mg/day (deGroot et al. 1973, in Clayton and Clayton 1981, p. 1946). Paralysis was produced in dogs after daily administration of stannous chloride in milk at a level of 500 mg/kg for 14 months (Stauden 1972, in Proctor, Hughes, and Fischman 1988, p. 475).

In humans, some inorganic tin compounds cause skin and eye irritation because of an acid or alkaline reaction produced when the compound reacts with water (Stauden 1972, in Proctor, Hughes, and Fischman 1988, p. 475). Stannic chloride is highly irritating to the eyes and mucous membranes (Grant 1986, p. 846). Workers involved in glass bottle production who were exposed to a stannic chloride mist in concentrations of 0.10 to 0.18 mg/m^3 and to hydrogen chloride at a concentration of 5 ppm experienced an excess of respiratory irritation when compared to workers exposed predominantly to hydrogen chloride (Levy, Davis, and Johnson 1985, in Proctor, Hughes, and Fischman 1988, p. 475). Because of low absorption and rapid tissue turnover, the ingestion of inorganic tin causes only moderate toxicity. The ingestion of food contaminated with tin compounds at levels greater than 1400 ppm causes symptoms of gastrointestinal irritation such as nausea, abdominal cramps, vomiting, and diarrhea (Schafer and Femfurt 1984, in Proctor, Hughes, and Fischman 1988, p. 475). Chronic absorption of low levels of tin has caused abdominal pain, nausea, constipation, weight loss, sore throat,

mild fever, a sensation of being chilled, myalgias, arthralgias, and moderate anemia (HSDB 1986).

Based on this evidence, OSHA preliminarily concludes that tin and its inorganic compounds cause primary irritation, gastrointestinal effects, and anemia. OSHA therefore believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed limit of 2 mg/m³ as an 8-hour TWA is necessary to substantially reduce the risks of these material health impairments. Promulgation of this limit also will make the PEL for tin and its inorganic compounds consistent across all OSHA-regulated sectors.

TRIMETHYLBENZENE

CAS: 25551–13–7; Chemical Formula: $(CH_3)_3C_6H_3$

H.S. No. 1412

OSHA currently has an exposure limit of 25 ppm as an 8-hour TWA for trimethylbenzene in general industry, construction, and maritime. There is no limit in agriculture. The ACGIH TLV® for all isomers of trimethylbenzene is 25 ppm as an 8-hour TWA. OSHA proposes a PEL of 25 ppm as an 8-hour TWA in agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the 25-ppm TWA limit being proposed. This action would make OSHA's PEL for trimethylbenzene consistent across all regulated sectors.

Trimethylbenzene is a liquid mixture of three isomers. The isomers are used as raw materials for chemical synthesis and in ultra-violet stabilizers (ACGIH 1986, p. 508). Trimethylbenzene also is used in solvents.

A study by Battig, Grandjean, and Turrian (1957/Ex. 1–104) provides the basis for the proposed limit; this work reports symptoms among 27 workers exposed to a solvent containing 30 percent 1,3,5-trimethylbenzene and 50 percent 1,2,3-trimethylbenzene. A "significant number" of these workers were reported to have experienced symptoms of nervousness, tension and anxiety, and asthmatic bronchitis. The peripheral blood of these workers "showed a tendency to hypochromic anemia" and a somewhat abnormal clotting ability. This group of workers had been occupationally exposed to total hydrocarbon concentrations ranging from 10 to 60 ppm for several years. The authors of the study recommended maintaining employee exposures below 35 ppm (Battig, Grandjean, and Turrian 1957/Ex. 1–104).

Based on this evidence, OSHA is proposing a 25-ppm 8-hour TWA PEL for trimethylbenzene in agriculture to reduce the significant risks of bronchitis and blood effects reported to occur in exposed workers. The Agency considers these effects material health impairments and believes that the proposed PEL is necessary to substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

TUNGSTEN, INSOLUBLE COMPOUNDS (as W)

CAS: 7440–33–7; Chemical Formula: W

H.S. No. 1416

OSHA's PEL for tungsten and its insoluble compounds is an 8-hour TWA of 5 mg/m³ (measured as tungsten) in construction and maritime. There is no limit in agriculture. The ACGIH has established 5 mg/m³ as an 8-hour TLV®–TWA and 10 mg/m³ as a 15-minute TLV®–STEL for these substances. NIOSH recommends a limit of 5 mg/m³ as a 10-hour TWA. In construction and maritime, OSHA is retaining the PEL of 5 mg/m³ as an 8-hour TWA and is proposing a 15-minute STEL of 10 mg/m³; the Agency is also proposing to extend these limits in agriculture. NIOSH concurs (Ex. 8–47, Table N1) with OSHA's proposed limits, which are the limits recently established for these substances in general industry.

Tungsten is a gray, hard metal. Metallic tungsten is used in ferrous and non-ferrous alloys, incandescent lamps, heating elements, welding electrodes, rocket nozzles, and in solar energy equipment. Tungsten compounds are used in cemented carbide tools, dies and wear-resistant parts, textiles, ceramics, and plastics. Tungsten compounds are also used as solid lubricants and as abrasives (ACGIH 1986, p. 614).

Rats fed a diet containing 0.5 percent insoluble tungsten compounds died, and another group of rats fed 0.1 percent of these compounds suffered noticeable weight loss (Kinard and Van de Erve 1941/Ex. 1–492). Studies in rats fed tungsten at 2, 5, or 10 percent of their diet showed that females in all dose groups had a 15-percent reduction in weight gain (Kinard and Van de Erve 1943/Ex. 1–493). The intraperitoneal LD_{50} for tungsten metal powder in rats was 5 g/kg body weight; survivors showed minor liver and spleen changes at necropsy (Fredrick and Bradley 1946, as cited in ACGIH 1986/Ex. 1–3, p. 614). Studies of the tissues of guinea pigs intratracheally injected with tungsten metal or with tungsten carbide revealed moderate interstitial cellular proliferation (tungsten metal) and no changes (tungsten carbide). However, Soviet studies involving similar intratracheal injections showed proliferation of the intra-alveolar septa (Kaplun and Mezentseva 1960, as cited in ACGIH 1986/Ex. 1–3, p. 614).

The NIOSH criteria document for tungsten (1977h) reports that Russian investigators found a 9- to 11-percent incidence of pulmonary fibrosis in workers exposed to tungsten (Kaplun and Mezentseva 1959/Ex. 1–961; and Mezentseva 1967, as cited in ACGIH 1986/Ex. 1–3, p. 614). NIOSH (1977h) recommended that the standard for tungsten and its insoluble compounds be set at 5 mg/m³ to protect against pulmonary effects. Stokinger (Clayton and Clayton 1981) reported on several epidemiological studies of workers in the "hard metal industry," in which tungsten carbide is machined. These studies describe a condition known as hard metal disease, which may be accompanied by pulmonary fibrosis. The disease is characterized by a moderate incidence of cough, dyspnea, and wheezing, a high incidence of minor radiological abnormalities with a few instances of marked abnormalities, and development of hypersensitivity asthma in some workers (which may be due to exposure to the cobalt that is used as a binding agent). The disease is progressive and potentially lethal. This author (Clayton and Clayton 1981, p. 1992) reported that, unlike other lung diseases produced by inorganic dust, there is no correlation between onset of symptoms, length of exposure, and the development of interstitial fibrosis. Analysis of the lung of one worker who had clinical signs and radiological changes showed the presence of large amounts of tungsten with much smaller amounts of other metals.

In construction and maritime, OSHA is retaining the 8-hour TWA limit of 5 mg/m³ and proposing to add a STEL of 10 mg/m³ for tungsten and its insoluble compounds, measured as tungsten; the Agency is also proposing to extend these limits to agriculture. OSHA preliminarily concludes that these limits are necessary to substantially reduce the significant risk of pulmonary fibrosis and other lung effects, which OSHA considers material impairments of health. In addition, promulgation of these limits will make OSHA's PELs for these substances consistent across all regulated sectors.

TUNGSTEN, SOLUBLE COMPOUNDS (as W)

CAS: 7440–33–7; Chemical Formula: W

H.S. No. 1417

Currently, OSHA has a limit for tungsten and its soluble compounds in construction and maritime of 1 mg/m³ (measured as tungsten) as an 8-hour

TWA. There is no limit in agriculture. The ACGIH TLV®–TWA is 1 mg/m³, with a TLV®–STEL of 3 mg/m³, measured as tungsten. NIOSH recommends a 1-mg/m³ 10-hour TWA for tungsten and its soluble compounds. OSHA is retaining the 8-hour TWA PEL of 1 mg/m³ and proposing to add a 15-minute STEL of 3 mg/m³ for tungsten and its soluble compounds in construction and maritime; the Agency also proposes these limits in agriculture. NIOSH concurs (Ex. 8–47, Table N1) with the addition of a STEL to the 1-mg/m³ TWA limit. These are the limits recently established for these substances in general industry.

Tungsten is a gray, hard metal. Metallic tungsten is used in ferrous and non-ferrous alloys, incandescent lamps, heating elements, welding electrodes, rocket nozzles, and in solar energy equipment. Tungsten compounds are used in cemented carbide tools, dies and wear-resistant parts, textiles, ceramics, and plastics. Tungsten compounds are also used as solid lubricants and as abrasives (ACGIH 1986, p. 614).

Animal studies have shown that the LD_{50} for soluble sodium tungstate when injected subcutaneously in rats ranges from 140 to 160 mg/kg (Kinard and Van de Erve 1940/Ex. 1–788). Soluble tungsten's lethal effects are the result of systemic poisoning that occurs as the compound is absorbed by multiple organs; this is followed by cellular asphyxiation (International Labour Office [ILO] 1934c, as cited in ACGIH 1986/Ex. 1–3, p. 614). Karantassis (1924, as cited in ACGIH 1986/Ex. 1–3, p. 614) also observed a systemic response in guinea pigs given soluble sodium tungstate or pure soluble tungsten either orally or intravenously; the animals developed anorexia, colic, trembling, and difficulty in breathing prior to death. Rats fed a diet containing 0.5 percent tungsten as soluble sodium tungstate or tungsten oxide died from this dose. Dietary doses of 0.1 percent tungsten oxide and the sodium salt caused weight loss in rats, but no deaths (Kinard and Van de Erve 1941/Ex. 1–492). Tungsten is believed to act by antagonizing the action of molybdenum (Higgins, Richert, and Westerfield 1956/Ex. 1–487). In its criteria document for tungsten, NIOSH (1977h) states that information on the effects of exposure to soluble tungsten compounds in the working population is not available.

OSHA is retaining the 8-hour TWA limit of 1 mg/m³ and proposing to add a STEL of 3 mg/m³ for tungsten and its soluble compounds, measured as tungsten, in construction and maritime; the Agency is also proposing to extend these limits to agriculture. OSHA preliminarily concludes that these limits will protect workers in these sectors against the significant risks of systemic toxicity, anorexia, colic, incoordination, trembling, and dyspnea, all of which constitute material health impairments that are associated with exposure to these compounds. In addition, promulgation of these limits will make OSHA's PELs for these substances consistent across all regulated sectors.

VINYLIDENE CHLORIDE (1,1-DICHLOROETHYLENE)

CAS: 75–35–4; Chemical Formula: $CH_2 = CCl_2$

H.S. No. 1428

OSHA has no limit for vinylidene chloride (VDC) in construction, maritime, or agriculture. The ACGIH has established a 5 ppm TLV®–TWA and a 20 ppm 15-minute TLV®–STEL. In 1978, NIOSH recommended that employee exposure to VDC be reduced to the lowest feasible level on the basis of VDC's carcinogenicity (Ex. 1–1119). OSHA is proposing a PEL of 1 ppm (8-hour TWA) for VDC in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Vinylidene chloride, also called 1,1-dichloroethylene, is a colorless liquid that polymerizes readily. It is used primarily for production of vinylidene copolymers such as Saran® and Velon®. It is also used in films and coatings (ACGIH 1986, p. 628).

In animals, exposure to vinylidene chloride causes irritation, kidney and liver damage, and cancer. The oral LD_{50} in male rats is 2500 mg/kg (Jenkins, Trabulus, and Murphy 1972/Ex. 1–960). The LC_{50} for rats exposed to a single 4-hour exposure of VDC vapor was reported as 6350 ppm in one study (Siegel, Jones, Coon, and Lyon 1971/Ex. 1–371) and 32,000 ppm in an earlier study (Carpenter, Smyth, and Pozzani 1949/Ex. 1–722). Liquid VDC causes transient irritation of the eyes in rats but has little effect on exposed skin if the VDC is allowed to evaporate (Torkelson and Rowe 1981b, as cited in ACGIH 1986/Ex. 1–3, p. 628). Prendergast and co-workers (1967/Ex. 1–926) exposed rats, rabbits, guinea pigs, and monkeys 8 hours/day, 5 days/week for 6 weeks to 395 mg/m³ (100 ppm); these authors saw no visible signs of toxicity while the exposure was in process, but rabbits and monkeys lost weight. Animals of these same species were exposed continuously to VDC concentrations of 5, 15, 25, or 47 ppm for 90 days; only the animals exposed to 5 ppm showed no increases in mortality (Prendergast, Jones, Jenkins, and Siegel 1967/Ex. 1–926).

Nasal irritation, liver cell degeneration, and retarded weight gain were reported in rats following 20 6-hour exposures to 500 ppm VDC (Gage 1970/Ex. 1–318); at 200 ppm, only nasal irritation occurred. Studies by Torkelson and Rowe (1981b, as cited in ACGIH 1986/Ex. 1–3, p. 628) in which rats, rabbits, guinea pigs, and dogs were exposed to 25, 50, or 100 ppm VDC for 8 hours per day, 5 days per week for 6 months revealed injury of the kidneys and liver in all animals at all levels of exposure. Maltoni (1977/Ex. 1–985) and Maltoni, Cotti, Morisi, and Chieco (1977/Ex. 1–1090) conducted an evaluation of VCD's carcinogenicity in which mice, rats, and hamsters were exposed to levels from 10 to 150 ppm for 4 hours per day, 5 days per week for 52 weeks, with results reported through week 98 of the study. In those mice exposed to 25 ppm VCD, 21 percent of the males and 1.5 percent of the females developed kidney adenocarcinomas; these tumors were not seen in rats exposed to amounts of VDC up to 150 ppm. Exposures of 100 or 150 ppm in rats did produce a significant increase in mammary adenocarcinomas, and this response was dose-related (Maltoni 1977/Ex. 1–985; Maltoni, Cotti, Morisi, and Chieco 1977/Ex. 1–1090). Overt toxicity and mortality occurred early in the studies after 4-hour exposures at levels of 50 ppm in mice and 200 ppm in rats; hamsters exposed to 20 ppm VDC showed no increase in tumor incidence (Maltoni 1977/Ex. 1–985; Maltoni, Cotti, Morisi, and Chieco 1977/Ex. 1–1090).

Two strains of rats exposed to 75 or 100 ppm VDC for 5 days/week, 6 hours/day for 12 months did not show a significant increase in tumors (Viola and Caputo 1977/Ex. 1–937). Other investigators exposed rats to 25 or 75 ppm by inhalation for 6 hours/day, 5 days/week for 18 months, or to 60, 100, or 200 ppm VDC in their drinking water for 2 years, and found no increase in tumor incidence in these animals (Rampy, Quast, Humiston et al. 1977, as cited in ACGIH 1986/Ex. 1–3, p. 628). In mice, VDC was not active either as a whole mouse skin carcinogen or by subcutaneous injection.

In an NTP gavage study of VDC in mice and rats (NTP 1982/Ex. 134B), the only observed significant increase in tumor incidence occurred in low-dose female mice; this increase was not considered to be related to VDC administration because similar effects were not observed in high-dose female mice, male mice, or rats. The NTP (1982/Ex. 134B) concluded that VDC was not

carcinogenic in mice or rats exposed by gavage, but cautioned that a maximum tolerated dose had not been demonstrated and that previously reported studies had shown that carcinogenicity is associated with VDC inhalation by animals.

In other studies, VDC proved mutagenic in both *E. coli* and *S. typhimurium* strains (Greim, Bonse, Radwan et al. 1975/Ex. 1–904; Bartsch, Malaveille, Montesano, and Tomatis 1975/Ex. 1–889). VDC has been implicated as a tumor initiator in a carcinogenesis bioassay by Van Duuren, Goldschmidt, Loewengart et al. (1979/Ex. 1–936). Studies by Reitz, Watanabe, McKenna et al. (1980/Ex. 1–927) suggest that VCD's tumorigenicity is a result of its ability to initiate cell injury, rather than of its ability to alter the genetic material of an injured cell. However, VDC has been shown to alkylate DNA *in situ* and to increase the rate of DNA repair to a small extent in mice (Norris and Reitz 1984/Ex. 134B). The actual cell injury is caused by VDC metabolites, which are highly reactive and cytotoxic (Maltoni 1977/Ex. 1–985; Hathway 1977/Ex. 1–906; Henschler and Bonse 1977/Ex. 1–908).

A study by Murray, Nitschke, Rampy, and Schwetz (1979/Ex. 1–920) investigated the embryotoxic, fetotoxic, and teratogenic effects of inhaled and ingested VDC (in rats) and inhaled VDC (in rabbits). In the inhalation studies, rats were exposed to 20, 80, or 160 ppm VDC for 7 hours per day. VDC was toxic to both the adults and their embryos at levels of 80 and 160 ppm among the rats, and at 160 ppm in rabbits. At exposure levels of 20 ppm in rats and 80 ppm in rabbits, neither maternal toxicity nor effects on embryonic or fetal development were noted. In the ingestion study with rats, drinking water containing 200 ppm VDC caused no toxic effects in either the rats or their offspring.

A cohort study of 138 VCD-exposed workers did not identify any VCD-related health effects in these workers (Ott, Fishbeck, Townsend, and Schneider 1976/Ex. 1–924). The cohort was too small to provide any evidence that VDC is not likely to be carcinogenic.

OSHA believes that these studies clearly demonstrate that VDC can cause adverse liver and kidney damage in animals at airborne concentrations as low as 25 to 50 ppm and suggest that VDC is a potential occupational carcinogen. Liver and kidney damage and cancer clearly constitute material health impairments within the meaning of the Act. Therefore, OSHA preliminarily concludes that a 1-ppm 8-hour TWA limit for vinylidene chloride is necessary to protect workers in construction, maritime, and agriculture from these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

WELDING FUMES (TOTAL PARTICULATE)

CAS: None; Chemical Formula: Varies
H.S. No. 1430

In construction, maritime, and agriculture, OSHA has no limit for welding fumes, which are defined as fumes that are generated by the manual metal arc or oxy-acetylene welding of iron, mild steel, or aluminum. NIOSH has no REL for welding fumes. The ACGIH has set an 8-hour TLV®–TWA of 5 mg/m³ for these welding fumes, measured as total particulate in the welder's breathing zone. OSHA is proposing an 8-hour TWA limit of 5 mg/m³ for these fumes in construction, maritime, and agriculture. This is the limit recently established for these fumes in general industry. This limit applies to the total fume concentration generated during the welding of iron, mild steel, or aluminum; the fumes generated by the welding of stainless steel, cadmium, or lead-coated steel, or other metals such as copper, nickel, or chrome are considerably more toxic and should be kept at or below the levels required by their respective PELs. Welding fumes consist of metallic oxides generated by the heating of metal being welded, the welding rod, or its coatings.

Although these types of welding generally produce fumes consisting of aluminum, iron, or zinc oxides, other toxic gases may also be produced in large amounts (Ferry and Ginther 1952/Ex. 1–900; Ferry 1954/Ex. 1–782; Silverman 1956/Ex. 1–1169; Homer and Mohr 1957/Ex. 1–787). The welding of iron metals may give off fumes of manganese, silicate, and various organic binders. Aluminum welding may generate fumes consisting of fluorine, arsenic, copper, silicon, and beryllium (NIOSH 1975h and American Welding Society 1974, both as cited in ACGIH 1986/Ex. 1–3, p. 634). Eighteen different substances, including fluoride, manganese, silicon, titanium, and sodium and potassium silicates, have been measured in the fumes resulting from the welding of mild steel (ACGIH 1986/Ex. 1–3, p. 634). Moreover, there are numerous and undefined additional compounds present in welding fumes whose health effects have not been fully assessed. These include naphthalene, phenol, cresol, dioxane, benzene, pyridine, and aromatic hydrocarbons. Because these unregulated compounds may present additional, unidentified risks, OSHA reasonably determined that exposure to welding fumes is likely to cause adverse health effects at concentrations above 5 mg/m³.

Excessive exposure to welding fumes can cause a variety of disorders, most notably metal fume fever. It has been estimated that 30 to 40 percent of all welders have experienced metal fume fever at some time (Ross 1974, *J. Occup. Med.* 24:125, 127). This disorder, which results from exposure to freshly formed metal fume, results in the appearance of delayed, flu-like symptoms, including dyspnea, coughing, pains in muscles and joints, fever, and chills. Recovery usually requires 1 or 2 days of time away from work. In addition to fume fever, exposure to welding fumes may damage the small airways, causing interstitial pneumonia (Ross 1974). The recent NIOSH Criteria Document (1988) on welding fumes reviews the literature on the health effects of exposure to these fumes. Studies such as those by Ross (1974) and Antti-Poika demonstrate that workers exposed to these fumes at approximately 5 mg/m³ develop welding fume fever, chronic bronchitis, and other respiratory diseases. NIOSH (1988) reported that welders experience excess morbidity and mortality even when they are exposed to welding fumes at concentrations below the limits for many of the individual constituents of the welding emissions (Ex. 8–47, p. iii).

In the prior rulemaking, several commenters asked OSHA to clarify whether the limit applied to exposure samples taken inside or outside of the welding helmet. OSHA responded that it is current Agency policy that welding fumes are to be measured in the breathing zone (inside the welding hood) of the welder as stated in the 1990 Field Operations Manual.

In the earlier rulemaking, NIOSH (Ex. 8–47) stated that welding fumes should be designated as a carcinogen; this view was shared by Dr. James Melius, of the New York State Department of Health (Tr. p. 11–104). In response to these commenters, OSHA states that more information and analysis are needed before this issue can be resolved. Accordingly, OSHA believes that it would be premature to identify these fumes as carcinogens.

OSHA intends that the proposed limit apply to all welding operations where the worker stands at or near the point where the welding takes place. This would include manual, some automatic welding where the rod is fed automatically, and other types of welding where the worker is located at

or near the point of the welding. The reason for OSHA's position is that coverage is needed where there is worker exposure—no matter what the name given to the welding. Fully automatic welding, in which the worker is located away from the welding operation and would have no exposure, is not covered by this limit. For several reasons, this limit is needed in addition to the limits for specific constituents of the welding fume. First, significant risk of material health impairment is associated with this type of welding. Second, individual substance limits have not proven sufficiently protective. Third, so many types of fumes are given off that an overall limit is necessary to help reduce possible synergistic effects. Fourth, for small-business employers and OHSA inspectors, measuring for overall welding fumes is easier and will thus encourage compliance. (However, OSHA PELs for the individual constituents of the fume—e.g., cadmium, lead, other metals—do remain in effect as applicable.)

OSHA preliminarily concludes that a PEL for welding fumes is needed to protect workers in construction, maritime, and agriculture who are involved in the welding of aluminum, iron, or mild steel from the significant risk of metal fume fever and respiratory irritation associated with the generation of welding fumes.

Accordingly, OSHA is proposing an 8-hour TWA limit of 5 mg/m³ for these particular types of welding fumes, measured as total particulate inside the welder's breathing zone. The Agency believes that this limit will substantially reduce the significant risk of material health impairment to which manual metal arc or oxy-acetylene welders of iron, mild steel, or aluminum are exposed in the absence of an OSHA limit in construction, maritime, and agriculture. In addition, promulgation of this limit will make OSHA's PEL for these fumes consistent across all regulated sectors.

ZINC OXIDE (FUME)
CAS: 1314–13–2; Chemical Formula: ZnO
H.S. No. 1437

In construction and maritime, OSHA's permissible exposure limit for zinc oxide fume is 5 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH recommends a 5 mg/m³ TLV®–TWA and also has a TLV®–STEL of 10 mg/m³. NIOSH recommends a 5-mg/m³ 10-hour TWA limit with a 15-minute ceiling of 15 mg/m³. In construction and maritime, OSHA is retaining the 5-mg/m³ 8-hour TWA limit and proposing to add a STEL of 10 mg/m³ for zinc oxide; the Agency is also proposing these limits in agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limits being proposed, which are the limits recently established for this substance in general industry.

Zinc oxide exposures of guinea pigs that lasted only an hour caused a drop in body temperature, followed 6 to 18 hours later by an increase above normal levels (Turner and Thompson 1926/Ex. 1–1124). The animals in the high-exposure group (2500 mg/m³ for 3 to 4 hours) died after exposure.

When heated, zinc oxide produces a white fume. The most prevalent toxic effect of zinc oxide fume is a condition known as "metal fume fever," whose symptoms include chills, fever, muscular pain, nausea, and vomiting (Turner and Thompson 1926/Ex. 1–1124). Studies in the workplace have shown that welders exposed to zinc oxide fume at concentrations of 320 to 580 mg/m³ reported nausea, with the development of chills, shortness of breath, and severe chest pains 2 to 12 hours later. Most workers took approximately 4 days to recover, and some eventually developed pneumonia (Hammond 1944/Ex. 1–981). Other studies have reported the frequent occurrence of chills in workers exposed to zinc oxide at levels as low as 5 mg/m³ (Hickish 1963 and Wall 1970, both as cited in ACGIH 1986/Ex. 1–3, p. 645). Hammond (1944/Ex. 1–981) reported that workers exposed to 8 to 12 mg/m³ of zinc oxide fume did not suffer from metal fume fever. However, subsequent experience has shown that exposures even to a concentration of 5 mg/m³ can cause this syndrome (Hickish 1963 and Wall 1970, both as cited in ACGIH 1986/Ex. 1–3, p. 646). NIOSH's criteria document (1975d) reported that exposures to the fume at the 5 mg/m³ level could cause chronic respiratory effects.

Based on this evidence in humans and animals, OSHA is retaining its 5-mg/m³ 8-hour TWA limit and proposing to add a STEL of 10 mg/m³ for zinc oxide in construction and maritime; the Agency is also proposing these limits in agriculture. OSHA preliminarily concludes that these limits will protect workers in these sectors from the significant risks of metal fume fever and chronic respiratory damage associated with exposure to zinc oxide fumes. These effects constitute material health impairments and are associated both with acute and chronic exposure to zinc oxide fumes. Promulgation of these limits will also make OSHA's PELs for this substance consistent across all regulated sectors.

ZIRCONIUM COMPOUNDS (as Zr)
CAS: 7440–67–7; Chemical Formula: Zr
H.S. No. 1439

The OSHA PEL for zirconium compounds in construction and maritime is an 8-hour TWA of 5 mg/m³, measured as zirconium. There is no limit in agriculture. The ACGIH has established a TLV®–TWA of 5 mg/m³, supplemented by a 10-mg/m³ STEL, for these substances. There is no NIOSH REL. OSHA is retaining the 8-hour TWA and proposing to add a STEL of 10 mg/m³ for these substances in construction and maritime and is also proposing to extend these limits to agriculture. This action would make OSHA's PEL for the compounds of zirconium consistent across all sectors.

Zirconium compounds may be either bluish-black powders or grayish-white lustrous metals. Zirconium metal is used in nuclear technology, photo flash bulbs, vacuum tubes, and in steel manufacture. Compounds of zirconium are used in ceramics, enamels, porcelain, abrasives, and in making refractories. The compounds are also used in high-temperature batteries and as textile water-repellant and tanning agents (ACGIH 1986, p .647). Some of these substances also found use in pesticides. When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The toxic effects of inhalation exposures to zirconium compounds include the formation of granulomas, both in the lungs and on the skin. Sax (1984) reports cases of pulmonary granulomas in workers exposed to zirconium aerosols. In laboratory animals, oral toxicity is low (NIOSH 1972b), and inhalation studies conducted in experimental animals for 1 year at levels of 3.5 mg zirconium/m³ dust and mist resulted in limited toxicity (Stokinger 1981c/Ex. 1–1134).

In the earlier rulemaking, NIOSH (Ex. 8–47) recommended that zirconium tetrachloride not be included in this group of zirconium compounds because an animal study by Spiegl et al. (1956, as cited in ACGIH 1986/Ex. 1–3, p. 647) showed effects in animals at 6 mg/m³. NIOSH (Ex. 8–47) recommended that OSHA establish a separate PEL for zirconium tetrachloride. However, because one of the program goals of this rulemaking is to make OSHA's limits consistent across all industry sectors, OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ and a 15-minute STEL of 10 mg/m³ for all zirconium compounds, including zirconium tetrachloride. OSHA preliminarily concludes that the 5 mg/m³ TWA and 10 mg/m³ STEL limits for the zirconium compounds, measured

as zirconium, will protect workers in construction, maritime, and agriculture from the significant risk of pulmonary effects potentially associated with exposure to these substances. The Agency believes that these effects constitute material health impairments and that the proposed PELs are necessary to substantially reduce the risks of these health effects. Promulgation of these limits also will make OSHA's PELs for these compounds consistent across all regulated sectors.

Preliminary Conclusions for This Group of Systemic Toxicants

For the group of systemic toxicants shown in Table C8–1, OSHA preliminarily concludes that the risks associated with occupational exposures are significant. As Table C8–2 shows, the systemic effects caused by such exposures include cancer, liver and kidney damage, testicular damage, fetal poisoning, central nervous system depression, and asthma, each of which constitutes material impairment of health within the meaning of the Act. Affected employees in construction, maritime, and agriculture may experience dizziness, nausea, generalized weakness, respiratory irritation, blood in the urine, chest tightness, hives, and necrosis of the cornea. These effects represent significant impairments of health and functional capacity, and reducing the limits for these systemic toxins will substantially reduce these significant risks. In addition, promulgation of the proposed limits will make OSHA's PELs for these systemic toxins consistent across all regulated sectors.

9. Substances for Which Proposed Limits Are Based on No-Observed-Adverse-Effect Levels

Introduction

For a group of 35 toxic substances, OSHA is proposing limits for the construction, maritime, and agriculture industries based on evidence that these substances cause toxic responses at higher levels but have been shown not to produce adverse effects in animals or exposed populations at the limits being proposed. The proposed PELs for this group of substances have thus been set in reference to no-observed-adverse-effect levels, or NOAELS. These substances are shown in Table C9–1, along with their CAS numbers and H.S. numbers. The 1987–88 ACGIH TLV®, the NIOSH REL (if any), the current OSHA PEL in construction and maritime, and the proposed OSHA PEL in construction, maritime, and agriculture are also shown in Table C9–1. Promulgation of the limits being proposed will make OSHA's PELs for these substances consistent across all regulated sectors.

TABLE C9–1.—SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON A NO-OBSERVED-ADVERSE-EFFECT LEVEL

H.S. number/ chemical name	CAS no.	Current OSHA PEL in construction and maritime[1]	1987–1988 ACGIH TLV® [2]	NIOSH REL[3]	Proposed OSHA PEL in construction, maritime, and agriculture[4]
1029 Atrazine	1912–24–9		5 mg/m³ TWA.		5 mg/m3 TWA.
2014 Azinphos Methyl.	86–50–0	0.2 mg/m³ TWA; skin	0.2 mg/m³ TWA; skin.		0.2 mg/m³ TWA; skin.
1041 Bromacil	314–40–9	–	1 ppm TWA		1 ppm TWA.
1056 p-tert-Butyltoluene.	98–51–1	10 ppm TWA	10 ppm TWA; 20 ppm STEL.		10 ppm TWA; 20 ppm STEL.
1085 Chlorodifluoromethane.	75–45–6		1000 ppm TWA; 1250 ppm STEL.		1000 ppm TWA.
1090 o-Chlorotoluene.	95–49–8		50 ppm TWA; 75 ppm STEL.		50 ppm TWA.
1110 Cyclonite	121–82–4		1.5 mg/m³ TWA; 3 mg/m³ STEL skin.		1.5 mg/m³ TWA; skin.
2056 Dibutylphthalate.	84–74–2	5 mg/m³ TWA	5 mg/m³ TWA.		5 mg/m³ TWA.
2059 1,2-Dichloroethylene.	540–59–0	200 ppm TWA	200 ppm TWA.		200 ppm TWA.
1117 2,6-di-tert-butyl-p-cresol.	128–37–0	–	10 mg/m³ TWA.		10 mg/m³ TWA.
1134 Diethanolamine.	111–42–2		3 ppm TWA		3 ppm TWA.
1136 Diethyl phthalate.	84–66–2		5 mg/m³ TWA.		5 mg/m³ TWA.
1144 Dinitolmide	148–01–6		5 mg/m³ TWA.		5 mg/m³ TWA.
1147 Diphenylamine.	122–39–4	10 mg/m³ TWA	10 mg/m³ TWA.		10 mg/m³ TWA.
1153 Diuron	330–54–1		10 mg/m³ TWA.		10 mg/m³ TWA.
2073 Endrin	72–20–8	0.1 mg/m³ TWA	0.1 mg/m³ TWA; skin.		0.1 mg/m³ TWA; skin.
2099 LPG	68476–85–7	1000 ppm TWA	1000 ppm TWA.		1000 ppm TWA.
1249 Methyl acetate.	79–20–9	200 ppm TWA	200 ppm TWA; 250 ppm STEL.		200 ppm TWA; 250 ppm STEL.
1275 Metribuzin	21087–64–9		5 mg/m³ TWA.		5 mg/m³ TWA.

TABLE C9-1.—SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON A NO-OBSERVED-ADVERSE-EFFECT LEVEL—Continued

H.S. number/ chemical name	CAS no.	Current OSHA PEL in construction and maritime[1]	1987-1988 ACGIH TLV® [2]	NIOSH REL[3]	Proposed OSHA PEL in construction, maritime, and agriculture[4]
2121 1-Nitropropane.	108-03-2	25 ppm TWA	25 ppm TWA		25 ppm TWA.
1297 Oil mist (mineral).	8012-95-1	5 mg/m³ TWA	5 mg/m³ TWA; 10 mg/m³ STEL.		5 mg/m³ TWA.
1312 Petroleum distillates (naphtha).	8002-05-9	[4]	400 ppm TWA.	350 mg/m³ TWA; 1800 mg/m³ ceiling (15-min).	400 ppm TWA.
1327 m-Phthalodinitrile.	626-17-5		5 mg/m³ TWA.		5 mg/m³ TWA.
1332 Platinum, metal.	7440-06-4		1 mg/m³ TWA.		1 mg/m³ TWA.
1346 Resorcinol	108-46-3		10 ppm TWA; 20 ppm STEL.	---	10 ppm TWA; 20 ppm STEL.
2139 Rotenone (commercial).	83-79-4	5 mg/m³ TWA	5 mg/m³ TWA.		5 mg/m³ TWA.
2146 Sulfur hexafluoride.	2551-62-4	1000 ppm TWA	1000 ppm TWA.		1000 ppm TWA.
1382 Tantalum, metal dust and oxide.	7440-25-7	5 mg/m³ TWA	5 mg/m³ TWA; 10 mg/m³ STEL.		5 mg/m³ TWA.
2153 1,1,2,2-Tetrachloro-1,2,-difluoroethane.	76-12-0	500 ppm TWA	500 ppm TWA.		500 ppm TWA.
2154 1,1,1,2-Tetrachloro-2,2-difluoroethane.	76-11-9	500 ppm TWA	500 ppm TWA.		500 ppm TWA.
2164 Trifluoromono-bromomethane.	75-63-8	1000 ppm TWA	1000 ppm TWA.		1000 ppm TWA.
1410 Trimethyl phosphite.	121-45-9		2 ppm TWA		2 ppm TWA.
1415 Triphenyl amine.	603-34-9		5 mg/m³ TWA.		5 mg/m³ TWA.
2165 Triphenyl phosphate.	115-86-6	3 mg/m³ TWA	3 mg/m³ TWA.		3 mg/m³ TWA.
1418 Uranium (insoluble compounds).	7440-61-1	0.2 mg/m³ TWA	0.2 mg/m³ TWA; 0.6 mg/m³ STEL.		0.2 mg/m³ TWA; 0.6 mg/m³ STEL.

[1] OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time; OSHA's PELs do not currently apply in Agriculture.

[2] The ACGIH TLV®-TWA is for an 8-hour exposure; its STELs are 15-minutes limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

[3] NIOSH TWA limits are for 10 hour/day, 40 hour/week exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

[4] Because the composition of this material varies greatly, the ACGIH recommends that the content of benzene, other aromatics, and additives should be determined to arrive at the appropriate TLV®.

Description of the Health Effects

The substances included in this group cause a wide range of adverse health effects in both animals and humans. Unlike most of the other groupings described in this preamble, these toxicants do not affect the same target organ or system: Some are central nervous system depressants, several are upper respiratory tract irritants, and still others have their primary effect on the heart, liver, or kidney. The commonality among these otherwise diverse substances is that apparent no-observed-adverse-effect levels (NOAELs) have been defined for all of them; that is, there are data demonstrating that overt toxic effects caused by exposure to these substances at higher levels do not occur below a certain "no-observed-adverse-effect" level. Permissible exposure limits have been developed for these chemicals on the basis of these no-observed-adverse-effect levels. Table C9-2 shows the health effects that have been observed in humans or animals that are believed likely to occur in humans who are exposed to these substances.

TABLE C9-2.—HEALTH EFFECTS ASSOCIATED WITH SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON NO-OBSERVED-ADVERSE-EFFECT LEVELS

H.S. number/chemical name	CAS No.	Health effects observed in animals	Health effects observed/projected in humans
1029 Atrazine	1912-24-9	Irritation, ataxia, dyspnea, convulsions, liver damage, cancer.	Irritation, systemic effects, contact dermatitis.
2014 Azinphos methyl	8650-0	Cholinesterase inhibition	Cholinesterase inhibition.
1041 Bromacil	314-40-9	Irritation, thyroid damage	Thyroid effects, irritation.

TABLE C9–2.—HEALTH EFFECTS ASSOCIATED WITH SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON NO-OBSERVED-ADVERSE-EFFECT LEVELS—Continued

H.S. number/chemical name	CAS No.	Health effects observed in animals	Health effects observed/projected in humans
1056 p-tert-Butyl-toluene	98–51–1	Central nervous system depression, irritation, liver and kidney changes.	Nasal irritation, nausea, headache, weakness.
1085 Chlorodifluoromethane	75–45–6	Central nervous system effects, cardiac sensitization, possible carcinogenicity.	Central nervous system effects, cardiac sensitization.
1090 o-Chlorotoluene	[illegible]	Weakness, vasodilation, incoordination, convulsions, irritation.	Neuropathic effects, irritation.
1110 Cyclonite	121–82–4	Convulsions, death	Central nervous system effects, convulsions, unconsciousness, death.
2056 Dibutylphthalate	84–74–2	Irritation, central nervous system effects, reproductive effects–.	Irritation, central nervous system effects, reproductive effects.
1117 2,6-Di-tert-butyl-p-cresol	128–37–0	Irritation, lung and liver damage, cancer promotion.	Irritation, skin sensitization.
2059 1,2-Dichloroethylene	540–59–0	Lung, liver, blood effects	Narcosis, irritation.
1134 Diethanolamine	111–42–2	Irritation, liver and kidney damage	Irritation.
1136 Diethyl phthalate	[illegible]	Irritation, central nervous system effects, liver damage.	Central nervous system effects, polyneuritis,–vestibular dysfunction.
1144 Dinitolmide	148–01–6	Liver changes	Hepatic effects.
1147 Diphenylamine	122–39–4	Liver, kidney, spleen changes, developmental effects.	Tachycardia, bladder symptoms, hypertension, eczema, eye and mucous –membrane irritation.
1153 Diuron	330–54–1	Anemia, methemoglobinemia, spleen and liver effects, teratogenic effects.	Anemia, methemoglobinemia, eye, skin, and respiratory irritation.
2073 Endrin	72–20–8	Reproductive, developmental effects	Convulsions, brain damage.
2099 LPG	68476–85–7	Central nervous system depression	Drowsiness, headache,–vomiting.
1249 Methyl acetate	79–20–9	Eye and skin irritation, CNS depression	Eye, mucous membrane irritation, chest tightness, narcosis, destruction of optic nerve.
1275 Metribuzin	21087–64–9	CNS depression, thyroid and liver changes	Narcosis, thyroid and liver damage.
2121 1-Nitropropane	108–03–2	Reproductive, developmental effects	Convulsions.
1297 Oil mist (mineral)	8012–95–1	Eye, skin, and respiratory irritation	Lung irritation, pneumonitis, photosensitization, dermatitis.
1312 Petroleum distillates (naphtha)	8030–30–6	Motor incoordination, convulsions	Eye and throat irritation, CNS depression.
1327 m-Phthalodinitrile	626–17–5	Skin irritation	Skin irritation.
1332 Platinum, metal	7440–06–4	Tumorigen by implantation	
1346 Resorcinol	108–46–3	Eye, skin irritation	Irritation, systemic effects (methemoglobinemia, cyanosis) pigmentary dermatosis.
2139 Rotenone (commercial)	83–79–4	Irritation, central nervous system effects, liver and kidney damage, cancer.	Irritation, nausea, vomiting, gastro-intestinal effects.
2146 Sulphur hexafluoride	2551–64–4	Central nervous system effects, asphyxiation	Central nervous system effects, asphyxiation.
1382 Tantalum, metal dust and oxide	7440–25–7	Bronchitis, pneumonitis, hyperemia, tumorigen by implantation –.	Pulmonary effects.
2153 1,1,2,2-Tetrachloro-2,2-difluoroethane	76–12–0	Central nervous system depression; blood and liver changes.	Dermatitis.
2154 1,1,1,2-Tetrachloro-2,2-difluoroethane	76–11–9	Irritation, narcosis	
2164 Trifluoromonobromomethane	75–63–8	Cardiac irregularities, seizures	Cardiac irregularities.
1410 Trimethyl phosphite	121–45–9	Teratogenicity, eye and skin irritation, lung damage.	Lung, skin, eye irritation.
1415 Triphenyl amine	[illegible]	Skin irritation	Skin irritation.
2155 Triphenyl phosphate	115–86–6	Cholinesterase inhibition	Cholinesterase inhibition.
1418 Uranium (insoluble compounds)	7440–61–1	Kidney damage, blood disorders	Kidney damage, blood effects.

Dose-Response Relationships and No-Observed-Adverse-Effect Levels

The concept of setting limits based on a NOAE level assumes that there is a concentration at which repeated and prolonged exposure to a toxic substance causes no observable adverse effect in the majority of workers. A similar concept is widely used by Federal agencies, such as the Food and Drug Administration and the Environmental Protection Agency, to set contaminant tolerances, acceptable daily intake values, and other limits.

All of the proposed limits for this group of substances have been set at or below a no-observed-adverse-effect or minimal effect level, regardless of the specific health endpoint against which protection is sought. At least in part, the exposure limits for the 35 substances listed in Table C9–1 are based on data indicating that these limits are already being maintained in work environments and that these levels are both feasible and unlikely to be associated with adverse health effects or signs and symptoms in exposed employees. The proposed limits will also protect against any effects these substances would cause through exposure to higher concentrations. Even at relatively low exposure concentrations, many of the substances in this group cause effects that can be severe and irreversible.

The following discussions describe OSHA's preliminary findings for the substances in this group and illustrate the material impairments of health potentially faced by workers exposed to these toxicants in the construction, maritime, and agriculture industries.

ATRAZINE

CAS: 1912–24–9; Chemical Formula: $C_8H_{14}ClN_5$

H.S. No. 1029

In construction, maritime, and agriculture, OSHA has no limit for atrazine. The ACGIH has a TLV®–TWA of 5 mg/m³; NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed for atrazine in these sectors. OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ for atrazine in the construction, maritime, and agriculture industries. This is the limit recently established for atrazine in general industry.

Atrazine is a white, crystalline compound. This substance is a herbicide for grasses, broadleaf weeds, corn, sorghum, and sugarcane and is also used in industrial and commercial

herbicidal applications (HSDB 1990). Atrazine is the best known of the s-triazine herbicides. When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Atrazine is a mild irritant of the eyes and skin in humans and animals; at acutely toxic doses, this substance causes ataxia, dyspnea, and convulsions in animals (Hayes 1982, p. 562). The LC_{50} in rats is 5200 mg/m³ for 4 hours, the oral LD_{50} in the same species is 672 mg/kg, and the dermal LD_{50} in rabbits is 7500 mg/kg (RTECS 1990). Applied to the skin of rats, atrazine caused marked erythema but no systemic toxicity (Hayes 1982, p. 562). Instilled into the eyes of rabbits, this substance caused severe irritation (RTECS 1990). In a subacute experiment in rats given atrazine by gavage at doses of 100, 200, or 400 mg/kg/day for 14 days or at 600 mg/kg/day for 7 days, a significant and dose-dependent increase in liver weight was seen in all groups; at autopsy, histopathological changes were seen in the livers of all but the 100-mg/kg group (Santa Maria, Moreno, and Lopez-Campos 1987, in J. Appl. Toxicol. 7(6):373–378). Atrazine was embryotoxic when administered to rats in high doses (800–2000 mg/kg) on days 3, 6, and 9 of gestation; no effects were seen at lower concentrations (Clayton and Clayton 1981, p. 2775). A recent study in rats and rabbits (Infurna, Levy, Meng et al. 1988, in J. Toxicol. Environ. Hlth. 24(3):307–320) indicates that atrazine is not teratogenic in these species at doses below those causing maternal toxicity. Atrazine has been tested for carcinogenicity in mice and rats. In mice, 21.5 mg/kg/day was administered by gavage from days 7 to 28 of life, and no increase in the incidence of tumors was seen (Innes et al. 1969, in EPA Health Advisory 1987). A recent study (Pinter, Torok, Surjan et al. 1989, in Magy. Onkol. [Hungarian Oncology] 33(4):211–221), however, found dose-dependent increases in all malignancies in rats of both sexes, benign breast tumors in males, and uterine carcinomas in females, confirming atrazine's carcinogenicity in animals.

In humans, there is one report of severe contact dermatitis in a farmer who sprayed atrazine; patch testing confirmed atrazine's involvement in this episode (Schlichter and Bent 1972, in J. Iowa Med. Soc. 62:419–420). Atrazine has also been found to induce unscheduled DNA synthesis in human cells, which suggests a potential genetic hazard in humans (Loprieno et al. 1980, Mutat. Res. 74(3):2500). The possible role of triazine herbicides in the etiology of ovarian cancer in women was evaluated in a recent case-referent study (Donna, Crosignani, Robutti et al. 1989, Scand. J. Work Environ. Hlth. 15(1):47–53). These authors concluded that previous exposure to triazines was associated with a significant relative risk of 2.7 for ovarian neoplasms.

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ for atrazine in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that this limit will protect employees in these sectors from the significant risk of neuropathic and metabolic effects associated with exposure to atrazine. OSHA considers these effects material health impairments and believes that the proposed PEL is necessary to substantially reduce these significant risks. In addition, promulgation of this PEL will make OSHA's limit for atrazine consistent across all regulated sectors.

AZINPHOS–METHYL

CAS: 86–50–0; Chemical Formula: $C_{10}H_{12}N_3O_3PS_2$

H.S. No. 2014

OSHA's PEL for azinphos methyl in general industry, construction, and maritime workplaces is 0.2 mg/m³ as an 8-hour TWA. OSHA also has a skin notation for this substance. There is no REL in agriculture. The ACGIH TLV®–TWA is 0.2 mg/m³, with a skin notation; NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed for azinphos methyl. OSHA is proposing an 8-hour TWA PEL of 0.2 mg/m³, and a skin notation, for this substance in agriculture. Promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

In pure form, azinphos methyl is a colorless or white crystalline substance, although the technical grade is a brown, waxy solid (Hayes 1982, p. 358). This substance is a widely used nonsystemic insecticide and acaricide (Hayes 1982, p. 358). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Azinphos methyl belongs to the organophosphate class of pesticides. It is an anticholinesterase agent which inhibits cholinesterase, causing acetylcholine to accumulate at synapses in the nervous system, skeletal muscle, and secretory glands. Azinphos methyl exerts this effect by inhalation, percutaneous absorption, or ingestion. The oral LD_{50} in rats is 7 mg/kg, and the LC_{50} in the same species is 69 mg/m³ for 1 hour (RTECS 1990). The dermal LD_{50} in rats is 220 mg/kg (RTECS 1990). Rats fed 20 ppm azinphos methyl in their diets for 60 days showed inhibition of brain and red blood cell cholinesterase (ACGIH 1986, p. 46). Rats inhaling this substance at a concentration of 4.7 mg/m³ for 6 hours/day, 5 days/week for 12 weeks showed both red blood cell and plasma cholinesterase inhibition; inhalation of a 1.24-mg/m³ concentration on the same regimen was without effect (Hayes 1982, pp. 358–359). Azinphos methyl has caused developmental and reproductive effects when administered orally to pregnant rats and mice (RTECS 1990). This substance also is mutagenic in mammalian test systems (RTECS 1990).

Overexposure to azinphos-methyl causes respiratory and ocular effects (including wheezing, a feeling of tightness in the chest, laryngeal spasms, excessive salivation, cyanosis, blurring of vision, lacrimation, runny nose, and frontal headache) (Proctor, Hughes, and Fischman 1988, p. 86). If this substance is accidentally ingested, gastrointestinal symptoms, including nausea, vomiting, cramps, and diarrhea occur within 2 hours of ingestion. Absorption of azinphos methyl through the skin causes localized sweating and muscular fasciculation, indicating localized poisoning (Proctor, Hughes, and Fischman 1988, p. 86).

Regardless of the route of exposure, azinphos methyl produces muscular weakness, twitching, convulsions, coma, loss of reflexes, and paralysis of the respiratory muscles. If the victim does not die during the acute episode, complete recovery may occur within a week, although the individual is likely to remain hypersensitive to anticholinesterase agents for some period of time thereafter (Proctor and Hughes 1978, p. 114). Long-term exposure to concentrations of azinphos methyl that are too small to cause acute symptoms may eventually lead to systemic poisoning (Proctor and Hughes 1978, p. 114).

Based on this evidence, OSHA is proposing an 8-hour TWA PEL for azinphos methyl of 0.2 mg/m³, with a skin notation for those agricultural uses of this substance that are not covered by FIFRA. The Agency believes that, in the absence of a PEL, workers in agricultural establishments may be subject to the significant risk of cholinesterase inhibition posed by exposure to this substance. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

BROMACIL
CAS: 314–40–9; Chemical Formula: $C_9H_{13}BrN_2O_2$
H.S. No. 1041

In construction, maritime, and agriculture, OSHA has no permissible exposure limit for bromacil. The ACGIH's TLV®–TWA for bromacil is 1 ppm. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. The Agency is proposing an 8-hour TWA PEL of 1 ppm for this substance, which is consistent with the PEL recently established for bromacil in general industry.

Bromacil is a white crystalline solid that is used as a herbicide in commercial, industrial, and agricultural applications (HSDB 1989). This substance is odorless (HSDB 1989). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Bromacil is an irritant of the eyes, mucous membranes, and upper respiratory tract. The oral LD_{50} in rats is 641 mg/kg (RTECS 1990). All rats survived inhalation of a bromacil concentration of 4800 mg/m³ for 4 hours (ACGIH 1986, p. 64). The dermal LD_{50} in rabbits is reported to be greater than 5000 mg/kg (Clayton and Clayton 1981, p. 2749). Applied to the skin of guinea pigs, bromacil caused mild to moderate skin irritation (Clayton and Clayton 1981, p. 2749). Instilled into rabbit eyes, this substance caused reversible conjunctival irritation but no corneal damage (Grant 1986, p. 536). Male rats were given bromacil 5 days/week for 2 weeks at oral doses of 650, 1035, or 1500 mg/kg; five of six rats died after five doses at the highest level, one died after 10 doses at 1035 mg/kg, and none died at the lowest dose. At autopsy, focal cell hypertrophy and hyperplasia of the liver were seen (NRC 1977, Drinking Water and Health, p. 540). Rats were fed bromacil at 50, 500, 2500, 5000, 6000, or 7000 ppm for 90 days. Rats in the 5000 ppm or higher groups developed hyperthyroidism (NRC 1977, p. 540). In another study in rats fed bromacil at 50, 250, or 1250 ppm for 2 years, rats in the 1250-ppm group developed hyperplasia of the thyroid; one female rat in the 1250-ppm group developed a follicular adenoma (NRC 1977, p. 541).

In 2-year feeding studies in rats and dogs, no-observed-adverse-effect dietary concentrations were determined to be 12.55 mg/kg/day (1.25 ppm) for rats and 1250 for dogs (Sherman and Kaplan 1975/Ex. 1–572).

Bromacil is known to cause eye, nose, and throat irritation in workers handling formulations containing this substance. Skin irritation has also been reported (HSDB 1990; Hazardous Substance Fact Sheet 1985). No reports of systemic toxicity caused by exposure to bromacil appear in the literature.

Based on this evidence, OSHA is proposing an 8-hour TWA permissible exposure limit of 1 ppm for bromacil in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that this limit will protect employees against the significant risk of thyroid damage and irritation, both material health impairments that occur with exposure to this substance. OSHA believes that the proposed limit is necessary to substantially reduce this risk among workers in these sectors. In addition, promulgation of this PEL will make OSHA's limit for bromacil consistent across all regulated sectors.

p-tert-BUTYLTOLUENE
CAS: 98–51–1; Chemical Formula: $(CH_3)_3C–C_6H_4CH_3$
H.S. No. 1056

OSHA's PEL for p-tert-butyltoluene in construction and maritime is an 8-hour TWA PEL of 10 ppm; the Agency is proposing to retain this limit in these industries, to supplement it with a 20-ppm STEL, and to extend both the 8-hour TWA and the STEL to workplaces in agriculture. The ACGIH has a TLV®–TWA of 10 ppm and a TLV®–STEL of 20 ppm for p-tert-butyltoluene. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. These are the limits recently established for p-tert-butyltoluene in general industry.

p-tert-Butyltoluene is a colorless liquid with an aromatic, gasoline-like odor. This substance is used as a solvent for resins, in the manufacture of pharmaceuticals, and as a chemical intermediate (HSDB 1989).

p-tert-Butyltoluene is an irritant of the eyes, mucous membranes, and upper respiratory tract with acute exposure and a liver and kidney toxin with chronic exposure. The oral LD_{50} in rats is 1800 mg/kg, the LC_{50} in the same species is 165 ppm for 8 hours, and the dermal LD_{50} in rabbits is 19,600 mg/kg (RTECS 1990). In contact with the eyes or skin of rabbits, p-tert-butyltoluene caused mild irritation (RTECS 1990; Grant 1974, p. 212). Acutely poisoned animals showed staggering, difficult breathing, and paralysis before death; some animals showed signs of eye and upper respiratory tract irritation and others convulsed (Proctor, Hughes, and Fischman 1988, p. 110; RTECS 1990). Rats repeatedly exposed to a 50-ppm concentration of p-tert-butyltoluene showed liver and kidney changes and damage to the spinal cord at autopsy (Hine, Ungar, Anderson, et al. 1954/Ex. 1–983). The no-effect level in rats exposed daily for 26 weeks was 25 ppm (Gerarde 1960a, in ACGIH 1986, p. 85/Ex. 1–3).

In humans, nasal irritation, nausea, malaise, headache, and weakness have been seen with exposure to p-tert-butyltoluene at concentrations between 5 and 160 ppm (Hine, Ungar, Anderson et al. 1954/Ex. 1–983). At a concentration of 80 ppm, a moderate degree of eye irritation was reported by these volunteers. These authors also noted cardiovascular effects, as well as effects on the central nervous system, the skin, and the respiratory tract. Half of the subjects exposed to p-tert-butyltoluene developed tremor and anxiety, and 25 percent of exposed individuals showed evidence of chemical contact irritation of the respiratory tract (Hine, Ungar, Anderson et al. 1954/Ex. 1–983).

Based on this evidence, OSHA is proposing to retain its 8-hour TWA of 10 ppm for p-tert-butyltoluene in the construction and maritime industries, to add a STEL of 20 ppm, and to extend both the TWA and STEL to agricultural workplaces. The Agency preliminarily concludes that the STEL and TWA will protect workers in these sectors against the significant risks of irritation and central nervous system effects associated with exposure to this substance. The Agency believes that these adverse health effects constitute material impairments of health and that the proposed limits are necessary to reduce these significant occupational risks substantially. In addition, promulgation of the proposed limits will make OSHA's PELs for p-tert-butyltoluene consistent across all regulated sectors.

CHLORODIFLUOROMETHANE
CAS: 75–45–6; Chemical Formula: $CHClF_2$
H.S. No. 1085

OSHA currently has no limit for chlorodifluoromethane (Freon 22) in the construction, maritime, or agriculture industries. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed by OSHA. The ACGIH has a TLV® of 1000 ppm as a TWA and 1250 ppm as a STEL. The Agency is proposing an 8-hour TWA PEL of 1000 ppm for this substance; this is the limit recently established for chlorodifluoromethane in general industry.

Chlorodifluoromethane is a colorless, nearly odorless, nonflammable gas. This substance is used as an aerosol propellant, a refrigerant, a foam-blowing agent, and a solvent (HSDB 1990).

Exposure to very high atmospheric levels of Freon 22 causes stimulation and then depression of the central nervous system, followed by asphyxiation. The LC_{50} in rats is 350,000 ppm for 15 minutes (RTECS 1990). Rats and guinea pigs exposed to concentrations of 75,000 to 100,000 ppm over a 2-hour period exhibited excitation and disequilibrium; narcosis was observed at 200,000 ppm and mortality at 300,000 and 400,000 ppm (Weigand 1971/Ex. 1–1102). In rabbits, the minimum concentration altering reflex responses was 11,000 to 20,000 ppm (Karpov 1963, as cited in ACGIH 1986/Ex. 1–3, p. 127). Studies in guinea pigs reported that no animals died as a result of exposure for 2 hours to a concentration of 200,000 ppm, but mild clinical changes were observed at 50,000 ppm and minimal effects were seen at 25,000 ppm (Underwriters' Laboratories, Inc. 1940, as cited in ACGIH 1986/Ex. 1–3, p. 127). Karpov (1963, as cited in ACGIH 1986/Ex. 1–3, p. 127) reported the results of a 10-month study on the effects of exposure by inhalation of chlorodifluoromethane in rats, guinea pigs, dogs, and cats who were exposed 6 hours/day, 5 days/week to 2,000 or 14,000 ppm; alterations in weight, endurance, and blood chemistry, and pathology of the lungs, central nervous system, heart, liver, kidney, and spleen were seen in rats, mice, and rabbits exposed to 14,000 ppm. At a concentration of 2000 ppm, rats and mice showed no effects (Karpov 1963, as cited in ACGIH 1986/Ex. 1–3, p. 127). In dogs, cardiac sensitization was not observed at the 25,000-ppm level but did occur at the 50,000-ppm level (Reinhardt, et al. 1971/ Ex. 1–78). Rats and mice were exposed to chlorodifluoromethane at concentrations of 1000 or 5000 ppm for 4 hours/day, 5 days/ week for 104 weeks (rats) or 78 weeks (mice). No carcinogenic responses were seen in animals of either sex or species (Maltoni, Lefemine, Tovoli, et al. 1988, in Annals NY Acad. Sci. 534:261–282). In a study in rats given chlorodifluoromethane orally, however, an increase in tumor incidence was seen, and a marginal increase in the incidence of subcutaneous fibrosarcomas and Zymbal-gland tumors occurred in male rats exposed to a high concentration of chlorodifluoromethane. The International Agency for Research on Cancer concluded in 1987 that the evidence for the carcinogenicity of chlorodifluro-methane in animals was limited (IARC 1987, Suppl. 7, p. 149).

In humans, exposure to chlorodifluoromethane causes central nervous system effects, including dizziness, disorientation, incoordination, nausea, vomiting, and narcosis (Braker and Mossman 1980, p. 170). In contact with the skin, liquid chlorodifluoromethane causes skin irritation and frostbite (Braker and Mossman 1980, p. 170). A study of pathology laboratory workers exposed to high short-term concentrations (300 ppm) of this substance while preparing frozen sections showed an association between episodes of heart palpitations and exposure and also noted that several of these young male workers had chronic arrhythmias (Rom 1983, p. 361). A study of 539 refrigeration workers occupationally exposed to a number of chlorofluorocarbons, including chlorodifluoromethane, for periods ranging from 6 months to 20 years was inconclusive (IARC 1987, Suppl. 7, p. 149).

Based on this evidence, the Agency is proposing an 8-hour TWA limit of 1000 ppm for chlorodifluoromethane in the construction, maritime, and agriculture industries. OSHA preliminarily concludes that this limit will provide protection for workers in these sectors against the central nervous system effects asphyxiant effects, and cardiac sensitization effects, that are associated with exposure to this substance. The Agency believes that this limit is necessary to reduce substantially these significant risks of material health impairment. In addition, promulgation of this PEL will make OSHA's limit for chlorodifluoromethane consistent across all regulated sectors.

O-CHLOROTOLUENE

CAS: 95–49–8; Chemical Formula: C_7H_7Cl

H.S. No. 1090

OSHA has no limit for o-chlorotoluene in the construction, maritime, and agriculture industries. The ACGIH has a TLV®–TWA of 50-ppm and a 75-ppm TLV®–STEL for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing a 50-ppm 8-hour TWA for o-chlorotoluene. This is the limit recently established for this substance in general industry.

O-Chlorotoluene is a colorless liquid. It is used as a solvent and an ingredient in pharmaceuticals, dyes, and rubber formulations (Hazardous Substance Fact Sheet 1985).

O-Chlorotoluene is an irritant of the eyes, mucous membranes, and upper respiratory tract. The oral LD_{50} in rats is greater than 1600 mg/kg (Ely 1971, in EPA 1989). The oral LD_{50} in mice and rats are 4400 and 5700 mg/kg, respectively; acutely poisoned animals showed incoordination, convulsions, and rapid breathing before death (Pis'ko et al. 1981, in EPA 1989). When the undiluted material was administered orally in doses ranging from 50 to 100 mg/kg, rats experienced weakness and vasodilation at the higher dose levels, but all survived and were gaining weight 2 weeks later (ACGIH 1986/Ex. 1–3, p. 137). When the undiluted liquid was applied to the skin of guinea pigs in doses of 1 ml/kg or 10 ml/kg for 24 hours, moderately severe skin irritation occurred at both dose levels. The guinea pigs lost weight over the 2-week period following application, indicating percutaneous absorption of this substance; however, no dermal LD_{50} has been established for o-chlorotoluene. One drop of undiluted material in the eyes of rabbits produced a delayed erythema of the conjunctiva, although this effect cleared after 14 days (Ely 1971, as cited in ACGIH 1986/Ex. 1–3, p. 137). Rats exposed to an atmosphere of 21 mg/L (or about 4000 ppm) for 6 hours exhibited loss of coordination within 1.5 hours, prostration at 1.75 hours, and tremors at 2 hours. At 14,000 ppm, rats showed loss of coordination, vasodilation, labored respiration, narcosis, and eye tearing: however, rats exposed to 4000 or 14,000 ppm survived. At 175,000 ppm, one of three rats died (Ely 1971, as cited in ACGIH 1986/Ex. 1–3, p. 137). Rats given 114 or 570 mg/kg/day o-chlorotoluene by gavage for 2 months showed dose-dependent effects on hemopoiesis, liver, kidneys, and the central nervous system (Pis'ko 1981, in EPA 1989). Sub-chronic studies in animals given oral doses (5, 20, or 80 mg/kg/day in dogs; 20, 80, or 320 mg/kg in rats) of o-chlorotoluene for 97 days (dogs) or 104 days (rats) reported no treatment-related changes in dogs; rats showed decreases in body weight and increases in adrenal weight (two high-dose groups) and in heart and testes weight (in the highest dose group only) (Gibson et al. 1974a and b, in EPA 1989).

Although no poisonings caused by o-chlorotoluene have been reported in humans, this substance is expected to cause irritation and central nervous system effects in exposed individuals on the basis of effects seen in animals.

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 50 ppm for o-chlorotoluene in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that this limit will protect workers in these sectors from the significant risks of eye and skin irritation and systemic poisoning associated with exposure to o-chlorotoluene. OSHA considers these adverse effects material impairments of health and believes that the proposed

PEL is necessary to reduce substantially these risks. In addition, promulgation of the proposed PEL will make OSHA's limit for o-chlorotoluene consistent across all regulated sectors.

CYCLONITE (RDX)

CAS: 121–82–4; Chemical Formula: $C_3H_6N_6O_6$

H.S. No. 1110

OSHA's permissible exposure limit for cyclonite (RDX) in the construction and maritime industries is 10 mg/m³ as an 8-hour TWA and 20 mg/m³ as a STEL. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the proposed limit. The 1987–1988 ACGIH assigned an 8-hour TLV®–TWA limit of 1.5 mg/m³ and a TLV®–STEL of 3 mg/m³, with a skin notation, to this substance. OSHA is proposing a 1.5-mg/m³ 8-hour TWA PEL for cyclonite, and a skin notation; this is the limit recently established for cyclonite in general industry.

Cyclonite is a white, crystalline powder. This substance is used as an explosive and a rat poison. When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Cyclonite is toxic to the central nervous system in humans and animals. The oral LD_{50} in rats is 100 mg/kg (RTECS 1990). Acutely poisoned animals showed labored breathing and convulsions before death (Cholakis, Wong, Van Goethen et al. 1980, in Midwest Research Institute report; Burdette, Cook, and Dyer 1988, Toxicol. Appl. Pharmacol. 92(3):436–444). Rats given oral doses of cyclonite ranging from 0 to 60 mg/kg were monitored for 8 hours after dosing. An increase in the incidence of spontaneous and audiogenic seizures was observed at doses as low as 10 to 12.5 mg/kg, and significant seizure activity was induced by doses of 25 mg/kg and above. The authors of this study concluded that cyclonite is a potent convulsant. Rats fed 40 mg/kg/day for 90 days showed decreased weight gain but no other signs of toxicity, while 17 of 35 rats fed 50 mg/kg/day died (Cholakis et al. 1980; Clayton and Clayton 1981, p. 4197).

In industry, reports of poisonings as a result of occupational exposures to cyclonite were widespread as late as 1962 (Kaplan, Berghout, and Peczenik 1965/Ex. 1–338). Occupational overexposure causes central nervous system effects, including nausea, vomiting, convulsions, and unconsciousness. These clinical signs result from oral and inhalation exposures, and from skin absorption (Kaplan, Berghout, and Peczenik 1965; Sunderman et al. 1944, as cited in ACGIH 1986/Ex. 1–3, p. 162; von Oettingen, Donahue, Yagoda et al. 1949/Ex. 1–398). Convulsions have occurred in cyclonite workers without warning or, in some cases, after 1 or 2 days of insomnia and restlessness. Seizures were followed by amnesia, fatigue, weakness, and malaise, but complete recovery followed (Clayton and Clayton 1981, p. 4197). In an epidemiological study, Hathaway and Buck (1977/Ex. 1–418) reported that 8-hour TWA exposures to cyclonite at concentrations ranging up to 1.57 mg/m³ and averaging 0.28 mg/m³ caused no identifiable exposure-related abnormalities.

Based on this evidence, OSHA is proposing an 8-hour TWA limit of 1.5 mg/m³ TWA and a skin notation for cyclonite in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that these limits are necessary to protect workers in these sectors from the significant risk of these exposure-related central nervous system effects, which constitute material health impairments. In addition, promulgation of the limit will make OSHA's PEL for cyclonite consistent across all regulated sectors.

DIBUTYL PHTHALATE

CAS: 84–74–2; Chemical Formula: $C_6H_4(CO_2C_4H_9)_2$

H.S. No. 2056

In general industry, construction, and maritime, OSHA's permissible exposure limit for dibutyl phthalate is 5 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 5 mg/m³ for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA limit of 5 mg/m³ for dibutyl phthalate in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Dibutyl phthalate is a colorless, oily liquid with a weak aromatic odor. It is used as an insect repellant in clothing and as a plasticizer, elastomer, solvent, textile lubricating agent, and perfume fixative and in paper coatings and explosives (NIOSH/OSHA Occupational Health Guideline 1981, p. 1; Hawley's 1987, p. 372). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Dibutyl phthalate causes primary irritation and central nervous system effects in humans and animals; it also causes reproductive effects in animals. The oral LD_{50} in various species are 8000 mg/kg in rats, 5289 mg/kg in mice, and 10,000 mg/kg in guinea pigs (RTECS 1990). The LC_{50} in mice is 25,000 mg/m³ for 2 hours (RTECS 1990). Mice exposed to a concentration of 250 mg/m³ dibutyl phthalate for 2 hours showed signs of eye and upper respiratory tract irritation; exposure to higher (not further specified) concentrations caused difficult breathing, ataxia, paresis, convulsions, and death due to respiratory paralysis (Varonin 1973, in ACGIH 1986, p. 176). Rats exposed to 0.5 mg/m³ concentrations of dibutyl phthalate mist 6 hours/day for 6 months gained weight more slowly and had greater lung and brain weights at autopsy than controls (Kawano 1980, in Proctor, Hughes, and Fischman 1988, p. 183). Rats given biweekly doses of dibutyl phthalate in oil at 1ml/kg of body weight for 6 weeks showed no adverse effects at autopsy, and a second group of rats maintained on this regimen for 1.5 years also displayed no adverse effects (Bornmann et al. 1956, in ACGIH 1986, p. 176). In female rats given 2520 mg/kg or 12,600 mg/kg dibutyl phthalate on days 1 through 21 of pregnancy, reproductive and embryotoxic effects were seen (RTECS 1990). Several species of animals were fed 2 g/kg of dibutyl phthalate for 10 days; there was evidence of testicular injury in mice and guinea pigs but not in hamsters (Gangolli 1982, in Proctor, Hughes, and Fischman 1988, p. 184). Oral or intraperitoneal administration of dibutyl phthalate to pregnant rats caused increased resorptions, fetal deaths, neural tube defects, and skeletal abnormalities (Peters and Cook 1973; Singh et al. 1972; and Shista and Nishimura 1982, in Proctor, Hughes and Fischman 1988, p. 183).

The lowest oral dose of dibutyl phthalate reported to be toxic in humans is 140 mg/kg (RTECS 1990). Contact of this substance with the eyes causes severe stinging and pain but no permanent injury (Grant 1986, p. 317). A chemical operator who accidentally swallowed 10 grams of dibutyl phthalate experienced nausea, dizziness, photophobia, lacrimation, and conjunctivitis, but promptly recovered (Cagianot 1954, in ACGIH 1986, p. 176). Women working with phthalates in the synthetic leather industry had a higher than normal incidence of miscarriages and menstrual disorders (Aldyreva et al. 1974, in HSDB 1985). Soviet investigators, who studied 147 workers employed in the artificial leather industry (where several phthalates are used) for 0.5 to 19 years reported that these workers complained of pain, numbness, and spasms in the

extremities; these effects were reported by workers employed for more than 6 years. Forty-seven of these workers had developed polyneuritis (Milkov et al. 1973).

Based on this evidence in humans and animals, OSHA preliminarily concludes that dibutyl phthalate causes primary irritation, central nervous system effects, and possible reproductive effects. OSHA believes that, in the absence of a limit for dibutyl phthalate, workers in agriculture are at significant risk of experiencing these exposure-related adverse health effects. The Agency believes that establishing an 8-hour TWA PEL of 5 mg/m³ for dibutyl phthalate is necessary to reduce significantly these risks of material health impairment. Promulgation of this limit for dibutyl phthalate in agriculture will make OSHA's PEL for this substance consistent across all OSHA-regulated sectors.

1,2-DICHLOROETHYLENE
CAS: 540–59–0; Chemical Formula: $C_2H_2Cl_2$
H.S. No. 2059

In general industry, construction, and maritime, OSHA currently has an 8-hour TWA limit of 200 ppm for 1,2-dichloroethylene; there is no PEL for this substance in agriculture. The ACGIH TLV®–TWA for 1,2-dichloroethylene is 200 ppm. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA limit of 200 ppm for 1,2-dichloroethylene in agriculture. This action will make the PEL for this substance consistent across all regulated sectors.

1,2–Dichloroethylene, which is available commercially as a 60:40 mixture of the cis- and trans-isomers, is a colorless liquid with a slightly acrid, ether-like odor (ACGIH 1986, p. 185; Merck 1983, p. 14). It is used as a general solvent for organic materials, for dye extraction, and in perfumes, lacquers, and thermoplastics; it is also used in organic synthesis (Hawley's 1987, p. 378).

1,2-Dichloroethylene causes eye irritation and central nervous system depression in humans and animals. The oral LD_{50} in rats is 770 mg/kg (RTECS 1990). After 8 minutes of exposure to a 16,000-ppm concentration of the cis-isomer, rats became anesthetized; 4 hours of exposure at the same concentration resulted in death. The trans-isomer proved to be twice as potent (Smyth 1956, in Proctor, Hughes, and Fischman 1988, p. 189). Dogs developed reversible superficial corneal opacities when exposed to high vapor concentrations (not further specified) (Grant 1986, pp. 325–326). Animals of four species (rats, rabbits, guinea pigs, and dogs) exposed to 1,2-dichloroethylene for 7 hours/day, 5 days/week for 6 months at 500 or 1000 ppm showed no significant changes in growth, mortality, clinical chemistry, or gross or microscopic examination (Torkelsen 1965, in ACGIH 1986, p. 185). Another study, however, did report effects in rats exposed chronically to a 200-ppm concentration of dichloroethylene (trans-isomer); the liver, lungs, and leukocyte counts of these animals were affected (Clayton and Clayton 1981, p. 3552).

In humans, 1,2-dichloroethylene formerly found use as a general anesthetic (Proctor, Hughes, and Fischman 1988, p. 189). Symptoms of vertigo, nausea, and burning of the eyes were caused in subjects exposed to the trans-isomer at a concentration of 2200 ppm. Prolonged or repeated contact of the skin with dichloroethylene causes defatting and dermatitis (HSDB 1990). Von Oettingen also reported a fatality caused by exposure to very high vapor concentrations (not further specified) in the workplace [von Oettingen 1955, in Proctor, Hughes, and Fischman 1988, p. 189).

Based on this evidence in humans and animals, OSHA preliminarily concludes that 1,2-dichloroethylene causes eye irritation and central nervous system depression in exposed workers and that, in the absence of a PEL, agricultural workers are at significant risk of experiencing these effects. The Agency believes that establishing an 8-hour TWA limit of 200 ppm will substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

2,6-di-tert-BUTYL-p-CRESOL
CAS: 128–37–0; Chemical Formula: $C_{15}H_{24}O$
H.S. No. 1117

OSHA has no limit for 2,6-di-tert-butyl-p-cresol (DBPD) in the construction, maritime, or agriculture industries. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed by OSHA. The ACGIH has a TLV®–TWA of 10 mg/m³ for DBPD. OSHA is proposing an 8-hour TWA PEL of 10 mg/m³ for this substance in construction, maritime, and agriculture. This is the limit recently established for DBPD in general industry.

DBPD is a white crystalline compound that is prepared from p-cresol and isobutylene. DBPD is widely used as a food preservative, a gasoline additive, and an ingredient in lubricating oils, waxes, rubbers, paints, plastics, and elastomers (ACGIH 1986, p. 227).

DBPD is an irritant of the eyes and mucous membranes; in animals, exposure to this substance has also caused lung and liver injury. The oral LD_{50} ranges from 0.89 g/kg in rats to 10.7 g/kg in guinea pigs (RTECS 1990). Applied to the skin of rabbits, DBPD caused moderate skin irritation, and instillation into the eyes of these animals also caused a moderate degree of eye irritation (RTECS 1990). One year of daily oral administration of 0.17 to 0.9 g/kg produced no effects in dogs, and 2 years of daily oral administration of 0.2, 0.5, or 0.8 percent DBPD also had no effects in rats (Deichmann, Clemmer, Rakoczy, Bianchine et al. 1955/Ex. 1–505). Some growth rate decreases and liver weight increases were demonstrated in rats fed 0.01 to 0.5 percent DBPD (total daily diet) for periods ranging from 12 days to 80 weeks (Brown, Johnson, and O'Halloran 1959/Ex. 1–621; Creaven, Davies, and Williams 1966/Ex. 1–547). These animals also exhibited reversible activation of microsomal liver enzymes. One study in pregnant rats fed DBPD showed teratogenic effects in the offspring, but other research has failed to confirm these results (Grant 1986, p. 164). Some carcinogenicity studies have suggested that DBPD may have a promoting effect; other bioassays have shown no carcinogenic effects. Rats and mice fed DBPD at doses of 3000 or 6000 ppm for 105 to 108 weeks did not show an increased tumor incidence compared with controls (NCI 1979, in HSDB 1989). Administration of DBPD following dosing with a known carcinogen (acetylaminofluorene or urethane) caused an increased incidence of tumors (hepatomas and pulmonary adenomas) (Klaassen, Amdur, and Doull 1986, p. 134). Another study in rats given DBDP after acetylaminofluorene reports that these animals developed bladder cancer (Klaassen, Amdur, and Doull 1986, p. 1118). The no-effect dietary level for DBPD in rats is 25 mg/kg (Gilbert and Golberg 1965/Ex. 1–902).

In humans, exposure to di-tert-butyl-p-cresol causes skin irritation and may cause dermal sensitization (Klaassen, Amdur, and Doull 1986, p. 65). A 48-hour test in human subjects showed that the irritant ocular dose for this period was 500 mg of DBDP (RTECS) 1990). No other information on DBDP's effects in humans is available.

Based on this evidence, OSHA is proposing an 8-hour TWA limit of 10 mg/m³ for 2,6-di-tert-butyl-p-cresol. The Agency preliminarily concludes that this limit will protect workers in

construction, maritime, and agriculture against a significant risk of sensory irritation, which is a material health impairment. OSHA believes that the proposed limit is necessary to reduce this significant risk substantially. In addition, promulgation of the proposed limit will make the PEL for DBPD consistent across all regulated sectors.

DIETHANOLAMINE

CAS: 111–42–2; Chemical Formula: $HO(CH_2)_2NH(CH_2)_2OH$

H.S. No. 1134

OSHA has no limit for diethanolamine in the construction, maritime, or agriculture industries. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. The ACGIH has an 8-hour TLV®–TWA of 3 ppm for this substance. OSHA is proposing an 8-hour TWA PEL of 3 ppm for diethanolamine; this is the limit recently established in general industry.

Diethanolamine is a solid or a liquid, depending on temperature; it has an ammonia-like odor. Diethanolamine is used to make cosmetics, cleaners, detergents, emulsifiers, dispensing agents, and alkalizing agents (Proctor, Hughes, and Fischman 1988, p. 198).

Diethanolamine is an irritant on acute exposure and a liver and kidney toxin on chronic exposure. The oral LD_{50} in rats is 710 mg/kg, and the dermal LD_{50} in rabbits is 12,200 mg/kg (RTECS 1990). Instilled into rabbit eyes, diethanolamine caused severe irritation, although skin contact caused only mild irritation in the same species (RTECS 1990). Rats given 100 or 500 mg/kg by interperitoneal injection showed cytoplasmic vacuolization of the liver and kidneys and, at the high dose, tubular degeneration of the kidneys (Grice et al. 1971, in Food Cosmet. Toxicol. 9:847). Rats fed diethanolamine at a dose of 0.17 g/kg for 90 days showed cloudy swelling and degeneration of the kidney tubules and fatty degeneration of the liver (Proctor, Hughes, and Fischman 1988, p. 198; Smyth, Carpenter, and Weil 1951/Ex. 1–439). Dietary studies in rats showed no ill effects after 90 days of feeding at 20 mg/kg/day (Smyth, Carpenter, and Weil 1951/Ex. 1–439).

In humans, diethanolamine has caused mild eye and nose irritation (HSDB 1989). Splashed into the eye, this substance may cause severe pain, irritation, and corneal damage (Rumack Poisindex). In contact with the skin, moderate irritation may develop (Rumack Poisindex).

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 3 ppm for diethanolamine. The Agency preliminarily concludes that this limit will protect workers in construction, maritime, and agriculture from the significant risks of eye damage and skin irritation associated with exposure to this substance. OSHA considers these effects material health impairments and believes that the proposed limit is necessary to reduce these risks substantially. In addition, promulgation of this limit will make the PEL for diethanolamine consistent across all regulated sectors.

DIETHYL PHTHALATE

CAS: 84–66–2; Chemical Formula: $C_6H_4(COOC_2H_5)_2$

H.S. No. 1136

OSHA has no limit for diethyl phthalate in the construction, maritime, or agriculture industries. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the proposed limit. The ACGIH has a TLV®–TWA of 5 mg/m³ for this substance, and OSHA is proposing 5 mg/m³ as an 8-hour TWA. This is the limit recently established for diethyl phthalate in general industry.

Diethyl phthalate is an oily colorless liquid with no odor. It is used in insecticidal sprays, as a mosquito repellant, a solvent, a plasticizer, a wetting agent, a camphor substitute, and as an alcohol denaturant (ACGIH 1986, p. 200; Genium MSDS 1987, No. 435; Hawley's 1987, p. 394). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Diethyl phthalate causes irritation of the eyes, skin, and mucous membranes and central nervous system effects. The oral LD_{50} in rats is 8600 mg/kg (RTECS 1990). Diethyl phthalate is readily absorbed through the skin; about one-fourth of a 30 to 40 mg/kg dose of radiolabelled diethyl phthalate applied to the skin of rats appeared in the urine of exposed rats 24 hours after application of the dose (Elsis et al. 1989). Guinea pigs exposed to diethyl phthalate at a concentration of 511 ppm for 6 hours showed signs of mild skin irritation (Clayton and Clayton 1982, p. 2347). Cats exposed to 10 mg/m³ for 5 hours showed signs of nasal irritation (Clayton and Clayton 1982, p. 2347). Rats fed diethyl phthalate showed decreases in testosterone concentrations and testicular atrophy (Oishi and Hiraga 1980, in Toxicol. Appl. Pharmacol. 53(1):35–41). In several species of experimental animals, central nervous system depression is a common effect of intraperitoneal and intravenous administration of diethyl phthalate (Cralley 1966, in J. Pharmaceut. Sci. 55:158–162). The results of reproductive studies conducted on diethyl phthalate have been inconclusive. In one study, pregnant Sprague-Dawley rats were given intraperitoneal injections of diethyl phthalate on days 5, 10, and 15 of gestation at doses of 0.506, 1.012 or 1.686 ml/kg. Some fetal resorptions occurred among rats given the low or high dose, but not among rats given the middle dose. All three dose levels produced skeletal malformations in offspring; no other abnormalities were noted (Singh et al., J. Pharmaceut. Sci. 61:51–55, as cited in HSDB 1986). Another study found no adverse effects on fetal growth, viability, or development among CD rats fed 198, 1909, or 3214 mg/kg per day during days 6 through 15 of gestation (Price et al. 1989); however an increased incidence of extra ribs occurred among offspring of rats given maternally toxic doses of diethyl phthalate.

In humans, exposure to diethyl phthalate causes sensory irritation and central nervous system effects. The lowest toxic inhalation concentration in humans is 1000 mg/m³ (RTECS 1990). A study of workers exposed to a mixture of diethyl phthalate, dibutyl phthalate, and di-2-ethyl hexyl phthalate vapors in air at concentrations of 8 to 53 mg/m³ resulted in findings of no phthalates in the blood (before or after the exposure) and no peripheral polyneuritis (Raleigh, personal communication, as cited in ACGIH 1986/Ex. 1–3, p. 200). Fassett (1963a, as cited in ACGIH 1986/Ex. 1–3 p. 200) reported transient nasal and throat irritation produced by exposure to the heated vapors of diethyl phthalate, but no cumulative effects have been noted. A Soviet study of workers (employed between 0.5 to 19 years) who were exposed to several phthalate plasticizers (e.g., butyl phthalate, the higher aryl phthalates, dioctyl phthalate, and benzyl butyl phthalate), as well as to sebacates, adipates, and tri-o-cresyl phosphate at concentrations ranging from 1.7 to 66 mg/m³, reported that there were complaints of pain, numbness, and spasms in the upper and lower extremities. These complaints were related to the duration of exposure and usually began after the sixth or seventh year of employment (Milkov, Aldyreva, Popova et al. 1973/Ex. 1–646). These investigators reported polyneuritis in 32 percent of the 47 persons examined for this health effect; of 81 persons evaluated for vestibular dysfunction, 78 percent showed depression of vestibular receptors (Milkov, Aldyreva, Popova et al. 1973/Ex. 1–646).

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ for diethyl phthalate. The Agency

preliminarily concludes that this limit will protect workers in construction, maritime, and agriculture against the significant risks of polyneuritis and vestibular dysfunction that are potentially associated with occupational exposure to this substance. Promulgation of this limit will substantially reduce these significant risks and make OSHA's PEL for this substance consistent across all regulated sectors.

DINITOLMIDE (3,5–DINITRO–O–TOLUAMIDE)

CAS: 148–01–6; Chemical Formula: $C_8H_7N_3O_5$

H.S. No. 1144

OSHA has no limit for dinitolmide in construction, maritime, or agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the selection of this PEL. The ACGIH has a TLV®–TWA of 5 mg/m³ for this substance, and OSHA is proposing 5 mg/m³ as an 8-hour TWA PEL. This is the limit recently established for this substance in general industry.

Dinitolmide, which is also called 3,5-dinitro-o-toluamide, is a yellowish solid. This substance is used as a coccidiostat and a food additive (ACGIH 1986, p. 218; Hawley's 1986, p. 1262). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Dinitolmide causes liver and blood effects in animals. The oral LD_{50} in rats is 600 mg/kg (RTECS 1990). In rats fed 6 mg/kg/day dinitolmide for 2 years, increases in liver weight occurred in females, and fatty changes were observed in the livers of both males and females at autopsy (Dow Chemical Company 1973e, as cited in ACGIH 1986, Ex. 1–3, p. 213). No adverse effects were seen either in rats fed 3 mg/kg/day for 2 years or in dogs fed on the same regimen at a dose of 10 mg/kg/day (Dow Chemical Company 1973e, in ACGIH 1986/Ex. 1–3, p. 213). Rats exposed to 150 mg/kg developed methemoglobinemia (Gosselin, Smith, and Hodge 1984, p. II–389). Dinitolmide is mutagenic in bacterial test systems (RTECS 1990).

No systemic effects of dinitolmide exposure have been reported in humans. Allergic contact dermatitis has been reported in workers handling animal feed that contained dinitolmide (Fischer 1975, in Cutis 2:701–702).

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ for dinitolmide. The Agency preliminarily concludes that this limit will protect workers in construction, maritime, and agriculture industries from the significant risk of dermatitis and systemic changes that are potentially associated with exposure to this substance. OSHA considers these effects material health impairments and believes that the proposed limit is necessary to reduce these risks substantially. In addition, promulgation of this limit will make the PEL for dinitolmide consistent across all OSHA-regulated sectors.

DIPHENYLAMINE

CAS: 122–39–4; Chemical Formula: $(C_6H_5)_2NH$

H.S. No. 1147

OSHA's limit for diphenylamine in construction, maritime, and general industry is 10 mg/m³ as an 8-hour TWA. There is no limit for this substance in agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the proposed limit. The ACGIH recommends a TLV®–TWA of 10 mg/m³ for this substance, and OSHA is proposing 10 mg/m³ as an 8-hour TWA for diphenylamine in agriculture. Promulgation of this PEL will make OSHA's limit for this substance consistent across all regulated sectors.

Diphenylamine is a colorless solid that takes the form of monoclinic leaflets that discolor when exposed to light; it has a floral-like odor. Diphenylamine is used as a stabilizer for plastics, nitrocellulose explosives, and celluloids, and in the manufacture of pesticides, pharmaceuticals, and dyes. It is also used in antioxidants (ACGIH 1986, p. 220; Sittig 1985, p. 1885). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Diphenylamine is a liver and kidney toxin and has caused adverse developmental effects in animals. The oral LD_{50} in guinea pigs is 300 mg/kg (RTECS 1990). This substance is known to be absorbed through the skin, but no dermal toxicity data are available. Animals acutely poisoned by oral administration develop anorexia, diarrhea, excitability, hypothermia, emaciation, and generalized weakness before death, which may be delayed for 2 to 3 weeks (Gosselin, Smith, and Hodge 1984, p. II–209). In cats, acute poisoning is associated with methemoglobinemia. Animals exposed to diphenylamine dust at unspecified concentrations showed changes in the liver, spleen, and kidneys at autopsy (Robert et al. 1937, in ACGIH 1986, p. 220). Dietary studies of rats fed 0.025, 0.1, 0.5, 1.0, or 1.5 percent diphenylamine for 226 days demonstrated nonmalignant renal cysts in animals fed at the three highest doses (Thomas, Cox, and Deeds 1957/Ex. 1–873). Rats fed diphenylamine crystals encapsulated in collodion developed bladder papillomas within 125 days (Yoshida, Shimauchi, and Kin 1941, as cited in ACGIH 1986/Ex. 1–3, p. 220). Mice gavaged with diphenylamine showed kidney damage involving the tubules and glomeruli; liver effects were also seen (Kronevi and Holmberg 1979, in Exp. Pathol. (Jena) 17(2):77–81). Fed to pregnant rats at a dose of 7500 mg/kg on days 17 through 22 of pregnancy, diphenylamine caused urogenital system abnormalities (polycystic kidneys) in the offspring (Klaassen, Amdur, and Doull 1986, p. 326).

In humans, exposure to diphenylamine may cause eye and mucous membrane irritation, dermatitis, bladder symptoms, and an increase in heart rate (Hazardous Substance Fact Sheet 1985). Acute overexposure may also cause methemoglobinemia (Genium MSDS 1989). A report of industrial diphenylamine poisoning in France described bladder symptoms, tachycardia, hypertension, and eczema in overexposed workers (Fairhall 1957g, as cited in ACGIH 1986/Ex. 1–3, p. 220). Diphenylamine has been linked in epidemiological studies (Orjelick 1975; in Intl. Occ. Hlth. Safety 44(5):46–47; Parkes and Evans 1984, in C.E. Searle, ed. Chemical Carcinogens, 2nd ed.) to the development of bladder cancer in exposed workers, although contamination by 2-naphthylamine, a recognized human carcinogen, may have been the cause of these tumors.

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 10 mg/m³ for diphenylamine in agriculture. The Agency preliminarily concludes that this limit will protect workers in this sector against the significant risks of liver, kidney, cardiovascular, and other systemic effects, all of which constitute material health impairments that are potentially associated with exposures to this substance. Because the proposed limit is identical to the PEL recently established for diphenylamine in general industry and currently in effect in construction and maritime, promulgation of the proposed PEL in agriculture will make OSHA's limit for diphenylamine consistent across all regulated sectors.

DIURON

CAS: 330–54–1; Chemical Formula: $C_9H_{10}Cl_2N_2O$

H.S. No. 1153

OSHA has no limit for diuron in construction, maritime, or agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being

proposed. The ACGIH has a TLV®–TWA of 10 mg/m³ for diuron. OSHA is proposing an 8-hour TWA PEL of 10 mg/m³ for diuron; this is the limit recently established for this substance in general industry.

Diuron is a white, crystalline, odorless substance. Diuron is a pre-emergence herbicide that is widely used as an industrial area weed control agent, an herbicide for railroad rights of way, and an agricultural herbicide (HSDB 1990). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Diuron is an irritant of the skin and mucous membranes. In animals, chronic exposure may cause growth retardation, anemia, and enlargement of the spleen and liver. This substance may also be teratogenic in animals. The oral LD_{50} in rats is 1017 mg/kg (RTECS 1990). The dermal LD_{50} in rabbits is greater than 2500 mg/kg (Taylor 1976b, in EPA 1987). Acutely poisoned animals showed signs of central nervous system depression before death (EPA 1987: Health Advisory). Applied to the abraded skin of guinea pigs, diuron caused moderate irritation (DuPont n.d., in ACGIH 1986, p. 228). Instilled into the eyes of rabbits, however, diuron caused no irritation (Larson and Schaefer 1976, in EPA 1987). Rats fed 50 to 5000 ppm diuron for 90 days showed the following effects: no changes at 50 ppm, reduced weight gain in females only at 500 ppm, and reduced body weight, enlarged spleens, and chronic methemoglobinemia in rats of both sexes (Hodge et al. 1967). Rats fed 25 to 2500 ppm diuron for 2 years showed the following effects: no effects at 25 ppm, abnormal blood pigment in some animals at 125 ppm, and hematological changes, growth depression, hemosiderosis of the spleen, and increased mortality at 2500 ppm (Hodge et al. 1946b, 1967). Administered to pregnant rats on days 6 through 15 of gestation, diuron caused a statistically significant increase in the incidence of wavy ribs and delayed ossification in the offspring at doses of 250 mg/kg or greater (Khera et al. 1979). In another experiment, administration of diuron to pregnant rats on days 15 and 22 of pregnancy caused reductions in maternal body weight and in mean fetal weights and an increase in the number of anomalous fetuses (Khera et al. 1979). In 2-year feeding studies in rats and dogs, the no-effect levels were reported to be 250 and 125 ppm, respectively. A diuron concentration of 125 ppm in the diet did not cause reproductive or carcinogenic effects in a three-generational study of rats (Hodge, Downs, Panner et al. 1967/Ex. 1–911; Hodge, Downs, Smith et al. 1968/Ex. 1–912); a dietary level of 1400 ppm did not have carcinogenic effects in mice (Innes, Ulland, Valerio et al. 1969/Ex. 1–270).

In humans, exposure to diuron causes irritation of the eyes, nose, and skin (Hayes 1982, p. 541). A woman who intentionally ingested an herbicidal formulation that contained 46 percent diuron and 30% amitrole survived and had no sequelae (Hayes 1982, p. 154).

Based on this evidence in humans and animals, OSHA is proposing an 8-hour TWA limit of 10 mg/m³ for diuron. OSHA preliminarily concludes that this limit will protect workers in construction, maritime, and agriculture from the significant risks potentially associated with occupational exposure to this substance at the levels permitted by the absence of an OSHA PEL. These risks include both skin and respiratory tract irritation, which constitute material impairments of health. The Agency believes that the proposed limit is necessary to reduce these risks substantially. In addition, promulgation of the proposed PEL will make OSHA's limit for diuron consistent across all OSHA-regulated sectors.

ENDRIN

CAS: 72–20–8; Chemical Formula: $C_{12}H_8Cl_6O$

H.S. No. 2073

In general industry, construction, and maritime, OSHA's current permissible exposure limit for endrin is an 8-hour TWA of 0.1 mg/m³, with a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. OSHA has no PEL for endrin in agriculture. The 1987–1988 ACGIH TLV®–TWA for this substance is 0.1 mg/m³, with a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the PEL being proposed. OSHA is proposing a PEL of 0.1 mg/m³, and a skin notation, for endrin in agriculture. Promulgation of this limit will make the PEL for endrin consistent across all OSHA-regulated sectors.

Endrin, which is the stereoisomer of dieldrin, is a white, non-flammable, crystalline powder. Endrin was formerly used as an insecticide, avicide, and rodenticide. Its manufacture and use has been discontinued in the United States (ACGIH 1986, p. 231; Proctor, Hughes, and Fischman 1988, p. 222). OSHA is proposing a limit for endrin in agriculture both to ensure consistency in PELs across sectors and to provide protection for workers in agriculture in the unlikely event that this substance is once again permitted for use as a pesticide.

Endrin causes convulsions in humans and reproductive and developmental effects in animals. The oral LD_{50}s in rats, mice, and monkeys are 3 mg/kg, 1.37 mg/kg, and 3 mg/kg, respectively (RTECS 1990). The dermal LD_{50} in rabbits is 60 mg/kg (RTECS 1990). Following a 1-hour inhalation exposure to approximately 2000 mg/m³ of endrin, 3 of 10 rats died (Anderson et al. 1953; Hine, Ungar, Anderson et al. 1954, in ACGIH 1986, p. 231). Rats and mice inhaling sublimed endrin at a concentration of 0.36 ppm (15 mg/m³) for 7 hours/day, 5 days/week for 26 weeks exhibited no signs of intoxication or growth retardation; however, two of four rabbits died under these exposure conditions (Treon et al. 1955, in ACGIH 1986, p. 231). Rats given oral doses of 2.32 mg/kg of endrin on the fourth day of pregnancy showed signs of reproductive effects, and the offspring had developmental abnormalities (RTECS 1990). Chernoff et al. reported an increased incidence of fused ribs and meningoencephaloceles among the young of hamsters given a single dose of up to 10 mg/kg endrin on day 8 of pregnancy; no deaths were reported in dams or young (Chernoff 1979, in Hayes 1982, p. 248). An oral dose of 7 mg/kg endrin on day 8 of pregnancy caused reproductive effects in the dams and embryotoxic and developmental effects in the offspring (RTECS 1990).

In humans, endrin intoxication often causes an epileptiform convulsion that occurs from 30 minutes to 10 hours after exposure, lasts for several minutes, and is followed by stupor (Jager 1970; Coble et al. 1967, in Proctor, Hughes, and Fischman 1988, p. 222). The signs and symptoms of endrin intoxication in humans include headache, dizziness, leg weakness, nausea, insomnia, and, occasionally, slight mental confusion (Jager 1970, in Proctor, Hughes, and Fischman 1988, p. 222; Hayes 1982, pp. 247–251). An oral dose in the range of 0.2 to 0.25 mg/kg produces convulsions in humans (Diechmann 1973, in ACGIH 1986, p. 231). In cases of severe endrin poisoning, repeated violent convulsions can occur (Hayes 1982, p. 249). Recovery is rapid in most cases, but symptoms such as headache, dizziness, lethargy, weakness, and anorexia may persist for 2 to 4 weeks (Coble et al. 1967, in Proctor, Hughes, and Fischman 1988, p. 222). Brain stem injury was evident in the encephalograms of 20 percent of endrin manufacturing and formulating workers; 3.3 percent of these workers had experienced epileptiform seizures (NRC 1977, in HSDB 1985). In England,

endrin poisoning occurred in more than 100 people who had eaten bread made from flour contaminated with this substance. One to 3 hours after ingestion, the most affected patients lost consciousness; other victims experienced convulsions, violent muscular contractions, and periods of unconsciousness. All patients were ill for several days but subsequently recovered (Gosselin, Smith, and Hodge 1984, p. 285).

Based on this evidence in humans and animals, OSHA preliminarily concludes that endrin causes convulsions and central nervous system effects in exposed individuals. OSHA preliminarily finds that, in the absence of a limit for endrin, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed limit of 0.1 mg/m^3 as an 8-hour TWA, and the skin notation, will substantially reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

L.P.G. (Liquefied Petroleum Gas)
CAS: 68476–85–7; Chemical Formula:
Mixture of C_3H_8 and C_4H_{10}
H.S. No. 2099

The OSHA PEL for liquefied petroleum gas (L.P.G.) in general industry, construction, and maritime is 1000 ppm as an 8-hour TWA. The Agency has no PEL for L.P.G. in agriculture. The ACGIH TLV®–TWA for this substance is 1000 ppm. NIOSH has no REL for L.P.G. but concurs with the PEL being proposed (Ex. 8–47, Table N3A). OSHA is proposing a PEL of 1000 ppm as an 8-hour TWA for L.P.G. in agriculture. Promulgation of this limit will make the PEL for L.P.G. consistent across all OSHA-regulated sectors.

L.P.G. is a mixture of propane and butane and is a flammable, explosive, colorless gas (or liquid under pressure). L.P.G. is a byproduct of petroleum refining. It may contain isomers and often has a foul-smelling odorant added to it to increase its odor warning properties. It is used primarily as a fuel, refrigerant, and propellant and in organic synthesis. L.P.G. is also used in the flame control of weeds and in metal cutting (ACGIH 1986, p. 350; Hazardous Substance Fact Sheet 1986, p. 6; HSDB 1986, "Butane;" HSDB 1988, "Propane").

L.P.G. is non-toxic at low concentrations; at very high concentrations, it causes central nervous system depression and, if exposures are high enough, asphyxiation. Cats exposed to L.P.G. at an 89-percent concentration (890,000 ppm) experienced a depression in blood pressure (HSDB 1988, "Propane"). Guinea pigs exposed to a 2.4 to 5.5 percent (24,000 to 55,000 ppm) concentration of propane (the major component of L.P.G.) showed dyspnea at the lower concentrations and tremor, nausea, retching, and stupefaction at the higher concentration (HSDB 1988, "Propane"). L.P.G. does not cause eye irritation or damage, even when injected into the anterior chamber of the rabbit eye (Grant 1986, p. 207). Pregnant mice exposed to L.P.G. at a concentration of 5 to 8 percent (50,000 to 80,000 ppm) for 1 hour on the eighth day of gestation delivered hydrocephalic offspring (HSDB 1988, "Propane").

In humans, the inhalation of a 10,000-ppm concentration of butane (a major component of L.P.G.) for 10 minutes may cause drowsiness (Patty and Yant 1929, in Clayton and Clayton 1981, p. 3182). Five workers exposed to propane, another major component of L.P.G., experienced headache, numbness, vomiting, and a feeling of being chilled (HSDB 1988, "Propane"). Acute exposure to propane at a 250-, 500-, or 1000-ppm concentration for 1 minute to 8 hours caused no effects in volunteers (HSDB 1988, "Propane"). Contact of the skin with pressurized (liquefied) L.P.G. may cause frostbite and skin burns (Clayton and Clayton 1981, p. 3182).

Based on this evidence, OSHA is proposing a 1000-ppm 8-hour TWA limit to protect workers in agriculture from the significant risk of central nervous system depression potentially caused by exposure to L.P.G. at the concentrations possible in the absence of an OSHA PEL. The Agency preliminarily concludes that this limit is necessary to reduce substantially a significant risk of material health impairment in exposed agricultural workers. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

METHYL ACETATE
CAS: 79–20–9; Chemical Formula:
CH_3COOCH_3
H.S. No. 1249

OSHA's current 8-hour TWA limit for methyl acetate in construction and maritime is 200 ppm; there is no PEL in agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limits being proposed. The ACGIH has an 8-hour TLV®–TWA of 200 ppm and a TLV®–STEL of 250 ppm for methyl acetate. OSHA is retaining the 8-hour TWA PEL of 200 ppm and proposing to add a 15-minute STEL of 250 ppm in construction and maritime and is also proposing to extend these limits to agriculture. These are the limits recently established for methyl acetate in general industry.

Methyl acetate is a highly volatile, colorless liquid with a pleasant, fruity odor. This substance is used as a solvent in lacquers and paint removers and as an ingredient in pharmaceuticals. Methyl acetate is also used in agriculture to destroy cocklebur seeds (Clayton and Clayton 1981, p. 2271).

Methyl acetate is an irritant of the eyes, mucous membranes, and respiratory tract; at high concentrations, it is a narcotic. The oral LD_{50} in rats is 5450 mg/kg, and the lowest lethal concentration in the same species is 32,000 ppm for 4 hours (RTECS 1990). Applied to the skin or eyes of rabbits, methyl acetate caused a moderate degree of irritation (RTECS 1990). Cats exposed to a 5000-ppm concentration of methyl acetate for 20 minutes showed signs of eye irritation; exposure to 10,000 ppm for 10 hours caused narcotic effects, and exposure to 18,480 ppm for 4 to 4.5 hours caused dyspnea, convulsions, vomiting, and narcosis (Clayton and Clayton 1981, p. 2272). Cats exposed for 6 hours/day for 8 days to a methyl acetate concentration of 6600 ppm lost weight, became weak, and recovered from the exposure slowly (Clayton and Clayton 1981, p. 2272).

In humans, exposure to methyl acetate at a concentration of 15,000 mg/m^3 (approximately 5000 ppm) causes eye and upper respiratory tract irritation; when the concentration is increased to 10,000 ppm, the irritation persists after exposure ceases (Clayton and Clayton 1981, p. 2272). A worker who inhaled an unspecified concentration of methyl acetate developed dizziness, headache, and other signs of central nervous system depression, as well as blindness caused by atrophy of the optic nerve (Lund 1944, in J. Ind. Hyg. Toxicol. 28:35). The atrophy may have been caused by hydrolysis of the methyl acetate to methanol (AIHA 1964). Long-term contact of this substance with the skin causes defatting and dermatitis (Proctor, Hughes, and Fischman 1988, p. 317.

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 200 ppm TWA and a 15-minute STEL of 250 ppm for methyl acetate in the construction, maritime, and agriculture industries. OSHA believes that the proposed limits are necessary to reduce substantially the significant risk of eye, skin, and respiratory tract irritation, and of narcosis, that is associated with exposure to methyl acetate. The Agency considers these adverse effects material impairments of health. Promulgation of these limits will also make OSHA's

PELs for methyl acetate consistent across all regulated sectors.

METRIBUZIN

CAS: 21087–64–9; Chemical Formula: $C_8H_{14}N_4OS$

H.S. No. 1275

OSHA has no limit for metribuzin in the construction, maritime, and agriculture industries. The ACGIH has a TLV®–TWA of 5 mg/m³ for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ for metribuzin. This is the limit recently established for this substance in general industry.

Metribuzin is a crystalline solid that has a slight musty odor and is used as a selective herbicide. When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Metribuzin is a mild irritant of the eyes and skin in experimental animals. The oral LD_{50} in rats is 1100 mg/kg, and the dermal LD_{50} in the same species is 2 g/kg (RTECS 1990). The LC_{50} in rats for a wettable granular formulation of metribuzin (concentration not specified) is greater than 20 mg/liter for 1 hour (Mobay MSDS 1985). In contact with the eyes or skin of rabbits, metribuzin caused a mild degree of irritation (Mobay MSDS 1985; EPA 1987, ODW Health Advisory). Rats were fed metribuzin for 3 months at doses of 2.5, 7.5, 25, or 75 mg/kg/day; enlarged thyroid glands were seen in the two high-dose groups, and females in the three highest dose groups also had enlarged livers (Loser et al. 1969, in EPA 1987). Female mice were fed metribuzin for 24 months at doses of 30, 120, or 480 mg/kg/day; those in the high-dose group developed increased liver and kidney weights (Hayes et al. 1981, in EPA 1987). No sensitizing effects were seen in skin tests of guinea pigs, and a skin application of the 70-percent wettable powder of 1000 mg/kg per day for three weeks produced no effects in rats (*Deutsche Forschungsgemeinschaft* 1981, as cited in ACGIH 1986/Ex. 1–3, p. 411). A no-effect level of 100 ppm was observed in a two-year dietary study of rats and dogs (*Deutsche Forschungsgemeinschaft* 1981, as cited in ACGIH 1986/Ex. 1–3, p. 411); these same investigators observed no teratogenic, embryotoxic, or reproductive effects in rats or rabbits. In a subacute inhalation study in rats, exposure to a concentration of 31 mg/m³ caused no observed effects (ACGIH 1986, p. 411, Ex. 1–3). In Chinese hamsters and mice, no mutagenic activity was observed (Siebert and Lemperle 1974/Ex. 1–689).

No human poisonings have been linked to metribuzin. In long-term oral studies in volunteers, the highest no-observed-effect levels (NOELs) were 2.5 to 5 mg/kg per day (ACGIH 1986/Ex. 1–3, p. 411). Single and repeated patch tests in humans did not show sensitization (*Deutsche Forschungsgemeinschaft* 1981, as cited in ACGIH 1986/Ex. 1–3, p. 411).

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ for metribuzin in the construction, maritime, and agriculture sectors. The Agency preliminarily concludes that this limit is necessary to protect workers in these sectors against the significant risks of metabolic and central nervous system effects that are potentially associated with occupational exposure to metribuzin. The Agency considers these adverse effects material impairments of health and believes that the proposed limit will substantially reduce these risks. Promulgation of this PEL will also make OSHA's limit for metribuzin consistent across all regulated sectors.

1–NITROPROPANE

CAS: 108–03–2; Chemical Formula: $CH_3CH_2CH_2NO_2$

H.S. No. 2121

OSHA's permissible exposure limit for 1-nitropropane in general industry, construction, and maritime is 25 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 25 ppm for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA limit of 25 ppm for 1-nitropropane in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

1-Nitropropane is a colorless liquid with a mild, fruity odor. This substance is used as a solvent, chemical intermediate, rocket propellant, and gasoline additive (Hawley's 1987, p. 832; NIOSH/OSHA Occupational Health Guideline 1981, p. 1).

1-Nitropropane causes primary irritation in humans and animals; in animals, exposure to this substance also causes damage to the liver and kidneys. The oral LD_{50}s in rats and mice are 455 mg/kg and 800 mg/kg, respectively (RTECS 1987). The LC_{50} in rats is 3100 ppm for 8 hours (RTECS 1987). Rabbits survived a 1-hour exposure to a 1-nitropropane concentration of 10,000 ppm but died from exposure to a 5000-ppm concentration for 3 hours (Machle, Scott, and Treon 1940, in ACGIH 1986, p. 377). Acutely poisoned animals showed the following signs and symptoms: conjunctival irritation, lacrimation, slow respiration with some rales, incoordination, ataxia, and weakness. Autopsy of acutely poisoned rabbits revealed severe fatty infiltration of the liver and moderate kidney damage (Machle, Scott and Treon 1940, in Proctor, Hughes, and Fischman 1988, p. 377). In rats exposed to a 100-ppm concentration of 1-nitropropane 7 hours/day, 5 days/week for up to 21 months, no systemic effects were observed (Griffin, Stein, and Coulston 1980, in ACGIH 1986, p. 440).

Human volunteers reported that exposure to concentrations above 100 ppm caused irritation after brief but not further specified periods of exposure (Silverman, Schulte, and First 1946, in Clayton and Clayton 1982, p. 4161), and exposure to a 150-ppm concentration of 1-nitropropane for 15 minutes caused eye irritation (RTECS 1987). The symptoms of chronic overexposure to 1-nitropropane include headache and nausea (Clayton and Clayton 1982, p. 4161).

Based on this evidence in humans and animals, OSHA preliminarily concludes that 1-nitropropane is a sensory irritant and also causes possible liver and kidney injury. OSHA believes that, in the absence of a limit for this substance, workers in agriculture are at significant risk of experiencing these effects. Establishing an 8-hour TWA PEL of 25 ppm is thus necessary to reduce substantially the risk of these material health impairments. Promulgation of this limit will also make the PEL for 1-nitropropane consistent across all OSHA-regulated sectors.

OIL MIST (MINERAL)

CAS: 8012–95–1; Chemical Formula: None

H.S. No. 1297

OSHA's limit for oil mist is 5 mg/m³ as an 8-hour TWA in construction, maritime, and general industry. There is no limit in agriculture. The ACGIH has a 5-mg/m³ TLV®–TWA and a 10 mg/m³ TLV®–STEL for oil mist (mineral). NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ for oil mist. This is the limit recently established for this substance in general industry.

Oil mist is the airborne mist of petroleum-based cutting oils or of white petroleum oil. The odor of oil mist is described as similar to that of burned lubrication oil, and this substance is a flammable, oily, colorless liquid

(Clayton and Clayton 1981, p. 3396). Mineral oils are used as lubricants and cutting oils.

Oil mist causes eye, skin, and respiratory tract irritation in animals and dermatitis and respiratory effects in exposed workers. If inhaled in sufficient quantities, oil mist can cause lipid pneumonitis (Cralley and Cralley 1985, p. 177). The oral LD_{50} in mice is 22 g/kg (RTECS 1990). In prolonged contact with the eyes or skin of rabbits, mineral oil causes mild irritation (RTECS 1990). Studies in animals have shown that repeated six-hour daily exposures to 5 mg/m^3 caused no adverse effects (Wagner, Wright, and Stokinger 1964, as cited in ACGIH 1986/Ex. 1–3, p. 449). At 100 mg/m^3, slight changes, including lung effects, were observed in exposed animals (Lushbaugh, Green, and Redemann 1950/Ex. 1–792). Dogs, rabbits, mice, and gerbils were exposed to 5 or 100 mg/m^3 mineral oil mist 6 hours/day, 5 days/week for 2 years; microgranulomas developed only in the dogs and rats (Stula and Kwon 1978, in AJIHA 39(5):393–399).

In humans, exposure to oil mist in the industrial setting has been associated with dermatitis and respiratory effects. A single case of lipid pneumonitis has been reported in a worker exposed to high (not further specified) concentrations of oil mist for 17 years (Proudfit, Van Orstrand, and Miller 1950, in AMA Arch. Ind. Hyg. Occup. Med. 1:105). A metal-polishing mechanic whose skin was repeatedly exposed to mineral oil developed chronic eczema on his hands, forearms, and neck that lasted for 2 years (Sakakibara, Kawabe, Mizuno 1989, in Contact Dermatitis 20(4):291–294). Results of photopatch testing in this worker were positive, indicating that the mineral oil had caused photosensitization dermatitis. Two epidemiological studies in similar populations suggested an association between some mineral oils and cancers of the skin and scrotum (Ex. 426, Ex. 207X, pp. 7–50). A number of scientists believe that these carcinogenic effects were likely to have been caused by contaminants in the oil, such as polycyclic aromatic hydrocarbons and certain additives. Moreover, modern refining techniques have generally eliminated these hazardous substances from mineral oils. OSHA believes that it may be premature to make a judgment on oil mist's carcinogenicity.

Based on this evidence in humans and animals, OSHA is proposing an 8-hour TWA PEL of 5 mg/m^3 for mineral oil mist in the construction, maritime, and agriculture industries. The Agency believes that this limit is necessary to reduce substantially the significant risk of eye, skin, and respiratory tract irritation, pneumonitis, and dermatitis potentially associated with exposure to this substance. OSHA considers all of these adverse effects material health impairments. Promulgation of the proposed limit will also make the PEL for oil mist consistent across all OSHA-regulated sectors.

Note: The 1970 TLV®s listed an entry for "oil mist, vapor"; this entry was deleted at some time prior to the publication of the 1987–88 TLV®s. No entry for "oil mist, vapor" has ever been included on OSHA's Z-tables. Because the term for this substance used by OSHA—oil mist (mineral)—by definition includes the vapor of oil mist, OSHA has determined that keeping the word "vapor" would be redundant and is therefore unnecessary. Accordingly, OSHA's designation of this substance on the new Z-tables for construction, maritime, and agriculture is simply "oil mist (mineral)." This decision is consistent with the evaluations undertaken as part of the PEL update process.

PETROLEUM DISTILLATES (NAPHTHA) (RUBBER SOLVENT)
CAS: 8002–05–9; Chemical Formula: None
H.S. No. 1312

OSHA has no limit in construction, maritime, or agriculture for petroleum distillates (naphtha), also called rubber solvent. The ACGIH has a TLV®–TWA of 400 ppm for petroleum distillates, and NIOSH has a REL of 87 ppm and a 15-minute ceiling of 450 ppm for these substances. OSHA has recently established an 8-hour TWA PEL of 400 ppm in general industry for petroleum distillates, and this is the limit being proposed in construction, maritime, and agriculture. These are the limits recently established for this substance in general industry.

Petroleum distillates (naphtha) are a mixture of hydrocarbons. They are flammable liquids that have a characteristic gasoline-like odor (HSDB 1986). Petroleum distillates are used as solvents and thinners, as a carrier solvent for pesticides, and in gasoline, adhesives, coatings, naphtha soaps, cleaning agents, and in organic synthesis (Hawley's 1987, p. 806; ACGIH 1986, p. 516). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

A study performed by Carpenter, Kinkead, Geary et al. (1975b/Ex. 1–53) exposed rats to concentrations of naphtha between 2800 and 24,200 ppm. Motor incoordination occurred at 5300 ppm, and convulsions and death occurred in all animals at 24,200 ppm. Animals exposed to 480 ppm for 63 days showed no signs of toxicity (Carpenter, Kinkead, Geary et al. 1975b/Ex. 1–53).

NIOSH (1977g, as cited in ACGIH 1986/Ex. 1–3, p. 516) noted that rubber solvent (naphtha) is composed primarily of C_6–C_9 alkanes and, thus, that the limit of 350 mg/m^3 (85 ppm) recommended for C_9–C_8 alkanes should also apply to naphtha. This recommendation presumes that all C_6–C_8 alkanes possess equivalent neurotoxicity; however, OSHA has preliminarily concluded that not all of the C_6–C_8 alkanes are neuropathic agents. Accordingly, OSHA is proposing an 8-hour TWA PEL of 400 ppm for petroleum distillates in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risk of irritation that is associated with exposure to these substances. OSHA considers such irritation to be a material health impairment. Because the proposed limit is identical to the PEL recently established for petroleum distillates in general industry, promulgation of this limit in construction, agriculture, and maritime will make OSHA's limit for petroleum distillates consistent across all regulated sectors.

m-PHTHALODINITRILE
CAS: 626–17–5; Chemical Formula: $C_8H_4N_2$
H.S. No. 1327

OSHA has no limit for m-phthalodinitrile in construction, maritime, or agriculture. NIOSH has no REL for this substance but concurs (Ex. 8–47, Table N1) with the limit being proposed. The ACGIH has a TLV®–TWA of 5 mg/m^3 for m-phthalodinitrile. OSHA is proposing an 8-hour TWA PEL of 5 mg/m^3 for m-phthalodinitrile; this is the limit recently established for this substance in general industry.

m-Phthalodinitrile is a white, odorless solid that occurs in the form of needles or powder. It is used as a chemical intermediate for amines, phthalocyanine pigments and dyes, and for high-temperature lubricants and coatings. It is also used to produce phthalate esters (HSDB 1989, Sittig 1985, p. 727).

m-Phthalodinitrile is an irritant of the eyes, mucous membranes, and skin. The oral LD_{50} in rats is 1660 mg/kg (RTECS 1990). Intraperitoneal administration of lethal doses of this substance caused mice to convulse before death (RTECS 1990). Instilled into the eyes of rabbits, m-phthalodinitrile caused mild irritation (RTECS 1990). In rabbits, slight skin reactions have been reported from dermal applications of m-phthalodinitrile to the intact or abraded

skin for 6 hours/day, 5 days/week over a 3-week period. The doses applied were 0.5, 1.0, or 2.0 g/kg; at the two higher dose levels, some changes in organ (unspecified) size, without histopathologic changes, were observed at autopsy. Female rabbits exposed at the highest dose also lost weight (Owen 1972, as cited in ACGIH 1986/Ex. 1–3, p. 488).

A 15-year review of industrial experience revealed no reports of adverse effects from exposure to m-phthalodinitrile (Zeller, Hofmann, Thiess, and Hey 1963, as cited in ACGIH 1986/Ex. 1–3, p. 488). Williams (1959/Ex. 1–1176) attributes this absence of exposure effects to the fact that the aromatic nitriles, of which m-phthalodinitrile is one, do not liberate cyanide in the body, as is the case with the aliphatic nitriles.

Based on this evidence, OSHA is proposing an 8-hour TWA limit for m-phthalodinitrile of 5 mg/m³ in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers from the significant risk of skin irritation, a material health impairment associated with exposure to m-phthalodinitrile. OSHA believes that the proposed limit is necessary to reduce this risk substantially. In addition, promulgation of the proposed PEL will make OSHA's limit for m-phthalodinitrile consistent across all OSHA-regulated sectors.

PLATINUM (METAL)

CAS: 7440–06–4; Chemical Formula: Pt

H.S. No. 1332

OSHA has no limit for platinum metal in construction, maritime, or agriculture. NIOSH has no REL for platinum but concurs (Ex. 8–47, Table N1) with the limit being proposed. The ACGIH has a TLV®–TWA of 1.0 mg/m³ for platinum metal (dust). OSHA is proposing an 8-hour TWA PEL of 1 mg/m³ for platinum; this is the limit recently established for this substance in general industry.

Platinum is a silver-gray, lustrous, malleable, ductile precious metal. It is used in jewelrymaking, electroplating, photography, and dentistry and in the chemical and electrical industries; it is also used in windings for high-temperature furnaces (ACGIH 1986, p. 492; Proctor, Hughes, and Fischman 1988, p. 417).

Unlike the platinum salts, which cause sensitization in exposed workers, platinum metal is of low toxicity. There are no acute toxicity data for platinum metal. In mice and rats receiving platinum metal implants, local-site tumorigenic responses were seen (RTECS 1990). The limit being proposed by OSHA reflects good industrial hygiene practice and acknowledges that heavy metal dusts are more toxic than particulates not elsewhere classified (for which the PELs are 8-hour TWAs of 10 mg/m³).

OSHA is proposing an 8-hour TWA limit of 1.0 mg/m³ for platinum metal. The Agency believes that this limit is necessary to protect workers in construction, maritime, and agriculture against the adverse health effects potentially associated with workplace exposures to this substance. In addition, promulgation of the proposed PEL will make the limit for platinum consistent across all OSHA-regulated sectors.

RESORCINOL

CAS: 108–46–3; Chemical Formula: $C_6H_4(OH)_2$

H.S. No. 1346

OSHA has no limit for resorcinol in construction, maritime, or agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limits being proposed. The ACGIH has an 8-hour TLV®–TWA limit of 10 ppm and a TLV®–STEL of 20 ppm for this substance. OSHA is proposing an 8-hour TWA of 10 ppm and a 15-minute STEL of 20 ppm for resorcinol. These are the limits recently established for this substance in general industry.

Resorcinol occurs in the form of sweet-tasting white crystals that, because of impurities, may turn pink on exposure to air and light or on contact with iron. Resorcinol is used to manufacture formaldehyde-resorcinol resins, cosmetics, styphnic acid, pharmaceuticals, dyes, and adhesives, as a crosslinking agent for neoprene, and as a rubber tackifier (ACGIH 1986, p. 511; Hawley's 1987, p. 1006).

Resorcinol is an irritant of the eyes and mucous membranes on acute exposure; chronic exposure causes systemic poisoning. The oral LD_{50} in rats is 301 mg/kg, and the dermal LD_{50} in rabbits is 3360 mg/kg (RTECS 1990). Acutely poisoned animals convulse before death (Gosselin, Smith, and Hodge 1984, p. II–191). Applied to the skin of rabbits, resorcinol caused moderate irritation; instillation into the eyes of rabbits caused pain, conjunctival inflammation, and corneal vascularization (Grant 1986, p. 792; RTECS 1990). Daily 6-hour exposures to a resorcinol concentration of 8 ppm for 2 weeks produced no ill effects in rats, guinea pigs, or rabbits. Acute inhalation exposures to a resorcinol-water aerosol at concentrations as high as 7800 mg/m³ for one hour and 2800 mg/m³ for eight hours caused no toxic effects in laboratory animals (Koppers Company 1974, as cited in ACGIH 1986/Ex. 1–3, p. 511). In skin painting studies in mice, resorcinol was not carcinogenic (IARC 1977, Vol. 15, p. 171). This substance is not mutagenic in microbial or tissue culture assays (HSDB 1990).

In humans, the cutaneous application of solutions or salves containing from 3 to 25 percent resorcinol may result in local hyperemia, itching, dermatitis, edema, corrosion, and the loss of the superficial layers of the skin. If this damage is severe, the following effects may be seen: enlargement of regional lymph glands, restlessness, methemoglobinemia, cyanosis, convulsions, tachycardia, dyspnea, and death (Clayton and Clayton 1981, p. 2588). An epidemiologic study of rubber workers exposed to a hexamethylene-tetramine-resorcinol rubber system revealed no specific symptoms caused by resorcinol; resorcinol concentrations at this plant were less than 0.3 mg/m³. In another study, workers reported no irritation or discomfort when concentrations of resorcinol were maintained at 10 ppm or less and exposure lasted for periods of at least 30 minutes (Clayton and Clayton 1981, p. 2588). A recent study reports that 42 workers in an Italian tire factory developed pigmentary dermatosis, characterized by a rusty red pigmentation of the surfaces of the hand; percutaneous absorption of resorcinol was identified as the cause of the condition, which resolved within 1 week of removal from exposure (Abbate, Polito, Puglisi et al. 1989, in Br. J. Ind. Med. 46(3):212–214).

Based on this evidence in humans and animals, OSHA is proposing a PEL of 10 ppm TWA and a STEL of 20 ppm for resorcinol. OSHA preliminarily concludes that these limits together will protect workers in construction, maritime, and agriculture from the significant risks of irritation and systemic toxicity, which are material impairments of health that are associated with occupational exposure to resorcinol. The Agency believes that the proposed limits are necessary to reduce these risks substantially. In addition, promulgation of the proposed PELs will make OSHA's limits for resorcinol consistent across all OSHA-regulated sectors.

ROTENONE

CAS: 83–79–4; Chemical Formula: $C_{23}H_{22}O_6$

H.S. No. 2139

In general industry, construction, and maritime, OSHA's permissible exposure limit for rotenone is 5 mg/m³ as an 8-hour TWA. There is no PEL in agriculture. The ACGIH has an 8-hour TLV®–TWA of 5 mg/m³ for this

substance. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ for rotenone in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Rotenone is a white, crystalline, odorless material. This substance is a selective contact herbicide or insecticide and is used in flea powders, fly sprays, and moth-proofing agents (ACGIH 1986, p. 515; Hayes 1982, p. 81). Rotenone is used in dairy barns, milk rooms, and animal shelters and on many vegetables and fruits (Hayes 1982, p. 82). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Rotenone causes irritation of the eyes and skin and central nervous system effects in humans and animals. There is also evidence in animals that rotenone causes liver and kidney damage, developmental abnormalities, and cancer. The lowest reported oral LD_{50}s in rats and mice are 60 mg/kg and 350 mg/kg, respectively (RTECS 1990). Acutely poisoned animals show signs of respiratory stimulation, followed by respiratory depression, incoordination, convulsions, and tremors before death (Hayes 1982, p. 82). Applied to the eyes of rabbits, rotenone causes intense irritation and pus formation (Hayes 1982, p. 82). An oral dose of 1600 mg/kg caused gastrointestinal effects in rabbits, including ulceration, hypermotility, and diarrhea (RTECS 1990). Intravenous administration of rotenone to experimental animals causes vomiting, incoordination, muscle tremors, clonic convulsions, and respiratory failure (Shepard 1949; Shimkin and Anderson 1936, in Gosselin, Smith, and Hodge 1984, p. III–366). Dogs fed 5 mg/kg/day rotenone for 1 month showed fatty changes of the liver and kidneys at autopsy (Hayes 1982, p. 83). Oral doses of 10 mg/kg given to pregnant rats on days 6 to 15 of pregnancy were fatal to 12 of 20 dams and caused a decrease in the number of live fetuses per surviving dam and an increase in the number of resorptions (Khera et al. 1982, in Proctor, Hughes, and Fischman 1988, p. 437). Musculoskeletal abnormalities were seen in the fetuses of rats given 5 mg/kg oral doses of rotenone on days 6 through 15 of pregnancy (RTECS 1990; Proctor, Hughes, and Fischman 1988, p. 437). Intraperitoneally injecting 40 female rats with 1.7 mg/kg of rotenone in sunflower oil for 42 days produced mammary tumors in 60 percent of the rats within 6 to 11 months of the end of treatment; none of the controls developed tumors after treatment (Gosalvez and Merchant 1973, in Proctor, Hughes, and Fischman 1988, p. 437). There was a slight increase in the incidence of parathyroid gland adenomas among rats given 75 ppm rotenone in their diets for 2 years; female rats given 38 ppm rotenone in the diet for 2 years showed an increased incidence of subcutaneous tissue tumors (Proctor, Hughes, and Fischman 1988, p. 437).

In humans, local effects of rotenone exposure include conjunctivitis, dermatitis, pharyngitis, and rhinitis; oral ingestion causes gastrointestinal irritation, nausea, and vomiting (Klaassen 1986, p. 552; Proctor, Hughes, and Fischman 1988, p. 436). The lowest lethal oral dose in humans is estimated to be 143 mg/kg; at this level, individuals experienced gastrointestinal effects (RTECS 1988). Inhalation of rotenone dust produces respiratory stimulation followed by depression and convulsions (Klaassen, Amdur, and Doull 1986, p. 552).

Based on this evidence in humans and animals, OSHA preliminarily concludes that rotenone causes severe eye and skin irritation and central nervous system effects in exposed individuals. OSHA preliminarily finds that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. OSHA believes that establishing a PEL for rotenone of 5 mg/m³ as an 8-hour TWA is necessary to reduce these risks of material health impairment. Promulgation of this limit will also make the PEL for rotenone consistent across all OSHA-regulated sectors.

SULFUR HEXAFLUORIDE

CAS: 2551–62–4; Chemical Formula: SF_6
H.S. No. 2146

OSHA's permissible exposure limit for sulfur hexafluoride in general industry, construction, and maritime is 1000 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA limit of 1000 ppm for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing a PEL of 1000 ppm as an 8-hour TWA for sulfur hexafluoride in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Sulfur hexafluoride is a colorless, odorless gas. It is used as a gaseous insulator for electrical equipment and as a tracer gas for ventilation measurements in occupied spaces (ACGIH 1986, p. 543(87); Braker and Mossman 1980, p. 649).

Exposure to sulfur hexafluoride can cause nervous system effects and asphyxiation in humans and animals. The intravenous LD_{50} in rabbits is 5790 mg/kg (RTECS 1989). Rats exposed to an atmosphere containing 80 percent (800,000 ppm) sulfur hexafluoride and 20 percent (200,000 ppm) oxygen for 16 to 24 hours showed no observable effects from the exposure (ACGIH 1986, p. 543(87)). Acute exposure to a sulfur hexafluoride concentration of 300 g/m³ produced effects on the nervous system and behavior of rats (Melnikova and Rozova 1981, in HSDB 1986). Under chronic conditions, guinea pigs and rats also showed signs of nervous system effects; changes in the livers and kidneys of these animals were seen at autopsy (Melnikova and Rozova 1981, in HSDB 1986).

Inhaling an 80:20 sulfur hexafluoride-oxygen mixture for 5 minutes caused peripheral tingling, excitement, and some alterations in hearing in humans (Glauser and Glauser 1966, in Proctor, Hughes, and Fischman 1988, p. 451). In a recent incident involving two workers exposed to sulfur hexafluoride (and its electrically induced breakdown products), both workers collapsed; the sulfur hexafluoride concentration was estimated to be 1500 ppm (Pilling and Jones 1988). Both men were disoriented and only semi-conscious, and both had headaches. One of these workers developed pulmonary edema within 90 minutes of rescue; signs and symptoms included cyanosis, dyspnea, and bloody sputum (Pilling and Jones 1988).

Based on this evidence in humans and animals, OSHA preliminarily concludes that sulfur hexafluoride causes asphyxia and affects the nervous system in exposed individuals. OSHA preliminarily finds that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these effects. The Agency believes that establishing an 8-hour TWA limit of 1000 ppm for sulfur hexafluoride in agriculture is necessary to reduce substantially the risks of these material health impairments. Promulgation of this limit will also make OSHA's PEL for this substance consistent across all OSHA-regulated sectors.

TANTALUM (METAL DUST AND OXIDE)

CAS: 7440–25–7; Chemical Formulas: (Tantalum metal) Ta; (Tantalum oxide) Ta_2O_5
H.S. No. 1382

OSHA's PEL for tantalum dust and oxide in construction, maritime, and

general industry is 5 mg/m³ as an 8-hour TWA. There is no limit for these substances in agriculture. NIOSH has no REL for tantalum dust or its oxide but concurs (Ex. 8–47, Table N1) with the limit being proposed. The ACGIH has a TLV®–TWA of 5 mg/m³ and a TLV®–STEL of 10 mg/m³ for these substances. OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ for tantalum metal dust and oxide in agriculture. Promulgation of this PEL will make the limit for these substances consistent across all OSHA-regulated sectors.

Tantalum dust is a black powder, and tantalum oxide is a white, microcrystalline powder. These substances are odorless. Tantalum metal is used in chemical equipment, rectifiers, furnace parts, tools, body implants, and electronic equipment. The oxide is used in optical glass and in piezoelectric, maser, and laser applications (ACGIH 1986, p. 544 (88); Hawley's 1987, p. 1120).

There are no acute toxicity data for the metal; however, the oral LD_{50} in the rat for tantalum oxide is 8000 mg/kg (RTECS 1990). Intratracheal administration of 100 mg tantalum oxide to guinea pigs produced "soft white circumscribed pigmented dust lesions" in their lungs; no fibrosis was seen (ACGIH 1986/Ex. 1–3, p. 554 (88)). Additionally, this study demonstrated transient bronchitis, interstitial pneumonitis, and hyperemia in these animals after administration of the oxide. Implanted into rats, 3760 mg/kg tantalum metal caused injection-site tumors (RTECS 1990).

In humans, tantalum oxide has been used as a dressing for burns (Olsen 1944/Ex. 1–651), and the use of tantalum gauze in surgical repair produced no long-term adverse effects (Dales and Kyle 1958/Ex. 1–587). No adverse health effects have been associated with industrial exposures to tantalum or tantalum oxide (Cochran, Doull, Mazur, and DuBois 1950/Ex. 1–586).

Based on this evidence, OSHA is proposing a 5-mg/m³ 8-hour TWA PEL for tantalum and tantalum oxide. OSHA preliminarily concludes that this limit will protect workers in agriculture from the significant risk of respiratory effects potentially associated with occupational exposure to these substances. These respiratory effects constitute a material impairment of health, and the Agency believes that the proposed limit is necessary to reduce this risk substantially. In addition, promulgation of the proposed limit will make OSHA's limit for tantalum metal dust and oxide consistent across all OSHA-regulated sectors.

1,1,2,2-TETRACHLORO-1,2-DIFLUOROETHANE

CAS: 76–12–0; Chemical Formula: $CFCl_2CFCl_2$

H.S. No. 2154

OSHA's limit for 1,1,2,2-tetrachloro-1,2-difluoroethane in general industry, construction, and maritime is 500 ppm as an 8-hour TWA. There is no PEL for this substance in agriculture. The ACGIH TLV®–TWA for this substance is 500 ppm. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing a 500- ppm 8-hour TWA PEL for 1,1,2,2-tetrachloro-1,2-difluoroethane in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

1,1,2,2-tetrachloro-1,2-difluoroethane, also called Freon 12, is a noncombustible, colorless liquid or solid with a faint, ether-like odor (ACGIH 1986, p. 560; HSDB 1989). This substance is used as a refrigerant. It is also used as a solvent, a blowing and foaming agent, and a corrosion inhibitor in the dry cleaning, polymer and plastics, and electrical industries. In addition, it is used in veterinary medicine as a flukicide and antihelminthic (HSDB 1989; ACGIH 1986, p. 560).

In animals, 1,1,2,2-tetrachloro-1,2-difluoroethane is a mild irritant of the skin and eyes; at high concentrations, it causes central nervous system depression. The oral LD_{50} in mice is 800 mg/kg, and the LC_{50} in rats is 2 parts per hundred (20,000 pph) for 15 minutes (RTECS 1989). Applied to the skin and eyes of guinea pigs, 1,1,2,2-tetrachloro-1,2-difluoroethane caused mild irritation (RTECS 1989). This substance also caused mild irritation in contact with the eyes of rabbits (Proctor, Hughes, and Fischman 1988, p. 460). All rats exposed to 1,1,2,2-tetrachloro-1,2-difluoroethane concentrations of 20,000 to 30,000 ppm for 1 to 2.5 hours died, and the lungs of these animals were severely hemorrhagic at autopsy (Greenberg and Lester 1950, in ACGIH 1986, p. 560). Exposure to 15,000 ppm for 4 hours was fatal to rats; before death, the animals exhibited excitability, incoordination, coma, rapid respiration, tremor, and convulsions, and autopsy showed pulmonary edema and congestion of the lungs and liver (Clayton, Sherman, and Morrison 1966, in ACGIH 1986, p. 560; Proctor, Hughes, and Fischman 1988, pp. 459–460). Rats exposed to 5000 ppm for 18 hours became comatose and died; autopsy revealed pulmonary damage (Greenberg and Lester 1966, in Proctor, Hughes, and Fischman 1988, p. 460). Rats exposed to a concentration of 3000 ppm developed difficult breathing, mild incoordination, and hyperresponsiveness, but these signs subsided after cessation of exposure (Clayton, Sherman, and Morrison 1966, in Proctor, Hughes, and Fischman 1988, p. 460). Rats given 2 g/kg 1,1,2,2-tetrachloro-1,2-difluoroethane daily for 23 to 33 days showed no pathologic changes at autopsy (Greenberg and Lester 1950, in ACGIH 1986, p. 560). Female rats exposed to a concentration of 1000 ppm for 6 hours/day for a total of 31 days had a decreased leucocyte count (Clayton, Sherman, and Morrison 1966, in Proctor, Hughes, and Fischman 1988, p. 460).

Based on effects seen in animals, 1,1,2,2-tetrachloro-1,2-difluoroethane is expected to cause central nervous system depression and pulmonary damage in humans exposed to high concentrations. Early industrial experience with fluorocarbon refrigerants showed that humans exposed to vapor concentrations of approximately 200,000 ppm experienced pulmonary irritation and tremors and became disoriented and comatose; removal from exposure reversed these effects, and there were no sequelae (Gosselin, Smith, and Hodge 1984, p. II–159). Prolonged skin contact with liquid fluorocarbons such as freon 12 causes defatting and may lead to dermatitis (Parmeggiani 1983, p. 897).

Based on this evidence, OSHA is proposing a PEL of 500 ppm as an 8-hour TWA for 1,1,2,2-tetrachloro-1,2-difluoroethane in agriculture. The Agency believes that this PEL is necessary to reduce substantially the significant risk of irritation and central nervous system depression, which OSHA considers material health impairments. Promulgation of the proposed limit will also make OSHA's PEL for 1,1,2,2-tetrachloro-1,2-difluoroethane consistent across all OSHA-regulated sectors.

1,1,1,2-TETRACHLORO-2,2-DIFLUOROETHANE

CAS: 76–11–9; Chemical Formula: CCl_3CF_2Cl

H.S. No. 2153

OSHA's limit for 1,1,1,2-tetrachloro-2,2-difluoroethane in general industry, construction, and maritime is 500 ppm as an 8-hour TWA. There is no PEL for this substance in agriculture. The ACGIH TLV®–TWA is 500 ppm. There is no NIOSH REL; however, NIOSH concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing a PEL of 500 ppm for 1,1,1,2-tetrachloro-2,2-difluoroethane in agriculture. Promulgation of this limit

will make the PEL for this substance consistent across all OSHA-regulated sectors.

1,1,1,2-Tetrachloro-2,2-difluoroethane is a noncombustible, colorless solid with a faint, ether-like odor. This substance is used as a refrigerant, a solvent, a blowing and foaming agent, and a corrosion inhibitor. It is also used in the dry cleaning, polymer and plastics, and electrical industries (HSDB 1989, ACGIH 1986, p. 560). Because of their adverse effects on stratospheric ozone, the use of chlorofluorocarbons as aerosol spray propellants was banned in 1979 (HSDB 1989).

In animals, 1,1,1,2-tetrachloro-2,2-difluoroethane is a central nervous system depressant at very high concentrations, and repeated exposure to lower levels has caused blood and liver changes. The lowest lethal concentration in rats is 20,000 ppm for 30 minutes (RTECS 1989). Rats exposed to a 10,000-ppm concentration for 90 to 120 minutes showed signs of mild central nervous system depression but no ataxia. Exposure to higher concentrations (20,000 to 30,000 ppm), however, was fatal to rats within 1 to 2.5 hours. Repeated exposure to a 1000-ppm concentration for 18 hours/day for 17 days caused no post-mortem lung, liver, or spleen changes in rats (Greenberg and Carter 1950, in ACGIH 1986, p. 560). However, rats exposed to a 1000-ppm concentration for 31 six-hour exposures showed leukopenia and liver changes at autopsy (Clayton, Sherman, Morrison et al. 1966, in ACGIH 1986, p. 560). Rats given 1,1,1,2-tetrachloro-2,2-difluoroethane at an oral dose of 2 mg/kg/day for 23 to 33 days showed no organ pathology at autopsy.

No information is available on industrial exposure to 1,1,1,2-tetrachloro-2,2-difluoroethane. Based on the effects seen in animals, exposure to high concentrations may cause central nervous system depression. In contact with the skin, this substance, like all solvents, is expected to cause defatting and dermatitis.

Based on this evidence, OSHA is proposing a PEL of 500 ppm for 1,1,1,2-tetrachloro-2,2-difluoroethane in agriculture. The Agency believes that this PEL is necessary to reduce substantially a significant risk of potential central nervous system depression, which is a material health impairment. Promulgation of the proposed limit would make OSHA's PEL for 1,1,1,2-tetrachloro-2,2-difluoroethane consistent across all OSHA-regulated sectors.

TRIFLUOROBROMOMETHANE

CAS: 75–63–8; Chemical Formula: $CBrF_3$

H.S. No. 2164

OSHA's limit for trifluorobromomethane in general industry, construction, and maritime is 1000 ppm as an 8-hour TWA. There is no PEL for this substance in agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. The ACGIH TLV®–TWA for trifluorobromomethane is 1000 ppm as an 8-hour TWA, and this is the limit being proposed by OSHA for agricultural workplaces. This is the limit recently established for this substance in general industry.

Trifluorobromomethane (also called Freon-13B1® and Freon 1301) is a colorless gas with a slight, ether-like odor (Braker and Mossman 1980, p. 70). This substance is used as a fire extinguishant and foam-blowing agent, a chemical intermediate, an erosion inhibitor in hydraulic fluids, a refrigerant in food processing and storage, and a metal hardener (HSDB 1987).

At very high concentrations, trifluorobromomethane causes cardiac irregularities in humans and animals. The LC_{50} in rats is 416 g/m³ for an unspecified duration (RTECS 1990). Dogs exposed to a 200,000-ppm concentration of trifluorobromomethane for 3 minutes became agitated and developed tremors; when the concentration was increased to between 500,000 and 800,000 ppm, half of the exposed dogs had epileptiform seizures (Van Stee and Back 1969, in Proctor, Hughes, and Fischman 1988, p. 495). Injection of epinephrine caused arrhythmias in all animals exposed to a 400,000-ppm concentration of trifluorobromomethane (Van Stee and Back 1969). After a 5- to 10-μg/m³ dose of epinephrine, dogs experienced ventricular fibrillation and cardiac arrest at 400,000 ppm trifluorobromomethane and monkeys experienced spontaneous defibrillation (Van Stee and Back 1969). No clinical signs of adverse effects were seen in dogs and cats exposed daily to trifluorobromomethane concentrations of 23,000 ppm for 18 weeks, and autopsy revealed no pathological changes in these animals (Comstock et al. 1953, in ACGIH 1986, p. 605).

Humans exposed to very high concentrations of trifluorobromomethane develop cardiac irregularities; volunteers exposed to a concentration of 169,000 ppm developed atrial-ventricular dissociation and premature ventricular beats (Hine et al. 1968, in ACGIH 1986, p. 605). Volunteers exposed for 3 minutes to a concentration of trifluorobromomethane of between 40,000 and 70,000 ppm showed increased reaction times (Call 1973, in ACGIH 1986, p. 605). In contact with the skin or eyes, liquid pressurized trifluorobromomethane causes frostbite (Clayton and Clayton 1981, p. 3102).

Based on this evidence in humans and animals, OSHA preliminarily concludes that, in the absence of a PEL, workers in agriculture are at significant risk of experiencing cardiac irregularities. Accordingly, OSHA is proposing an 8-hour TWA PEL of 1000 ppm for trifluorobromomethane in agriculture. In addition, promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

TRIMETHYL PHOSPHITE

CAS: 121–45–9; Chemical Formula: $(CH_3O)_3P$

H.S. No. 1410

OSHA has no limit for trimethyl phosphite in construction, maritime, and agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. The ACGIH TLV® for this substance is 2 ppm as an 8-hour TWA. OSHA is proposing an 8-hour TWA PEL of 2 ppm for trimethyl phosphite; this is the limit recently established for this substance in general industry.

Trimethyl phosphite is a colorless liquid with a pungent odor. This substance is used as an intermediate in the manufacture of pesticides, fire retardants, and organic phosphorus additives (ACGIH 1986, p. 609; HSDB 1989).

Trimethyl phosphite's toxic effects include eye, mucous membrane, skin, and pulmonary irritation. The oral LD_{50} in rats is 1600 mg/kg, and the lowest lethal dermal concentration in rabbits is 2200 mg/kg (RTECS 1990). Applied to the skin of rabbits, trimethyl phosphite caused severe irritation; instilled into the eyes of rabbits, it produced only mild irritation (RTECS 1990). Rats exposed to concentrations ranging from 100 to 600 ppm showed dose-dependent effects, including labored breathing, lacrimation, salivation, reduced activity, cold feet, and corneal opacities; at the highest concentration, mature and irreversible cataracts were formed (EPA 1982, in Dialog NIOSHTIC off-prints). Rats inhaling 500 ppm for 8 weeks showed premalignant changes in the lungs at autopsy (EPA 1982). In a sub-chronic inhalation study of rats, Levin and Gabriel (1973/Ex. 1–746) found that exposure to trimethyl phosphite at concentrations of 500±75 ppm for 7.5 hours/day, 5 days/week for 8 weeks caused an adverse effect on body

weight; necropsy revealed evidence of severe pulmonary and cutaneous pathology. At doses of 164 mg/kg during gestation days 8 to 15, trimethyl phosphite caused gross abnormalities in the offspring of treated rats (Mehlman, Craig, and Gallo 1984, in Toxicol. Appl. Pharmacol. 72(1):119–123).

In humans, exposure to trimethyl phosphite causes eye, skin, and upper respiratory tract irritation (Levin and Gabriel 1973/Ex. 1–746). In a group of 179 workers exposed to average concentrations of trimethyl phosphite of between 0.3 and 4 ppm, no ocular changes were observed (Mobil Chemical Company 1980, as cited in ACGIH 1986/Ex. 1–3, p. 609).

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 2 ppm for trimethyl phosphite. The Agency preliminarily concludes that this limit will protect workers in construction, maritime, and agriculture from the significant risk of eye damage, skin irritation, and upper respiratory tract irritation, all material impairments of health that are potentially associated with exposures to this substance at levels permitted in the absence of a limit. In addition, promulgation of the proposed PEL will make the limit for trimethyl phosphite consistent across all OSHA-regulated sectors.

TRIPHENYL AMINE

CAS: 603–34–9; Chemical Formula: $(C_6H_5)_3N$

H.S. No. 1415

OSHA has no exposure limit for triphenyl amine in construction, maritime, or agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. The ACGIH has a 5-mg/m³ TLV®–TWA limit for this substance. OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ for triphenyl amine; this is the limit recently established for this substance in general industry.

Triphenyl amine is a colorless solid that takes the form of monoclinic prisms. It is used as a primary photoconductor to coat film bases (ACGIH 1986, p. 612; HSDB 1989).

Animal studies conducted by the Eastman Kodak Company (Roudabush 1973, as cited in ACGIH 1986/Ex. 1–3, p. 612) showed an oral LD_{50} in rats of 3200 to 6400 mg/kg and an oral LD_{50} in mice of 1600 to 3200 mg/kg. The LD_{50} by intraperitoneal administration for both rodent species exceeded 6400 mg/kg. Skin and eye sensitivity tests in both rabbits and guinea pigs were essentially negative, except that application to the skin of 5 to 20 ml/kg occlusively for four hours produced slight dermal erythema (Roudabush 1973, as cited in ACGIH 1986/Ex. 1–3, p. 612).

Based on this animal evidence, OSHA is proposing a 5-mg/m³ TWA limit for triphenyl amine. OSHA preliminarily concludes that this limit is necessary to protect workers in construction, maritime, and agriculture from the significant risk of skin irritation, a material health impairment that is potentially associated with occupational exposure to this substance at levels permitted by the absence of an OSHA PEL. In addition, promulgation of the proposed PEL will make OSHA's limit for triphenyl amine consistent across all OSHA-regulated sectors.

TRIPHENYL PHOSPHATE

CAS: 115–86–6; Chemical Formula $(C_6H_5O)_3PO_4$

H.S. No. 2165

In general industry, construction, and maritime, OSHA has an 8-hour TWA PEL of 3 mg/m³ for triphenyl phosphate. The Agency has no PEL for this substance in agriculture. The ACGIH has a TLV®–TWA of 3 mg/m³ for this substance; NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL in agriculture of 3 mg/m³ for triphenyl phosphate. This is the limit recently established for this substance in general industry.

The principal use for triphenyl phosphate is as a noncombustible plasticizer for cellulosics; however, this substance is also used in impregnating roofing paper and upholstery, as a plasticizer in lacquers and varnishes, and in rendering acetyl cellulose, nitrocellulose, and airplane "dope" stable and fireproof (HSDB 1985). Triphenyl phosphate is a colorless to white crystalline solid with a phenol-like odor. It may also take the form of white platelets (HSDB 1985).

Triphenyl phosphate causes cholinesterase inhibition in both animals and humans; in laboratory animals, this substance also causes neurotoxic effects. The oral LD_{50} in rats is 3500 mg/kg, and the lowest lethal dose by subcutaneous injection in the monkey is 500 mg/kg (RTECS 1989; Clayton and Clayton 1981, p. 2367). Acutely poisoned rats developed tremors and lost motor coordination before death (RTECS 1989). Mice given triphenyl phosphate orally or intraperitoneally at a dose of 0.2 g/kg showed a 50 percent decrease in plasma cholinesterase levels, and exposure to triphenyl phosphate at a concentration of 757 mg/m³ for 2 hours produced a slight but statistically significant decrease in whole blood cholinesterase activity (Sutton, Terhaar, Miller, Scherberger, Riley, Roudabush, and Passet 1980). Recent work suggests that triphenyl phosphate may not have been responsible for the delayed neurotoxicity seen in earlier studies but that an impurity in the triphenyl phosphate may have been responsible (Wills et al. 1979, cited in HSDB 1985). Applied to the skin of rats and mice, triphenyl phosphate caused no irritation and no inhibition of cholinesterase activity (Antonyuk 1974; Sutton et al. 1980). Slightly depressed growth rates and increased liver weights were seen in rats fed 0.5 percent triphenyl phosphate for 35 days; these effects were not observed in rats fed 0.1 percent triphenyl phosphate for this period (Sutton et al. 1980).

No adverse clinical effects were observed in 16 workers who were exposed for 8 to 10 years to the vapor, mist, and dust forms of triphenyl phosphate at an average concentration of 3.5 mg/m³ (with brief exposures to peaks of 40 mg/m³). However, a slight but statistically significant reduction in red blood cell cholinesterase activity was noted in these workers (Sutton et al. 1980). A study of workers manufacturing aryl phosphates (including triphenyl and tricresyl phosphate) failed to show a correlation between airborne concentration (0.2 to 3.4 mg/m³ aryl phosphate) or duration of exposure to these substances and the gastrointestinal or neuromuscular symptoms reported by these workers (Tabershaw, Kleinfeld, and Feiner 1957a, b). One apparent case of skin sensitization caused by exposure to triphenyl phosphate has been recorded (Clayton and Clayton 1981, p. 2381).

Based on this evidence, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing the cholinesterase inhibition associated with exposure to triphenyl phosphate. The Agency believes that establishing an 8-hour TWA PEL of 3.0 mg/m³ will substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

URANIUM (INSOLUBLE COMPOUNDS)

CAS: 7440–61–1; Chemical Formula: U

H.S. No. 1418

OSHA's PEL for insoluble uranium compounds in the construction and maritime industries is 0.25 mg/m³ as an 8-hour TWA. There is no limit for these substances in agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limits being proposed. The

ACGIH has a TLV®–TWA of 0.2 mg/m³ and a TLV®–STEL of 0.6 mg/m³ for these substances. OSHA is proposing an 8-hour TWA of 0.2 mg/m³ and a STEL of 0.6 mg/m³ for the insoluble uranium compounds; these are the limits recently established for these compounds in general industry.

Uranium is a silver-white radioactive metal that has no odor. Uranium is used in nuclear fuels and weapons, in research, as a catalyst in gas manufacture, and as a source of radium salts and fissionable isotopes (ACGIH 1986, p. 617; HSDB 1986). The proposed PEL would apply to metallic uranium and such compounds as triuranium octaoxide, uranium dioxide, uranium hydride, uranium tetrafluoride, and uranium trioxide.

Exposure to uranium and the insoluble uranium compounds may produce both chemical poisoning and radiation injury to the kidneys and lungs of exposed animals. The insoluble uranium compounds are less toxic chemically than the soluble compounds, but uranium and all uranium compounds have the potential to cause radiation damage. Acute inhalation of 20 mg/m³ concentrations of uranium tetrafluoride, uranium dioxide, or high-grade uranium ore is occasionally fatal to some laboratory animals; exposure to 2.5 mg/m³ concentrations of these substances, however, caused no fatalities and evidence of mild kidney damage only in a few animals (Clayton and Clayton 1981, p. 2002).

Chronic exposure to uranium or its compounds causes radiation injury in animals. Dogs and monkeys exposed to 5 mg/m³ uranium dioxide 6 hours/day, 5 days/week for up to 5 years developed fibrotic changes (suggestive of radiation injury) in their tracheobronchial lymph nodes (both species) and, in monkeys, in the lungs (Clayton and Clayton 1981, p. 2002). Dogs tolerated exposure to a 10 mg/m³ concentration of uranium dioxide for 1 year and to a 10 g/kg/day dietary dose of the same substance for 1 year. Rats injected with metallic uranium in the femoral bone marrow and chest wall developed site-of-contact tumors (sarcomas); the effects of chemical injury and radiation damage could not be distinguished in these lesions (Clayton and Clayton 1981, p. 2003).

In humans, over-exposure to uranium dust or an insoluble uranium compound causes respiratory irritation, difficult breathing, and coughing. A recent review of uranium's nephrotoxicity suggests that it causes damage to the human kidney at renal uranium concentrations substantially below those previously believed to cause harm (Leggett 1989). Uranium workers are at increased risk of death due to pulmonary insufficiency and respiratory, lymphatic, and hematopoietic cancers; these effects are believed to be caused by the decay products of uranium rather than by the uranium itself (Parmeggiani 1983, p. 2238). A study of the risk of respiratory deaths among uranium miners in the United States showed the following dose response: Miners exposed for 5 to 9.9 years had a twofold increase in risk; miners exposed for 10 to 24.9 years increase their risk by 3.6; and those exposed for longer than 24.9 years increased their risk by 3.75. Smoking was shown both to increase the risk of death from respiratory disease and to shorten the neoplastic latency period (Clayton and Clayton 1981, pp. 2010–2011).

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 0.2 mg/m³ and a STEL of 0.6 mg/m³ in construction, maritime, and agriculture for uranium and the insoluble compounds of uranium. The Agency preliminarily concludes that these limits are required to protect workers in these sectors from the significant risks of kidney or blood disorders and radiological damage potentially associated with both long-term and excursion exposures to these compounds. The Agency considers these adverse effects to be material impairments of health and believes that these limits will substantially reduce these risks. In addition, promulgation of the proposed limits will make the PELs for the insoluble uranium compounds consistent across all OSHA-regulated sectors.

Preliminary Conclusions for this Group of Substances

For the group of substances shown in Table C9–1, OSHA preliminarily concludes that occupational exposure causes a broad range of adverse health consequences in exposed workers in construction, maritime, and agriculture; these effects include, among others, central nervous system depression, respiratory irritation, liver and kidney damage, cardiac sensitization, and hepatocellular cancer; OSHA considers all of these effects material impairments of health. The Agency believes, based on a thorough review of the available toxicological evidence, that the proposed limits are necessary to reduce the significant risk of material health impairment associated with occupational exposures to these systemic toxins. Promulgation of these proposed limits will also make OSHA's PELs for these substances consistent across all OSHA-regulated sectors, including general industry, construction, maritime, and agriculture.

10. Substances for Which Proposed Limits Are Based on Avoidance of Physical Irritation and Other Effects

Introduction

In construction, maritime, and agriculture, OSHA is proposing to establish permissible exposure limits for a large group of substances that cause a variety of irritant and other adverse effects. These substances are called "particulates not otherwise regulated" because they are dusts and particulates* that have not been classified in any of the target organ categories addressed in this preamble.

OSHA previously had no substance-specific limits for these individual physical irritants in construction, maritime, or agriculture; the Agency's current generic limit for particulates is 15 mg/m³ as total particulate and 5 mg/m³ as respirable particulate (see Table Z–3 of 29 CFR 1910.1000). OSHA is listing these substances individually in the proposed rule and is proposing either the 15 mg/m³ total particulate limit and the respirable fraction limit of 5 mg/m³ or a 10 mg/m³ 8-hour TWA limit (total particulate) and a 5 mg/m³ (respirable fraction limit).

The health effects information available for these substances has led OSHA to separate them into two groups and to propose different limits for the substances in each group. For one group, which contains substances for which few data are available and which are believed to be of relatively low toxicity, OSHA is retaining the Agency's 8-hour TWA generic particulate limit of 15 mg/m³ (measured as total particulate) in construction and maritime; in addition, where the Agency previously had a 5 mg/m³ 8-hour TWA respirable fraction limit for a substance in this group, this limit is being retained in construction and maritime. For all of the substances in this first group, OSHA is proposing 8-hour TWA PELs of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction) in agriculture; OSHA does not currently regulate particulates in agriculture.

For the second group of particulates not otherwise regulated, additional toxicological information is available that indicates that exposure leads both to physical irritation and to other health effects. For the substances in this group, OSHA is proposing 8-hour TWA PELs of

* Because the term particulate applies to dusts, aerosols, and mists, OSHA uses this term generically in this section to apply to all of these states of matter.

10 mg/m³ (measured as total particulate) and, where applicable, 5 mg/m³ (measured as the respirable fraction) for workplaces in all three of the sectors of interest in this rulemaking. Table C10–1 shows all of the particulates included in this section; those whose principal health effects are physical irritations have proposed PELs of 15 mg/m³ (total particulate and 5 mg/m³ (respirable fraction).

TABLE C10–1.—SUBSTANCES FOR WHICH THE PROPOSED EXPOSURE LIMIT IS BASED ON THE AVOIDANCE OF PHYSICAL IRRITATION AND OTHER EFFECTS

H.S. Number/chemical name	CAS Number	Current generic particulate in construction and maritime[1]	1987–1988 ACGIH TLV® [2]	Proposed OSHA PEL in construction, maritime, and agriculture[1]
1014 alpha-Alumina	1344–28–1	15 mg/m³ TWA	10 mg/m³ TWA	10 mg/m³ TWA; 5 mg/m³ TWA.
1016 Aluminum metal dust	7429–90–5	15 mg/m³ TWA	10 mg/m³ TWA –	15 mg/m³ TWA; 5 mg/m³ TWA.
1024 Ammonium sulfamate	7773–06–0	15 mg/m³ TWA	10 mg/m³ TWA–	10 mg/m³ TWA; 5 mg/m³ TWA.
1031 Barium sulfate–	7727–43–7	15 mg/m³ TWA	10 mg/m³ TWA	10 mg/m³ TWA; 5 mg/m³ TWA.
1032 Benomyl	17804–35–2	15 mg/m³ TWA	10 mg/m³ TWA	10 mg/m³ TWA; 5 mg/m³ TWA.
1035 Bismuth telluride (undoped)	1304–82–1	15 mg/m³ TWA	10 mg/m³ TWA	15 mg/m³ TWA; 5 mg/m³ TWA.
1039 Boron oxide	1303–86–2	15 mg/m³ TWA	10 mg/m³ TWA	10 mg/m³ TWA.
1057 Calcium carbonate	1317–65–3	15 mg/m³ TWA	10 mg/m³ TWA–	15 mg/m³ TWA; 5 mg/m³ TWA.
1061 Calcium silicate	1344–95–2	15 mg/m³ TWA	10 mg/m³ TWA–	15 mg/m³ TWA; 5 mg/m³ TWA.
1062 Calcium sulfate	7778–18–9	15 mg/m³ TWA	10 mg/m³ TWA–	15 mg/m³ TWA; 5 mg/m³ TWA.
1076 Cellulose	9004–34–6	15 mg/m³ TWA	10 mg/m³ TWA–	15 mg/m³ TWA; 5 mg/m³ TWA.
1082 2-Chloro-6-trichloromethyl pyridine	1929–82–4	15 mg/m³ TWA–	10 mg/m³ TWA	15 mg/m³ TWA; 5 mg/m³ TWA.
1095 Clopidol	2971–90–6	15 mg/m³ TWA	10 mg/m³ TWA–	15 mg/m³ TWA; 5 mg/m³ TWA.
1102 Crag herbicide (sesone)	136–78–7	15 mg/m³ TWA	10 mg/m³ TWA–	10 mg/m³ TWA; 5 mg/m³ TWA.
1133 Dicyclopentadienyl iron	102–54–5	15 mg/m³ TWA	10 mg/m³ TWA–	10 mg/m³ TWA; 5 mg/m³ TWA.
1155 Emery	112–62–9	15 mg/m³ TWA	10 mg/m³ TWA	10 mg/m³ TWA; 5 mg/m³ TWA.
1176 Ferbam–	14484–64–1	15 mg/m³ TWA	10 mg/m³ TWA	10 mg/m³ TWA.
1188 Glycerin (mist)	56–81–5	15 mg/m³ TWA	10 mg/m³ TWA–	10 mg/m³ TWA; 5 mg/m³ TWA.
1191A Graphite, synthetic		15 mg/m³ TWA	10 mg/m³ TWA–	10 mg/m³ TWA; 5 mg/m³ TWA.
1192 Gypsum–	13397–24–5	15 mg/m³ TWA	10 mg/m³ TWA	15 mg/m³ TWA; 5 mg/m³ TWA.
1230 Kaolin–	1332–58–7	15 mg/m³ TWA	10 mg/m³ TWA	10 mg/m³ TWA; 5 mg/m³ TWA.
1232 Limestone	1317–65–3	15 mg/m³ TWA	10 mg/m³ TWA–	15 mg/m³ TWA; 5 mg/m³ TWA.
1233 Magnesite	546–93–0	15 mg/m³ TWA	10 mg/m³ TWA–	15 mg/m³ TWA; 5 mg/m³ TWA.
1234 Magnesium oxide (fume)–	1309–48–4	15 mg/m³ TWA	10 mg/m³ TWA–	10 mg/m³ TWA.
1235 Malathion	121–75–5	15 mg/m³ TWA	10 mg/m³ TWA, skin	10 mg/m³ TWA skin.
1239 Marble–	1317–65–3	15 mg/m³ TWA	10 mg/m³ TWA	15 mg/m³ TWA; 5 mg/m³ TWA.
1246 Methoxychlor	72–43–5	15 mg/m³ TWA	10 mg/m³ TWA–	10 mg/m³ TWA.
1278 Molybdenum (insoluble compounds)	7439–98–7	15 mg/m³ TWA–	10 mg/m³ TWA	10 mg/m³ TWA.
1294 Particulates (not otherwise regulated)		15 mg/m³ TWA–	10 mg/m³ TWA	15 mg/m³ TWA; 5 mg/m³ TWA.
1305 Pentaerythritol	115–77–5	15 mg/m³ TWA	10 mg/m³ TWA–	10 mg/m³ TWA; 5 mg/m³ TWA.
1310 Perlite	93763–70–3	15 mg/m³ TWA	10 mg/m³ TWA	15 mg/m³ TWA; 5 mg/m³ TWA.
1328 Picloram	1918–02–1	15 mg/m³ TWA	10 mg/m³ TWA; 20 mg/m³ STEL	10 mg/m³ TWA; 5 mg/m³ TWA.
1331 Plaster of Paris	26499–65–0	15 mg/m³ TWA	10 mg/m³ TWA–	15 mg/m³ TWA; 5 mg/m³ TWA.
1333 Portland cement	65997–15–1	15 mg/m³ TWA	10 mg/m³ TWA–	10 mg/m³ TWA; 5 mg/m³ TWA.
1351 Rouge		15 mg/m³ TWA	10 mg/m³ TWA	10 mg/m³ TWA; 5 mg/m³ TWA.
1359 Silicon	7440–21–3	15 mg/m³ TWA	10 mg/m³ TWA	10 mg/m³ TWA; 5 mg/m³ TWA.
1360 Silicon carbide	409–21–2	15 mg/m³ TWA	10 mg/m³ TWA–	10 mg/m³ TWA; 5 mg/m³ TWA.
1369 Starch	9005–25–8	15 mg/m³ TWA	10 mg/m³ TWA	15 mg/m³ TWA; 5 mg/m³ TWA.
1374 Sucrose	57–50–1	15 mg/m³ TWA	10 mg/m³ TWA	15 mg/m³ TWA; 5 mg/m³ TWA.
2145 2,4,5-T (Trichlorophenoxyacetic acid)	93–76–5	15 mg/m³ TWA–	10 mg/m³ TWA	10 mg/m³ TWA.
1383 Temephos	3383–96–8	15 mg/m³ TWA	10 mg/m³ TWA	10 mg/m³ TWA; 5 mg/m³ TWA.
1391 4,4'-Thiobis (6-tert-butyl-m-cresol)	96–69–5	15 mg/m³ TWA–	10 mg/m³ TWA	10 mg/m³ TWA; 5 mg/m³ TWA.
1396 Titanium dioxide	13463–67–7	15 mg/m³ TWA	10 mg/m³ TWA–	10 mg/m³ TWA.
1423 Vegetable oil mist		15 mg/m³ TWA	10 mg/m³ TWA	15 mg/m³ TWA; 5 mg/m³ TWA.
1434 Zinc stearate	557–05–1	15 mg/m³ TWA	10 mg/m³ TWA–	10 mg/m³ TWA; 5 mg/m³ TWA.
1438 Zinc oxide	1314–13–2	15 mg/m³ TWA	10 mg/m³ TWA–	10 mg/m³ TWA; 5 mg/m³ TWA.

[1] OSHA is proposing to retain the Agency's generic 5-mg/m³ respirable-fraction PEL for particulates where it currently applies in construction and maritime. OSHA's TWA PELs are for 8-hour exposures.

[2] The ACGIH TLV®–TWA is for an 8-hour exposure.

For the second group of substances in this category, however, OSHA has reviewed the available toxicological literature and has preliminarily determined that the current generic total particulate limit of 15 mg/m³ is not sufficiently protective. For these substances, the adverse health effects associated with exposure include, in addition to physical irritation, chronic pulmonary disease, cancer, and mutagenic, reproductive, and teratogenic effects. In the past, many of these substances were designated as "nuisance" dusts or particulates; however, recent toxicologic studies or evidence in humans has increasingly shown that exposure leads to serious health effects. Thus, as applied to these substances, the term nuisance dust is a misnomer, because the hazards these substances pose in the workplace are real, well-documented, and potentially serious. NIOSH shares OSHA's concern about this trend in the toxicology of particulates and has already either designated several of these substances, which were formerly considered "inert," as potential occupational carcinogens or labeled them as causing other target-organ effects. Examples of substances in this category that the recent toxicological literature has suggested may cause more serious effects are: benomyl (reproductive effects); kaolin (pulmonary fibrosis); methoxychlor (cancer); picloram (liver and kidney damage); synthetic graphite (pneumoconiosis); and titanium dioxide (cancer).

In addition to these diseases, toxicologists have recently expressed

concern over the identification of a condition known as pulmonary alveolar proteinosis. This condition, which is apparently caused by the physical effects of particulate exposure, can be fatal if not properly diagnosed, although treatment with lung lavage is sometimes effective (NIOSH–ILO 1988). Pulmonary alveolar proteinosis has occurred in workers exposed to several different particulates, including the so-called inert dusts. The onset of this condition may not occur until months after first exposure to the dust.

For the group of physical irritants identified by OSHA as having other adverse health effects, the proposed rule would establish an 8-hour TWA total particulate limit of 10 mg/m³ and a respirable fraction limit of 5 mg/m³ in construction, maritime, and agriculture. The 10 mg/m³ limit is consistent with the limits established for these substances in the recent general industry rulemaking. That is, the limits being proposed today are those recently established for these substances in general industry. Promulgation of these limits will thus make OSHA's PELs for them consistent across all regulated sectors.

Description of the Health Effects

The adverse exposure effects caused by the substances in this group for which the Agency's current 15-mg/m³ limit is being retained in construction and maritime and proposed in agriculture include: interference with vision; deposition of these substances in the eyes, ears, nasal passages, and upper respiratory tract; and skin and corneal irritation. For the group of substances for which a limit of 10 mg/m³ is being proposed in construction, maritime, and agriculture, the additional adverse exposure effects include pulmonary alveolar proteinosis, reproductive effects, irreversible pulmonary effects, liver and kidney effects, systemic poisoning, and cancer. As discussed above, these latter effects are increasingly being linked to exposure to these substances, many of which were formerly considered biologically inert.

Thus, workers exposed to excessive airborne concentrations of any of these physical irritants may have difficulty seeing, may cough uncontrollably, may develop conjunctivitis or dermatitis, or may develop disabling or even life-threatening disease. In addition to these primary effects, workers distracted by physical-irritant effects may be more likely than nonexposed workers to have accidents and thus to endanger both themselves and others. (These adverse health effects also clearly have substantial productivity impacts.)

In the prior rulemaking, many commenters opposed any reduction in the PELs for these substances on the grounds that the evidence that exposure leads to material impairment of health was inadequate (Exs. 3–1123, 3–726, 3–755, 3–887, 3–898, 3–939, 3–1012, 3–1016, and 8–22). In that rulemaking, OSHA was not persuaded by these arguments and cited the International Labour Organization to the effect that:

> [T]he biological effects of these inert dusts are of a long-term nature and are neither fibrogenic nor carcinogenic, toxic or allergenic. In excessive quantities they will overcharge the protective and scavenging mechanisms, thereby leading to respiratory disease. The extent to which any type of dust represents a health risk thus depends on exposure, which includes the nature of the dust, its concentration and the duration of exposure, as well as upon individual factors such as the general constitution and state of health of the person concerned, including the functional state of the upper respiratory tract, the lung function and its structure, the general immunological status and specific immunological reactivity, and the biochemical reactivity. All these factors will play a part in the onset of disease (Parmeggiani 1983).

OSHA also noted in the earlier rulemaking that a particulate standard of 10 mg/m³ or less (measured as total particulate) is the official standard in a great many countries, including Finland, Denmark, Norway, Sweden, the United Kingdom, Japan, Poland, Czechoslovakia, and the Republic of China (Cook 1987/Ex. 1–187, pp. 234–241).

In addition, OSHA notes with concern the trend in the toxicology of these substances, which is to find increasingly that substances formerly believed to be inert are in fact associated with serious and sometimes life-threatening effects. When exposures to the substances shown in Table C10–1 are kept under good industrial hygiene control in the workplace, as represented by the proposed PELs, OSHA believes that exposures are not likely to result in significant organic disease or irreversible toxic effects among workers exposed to them in construction, maritime, and agriculture.

The following discussions describe OSHA's preliminary findings for this group of physical irritants. In all cases, the limits being proposed for construction, maritime, and agriculture are those that were recently established for these substances in general industry.

ALPHA-ALUMINA

CAS: 1344–28–1; Chemical Formula: Al_2O_3

H.S. No. 1014

OSHA has no specific limit for alpha-alumina in the agriculture, construction, or maritime industries, although OSHA's generic total particulate limit of 15 mg/m³ (5 mg/m³ for the respirable fraction) applies to this substance in construction and maritime. The ACGIH has an 8-hour TWA of 10 mg/m³, measured as total dust, for alpha-alumina. NIOSH has no REL for alpha-alumina. OSHA is proposing an 8-hour TWA of 10 mg/m³ (total particulate) for this substance in the construction, maritime, and agriculture industries. The 5-mg/m³ respirable-fraction limit that applies to alpha-alumina is being retained.

Alpha-alumina, also called aluminum oxide, is a white powder that is widely used as an abrasive grinding material. Alpha-alumina is also used in the production of aluminum; in refractories, ceramics, electrical insulation, paper, spark plugs, crucibles, and laboratory wares, in chromatographic analysis, and in fluxes, artificial gems, and light bulbs (Hawley's 1987, p. 48).

A study by Miller and Sayers (1941/Ex. 1–595) determined that alumina particles with diameters of less than 40 microns produced no reaction in laboratory animals. The results of a study by Stacy, King, Harrison et al. (1959/Ex. 1–761) confirmed the findings of Miller and Sayers; these authors found a-alumina to be nearly inert when injected into the lungs of rats (Stacy, King, Harrison et al. 1959/Ex. 1–761). Inhalation of fine aluminum powders at unspecified levels did not cause fibrosis in rats, guinea pigs, or hamsters (Gross, Harley, and deTreville 1973/Ex. 1–696).

In 1923, shortly after a-alumina replaced sandstone as the industrial abrasive of choice, Macklin and Middleton (1923, as cited in ACGIH 1986/Ex. 1–3, p. 21) reported that workers exposed to aluminum oxide dust using the new, synthetic abrasive had much less pulmonary disease than had workers using sandstone abrasives. Other studies (Sutherland, Meiklejohn, and Price 1937/Ex. 1–674; Meiklejohn and Posner 1957/Ex. 1–1060; Meiklejohn and Jones 1948/Ex. 1–964) reported that workers exposed to aluminum oxide dust in the chinaware industry and in aluminum production showed no evidence of pneumoconiosis. However, some early studies (Clark and Simmons 1925/Ex. 1–725; Clark 1929/Ex. 1–1048) reported that workers engaged in aluminum oxide production and exposed to dust levels generally between 50 and 100 mppcf showed X ray evidence of pulmonary fibrosis; these workers are likely also to have been exposed to silica. Workers

exposed during World War II to bauxite fumes containing both alumina and silica developed pulmonary fibrosis and emphysema; the authors believe that silica fume was involved in the development of these diseases (Shaver and Riddell 1947/Ex. 1–666). NIOSH (Ex. 8–47, Table N4) reports that two studies in animals (Stacy, King, Harrison et al. 1959/Ex. 1–761; Stanton, Laynard, Tegeris et al. 1981) have found that exposure to alpha-alumina is associated with the development of respiratory effects.

OSHA is proposing 8-hour TWA limits of 10 mg/m³ total particulate and 5 mg/m³ respirable particulate for alpha-alumina in construction, maritime, and agriculture; these limits are being proposed for all physical irritants having identified health effects. The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant risk potentially associated with exposures to alpha-alumina dust in the workplace. OSHA believes that skin, eye, and upper respiratory tract irritation and other possible respiratory effects constitute material health impairments, and that the proposed PELs are necessary to substantially reduce these risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

ALUMINUM METAL DUST

CAS: 7429–90–5; Chemical Formula: Al

H.S. No. 1016

OSHA has no specific permissible exposure limit for aluminum metal dust in the agriculture, construction, or maritime industries, although the Agency's generic 15 mg/m³ TWA limit for total particulate applies. The ACGIH has an 8-hour TLV®–TWA limit of 10 mg/m³ as total dust for this substance. NIOSH has no REL for aluminum metal dust. OSHA is proposing a PEL of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction) for aluminum metal dust in the construction, maritime, and agriculture industries. These are the limits recently established in general industry.

In its elemental form, aluminum is a white, malleable, ductile metal. Aluminum is used in building and construction, in the electrical industry, and in auto parts, photoengraving plates, and toothpaste; it also finds use as a powder in paints, as foil in packaging, in decorative stamping, and as flakes for the insulation of liquid fuels (Hawley's 1987, p. 44; Merck 1983, p. 48).

Aluminum metal dust has been shown to present a minimal health hazard, according to results from the McIntyre Foundation's 27-year study of aluminum oxide dust (discussed in Stokinger 1981a, pp. 1500–1503). No deleterious lung or systemic effects were observed as a result of exposure to aluminum metal dust having a particle size of 1.2 μm at calculated concentrations equivalent to 2 mg/m³ over an 8-hour workshift. Even much higher concentrations (not further specified) over 10- or 20-minute periods produced no adverse effects (ACGIH 1986/Ex. 1–3, p. 22). A comment submitted to the docket in the earlier rulemaking by the Reynolds Aluminum Company endorses OSHA's classification of aluminum metal dust under the general dust and particulate heading (Ex. 3–135).

OSHA has preliminarily concluded that aluminum metal dusts are appropriately controlled by retaining the Agency's PELs of 15 mg/m³ TWA (total particulate) and 5 mg/m³ (respirable fraction) in the construction and maritime industries and by proposing to extend these limits to agriculture. OSHA has preliminarily determined that these limits will provide protection against the significant risk of physical irritation posed by exposure to this substance. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

AMMONIUM SULFAMATE

CAS: 7773–06–0; Chemical Formula: $NH_4SO_3NH_2$

H.S. No. 1024

In the construction and maritime industries, OSHA currently regulates ammonium sulfamate under its generic total particulate limit of 15 mg/m³. There is no PEL in agriculture. The ACGIH has established a TLV® of 10 mg/m³ for this substance as an 8-hour TWA. There is no NIOSH REL for ammonium sulfamate. A limit of 10 mg/m³ (and 5 mg/m³ for the respirable fraction) is the limit being proposed by the Agency for construction, maritime, and agricultural operations. NIOSH (Ex. 8–47, Table N4) concurred with this limit when the Agency recently established it in general industry.

Ammonium sulfamate is a combustible, hygroscopic, white, crystalline substance. It is used to manufacture contact and translocated herbicides, such as the weed killer Ammate. Ammonium sulfamate is also used to make fire-retardant materials, to generate nitrous oxide gas, and in electroplating (ACGIH 1986, p. 28). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Lehman (1951/Ex. 1–790) identified oral LD_{50}s of 3900 and 5700 mg/kg in rats and mice, respectively. He also reported that no effects were noted in rats administered 10,000 ppm ammonium sulfamate in the diet for 105 days. The intraperitoneal injection of 0.8 g/kg of ammonium sulfamate caused stimulation of respiration, followed by prostration, in rats. Six of 10 rats so exposed died (Ambrose 1943). The hazards associated with exposure to ammonium sulfamate include eye and nose irritation, interference with vision, and the danger of accidents caused by the distraction and avoidance reactions typical of workers overexposed to dusts in the workplace.

In construction and maritime, OSHA is proposing a PEL of 10 mg/m³ TWA, total particulate, and is retaining the 5-mg/m³ TWA PEL for respirable particulate for ammonium sulfamate; the Agency is also proposing to extend these limits to agriculture. OSHA preliminarily concludes that these limits will protect workers in the construction, maritime, and agriculture industries against the significant risk of material health impairment in the form of physical and other irritation that is associated with exposure to this substance. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

BARIUM SULFATE

CAS: 7727–43–7; Chemical Formula: $BaSO_4$

H.S. No. 1031

OSHA has no specific limit for barium sulfate in the agriculture, construction, and maritime industries, although OSHA's generic 15-mg/m³ total particulate limit applies in construction and maritime. The ACGIH has a TLV®–TWA of 10 mg/m³, total dust, for this substance. There is no NIOSH REL for barium sulfate. The Agency is proposing 10 mg/m³ as an 8-hour TWA PEL for barium sulfate (total particulate) in construction, maritime, and agriculture. OSHA is retaining the 5-mg/m³ PEL for the respirable fraction in construction and maritime and is proposing to extend this limit to agriculture. These are the limits recently established in general industry for barium sulfate.

Barium sulfate is a white or yellowish, odorless, tasteless powder. It is used as an opaque medium in X-ray examinations, for weighting mud in oil-drilling, in paper coatings, paints, rubber, plastics, and lithograph inks, in battery plate expanders, and as a filler

and delustrant for textiles (Hawley's 1987, p. 121).

Einbrodt, Wobker, and Klippel (1972/Ex. 1–1020) exposed rats to a concentration of 40 mg/m³ for two months and concluded that barium sulfate is not toxic. As an inert dust of the noncollagenous type, however, barium sulfate has the potential to cause pneumoconiosis through tissue reactions to accumulated dust in the lung (Anonymous, British Medical Journal 1972). Barium sulfate has not been known to cause adverse effects in industrial workers exposed over periods of several years (Doig 1976/Ex. 1–551).

In construction and maritime, OSHA is proposing an 8-hour TWA PEL for barium sulfate of 10 mg/m³ (total particulate) and is retaining the 5-mg/m³ 8-hour TWA (respirable particulate). The Agency also is proposing to extend both limits to agriculture. OSHA preliminarily concludes that these limits will protect workers in the construction, maritime, and agriculture industries against the significant risks of eye, nose, and upper-respiratory-tract irritation and, perhaps, of pneumoconiosis that are potentially associated with exposure to barium sulfate. The Agency believes these adverse health effects to be material impairments of health and preliminarily finds that the proposed limits are necessary to substantially reduce these risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

BENOMYL

CAS: 17804–35–2; Chemical Formula: $C_{14}H_{18}N_4O_3$

H.S. No. 1032

OSHA has no specific permissible exposure limit for benomyl in the agriculture, construction, or maritime industries. OSHA currently covers benomyl in construction and maritime under the generic total particulate limit of 15 mg/m³. The ACGIH has established a total dust TLV®–TWA of 10 mg/m³ for benomyl. NIOSH has no REL for this substance. OSHA is proposing a PEL of 10 mg/m³ as total particulate for benomyl in the construction and maritime industries, retaining the 5-mg/m³ respirable fraction limit in these sectors, and proposing to extend both limits to agriculture. These are the limits recently established for this substance in general industry.

Benomyl is a white crystalline solid; exposures to this substance occur in its particulate form. Benomyl is used as an ascaricide and a post-harvest fungicide for peaches, apples, etc. It is also used as an oxidizer in sewage treatment (ACGIH 1986, p. 49; Hawley's 1987, p. 127). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Studies of rats and rabbits indicate that the oral and skin absorption LD_{50}s are greater than 10,000 mg/kg, and studies of guinea pigs show a very low risk of skin irritation. Application to the shaved intact skin (as aqueous suspensions containing 5, 12.5, and 25 percent benomyl) of ten male guinea pigs resulted in slight irritation; one of ten guinea pigs had mild erythema two days after application of the high concentration (E.I. du Pont de Nemours and Co., Inc. 1974, as cited in ACGIH 1986/Ex. 1–3, p. 49). In another study, instillation of 10 mg of dry 50-percent powder or of 0.1 ml of a 10-percent suspension in mineral oil caused only temporary mild conjunctival irritation (E.I. du Pont de Nemours and Co., Inc., unpublished data, as cited in ACGIH 1986/Ex. 1–3, p. 49). NIOSH notes that benomyl exposure may cause adverse reproductive effects (Ex. 8–47, p. 12). Mice given an oral dose of 500 mg/kg on the 13th day of pregnancy had offspring with developmental abnormalities of the central nervous and musculoskeletal systems. Rats given oral doses of 156 mg/kg benomyl on days 7 through 16 of pregnancy also had offspring with musculoskeletal abnormalities. Other reproductive effects from exposure to benomyl in mice and rats include adverse effects on spermatogenesis (RTECS 1991).

In humans, skin exposures to a 0.1 percent solution of benomyl caused mild irritation (RTECS 1991). Contact dermatitis occurred among a group of Japanese women who worked in warm moist greenhouses where benomyl was sprayed; this effect occurred after a second spraying of benomyl (Hayes 1982, p. 612).

OSHA is proposing 8-hour TWA PELs in construction, maritime, and agriculture of 10 mg/m³ (total particulate), retaining the 5-mg/m³ respirable particulate limit in construction and maritime, and proposing to extend the respirable fraction limit to agriculture. The Agency preliminarily concludes that these limits will protect workers in the construction, maritime, and agriculture industries from the significant risks of benomyl's effects, which include irritation, erythema, and possible reproductive effects. OSHA believes that these health effects constitute material impairments of health and that the proposed PELs are necessary to reduce these risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

BISMUTH TELLURIDE (UNDOPED)

CAS: 1304–82–1; Chemical Formula: Bi_2Te_3

H.S. No. 1035

OSHA has no specific limit for undoped bismuth telluride in the construction, maritime, or agriculture industry, although OSHA's generic total particulate limit of 15 mg/m³ currently applies in construction and maritime. The ACGIH has a total dust TLV®–TWA of 10 mg/m³ for the undoped form of this substance. NIOSH has no REL for undoped bismuth telluride. OSHA is proposing to retain the 15 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) 8-hour TWA PELs for this substance in construction and maritime and is proposing to extend these limits to agriculture. These are the limits recently established in general industry.

Bismuth telluride appears as gray, hexagonal platelets; it is also available as ingots or single crystals. It is used for semi-conductors and in thermoelectric cooling and power generation applications (ACGIH 1986, p. 59).

An 11-month inhalation study of dogs, rabbits, and rats exposed to pure undoped bismuth telluride dust at 15 mg/m³ showed the pulmonary responses typical of exposures to inert dusts (Wagner, Madden, Zimber, and Stokinger 1974).

OSHA is retaining its permissible exposure limits of 15 mg/m³ TWA, as total particulate, and 5 mg/m³, as the respirable fraction, for pure undoped bismuth telluride in the construction and maritime industries and is proposing to extend these limits to agriculture. The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant risks associated with workplace exposures to bismuth telluride. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

BORON OXIDE

CAS: 1303–86–2; Chemical Formula: B_2O_3

H.S. No. 1039

In construction and maritime, OSHA currently regulates boron oxide under its generic total particulate limit of 15 mg/m³ (5 mg/m³ for the respirable fraction). There is no PEL in agriculture. The ACGIH recommends a total dust TLV®–TWA of 10 mg/m³. OSHA is proposing a total particulate PEL for boron oxide of 10 mg/m³ in construction and maritime, retaining the 5-mg/m³ PEL for the respirable fraction in these two sectors,

and proposing to extend both limits to agriculture. NIOSH (Ex. 8–47, Table N4) concurred with these limits when the Agency recently established them for boron oxide in general industry.

Boron oxide occurs as either a white powder or a granular solid, and it has a bitter taste. It is used as an herbicide, in the production of boron, in heat-resistant glassware, as a fire-resistant additive for paints, in electronics, and in liquid encapsulation techniques (Hawley's 1987, p. 162). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Animal studies indicate that eye and skin irritation were caused by the ocular instillation and the topical application, respectively, of boron oxide to the skin and eyes of rabbits. Aerosol administration at various exposure levels for varying time periods caused mild nasal irritation and an increase in urine acidity and the creatinine coefficient in dogs and rats (Wilding, Smith, Yevich et al. 1959/Ex. 1–599). Young rats that were force-fed a 10-percent slurry of boron oxide in water for three weeks showed no growth retardation or other effects (Wilding, Smith, Yevich et al. 1959/Ex. 1–599).

Garabrant and co-workers (1984/Ex. 1–555) determined the prevalence of eye and respiratory irritation among boron oxide-exposed workers; the signs and symptoms of those exposed to boron oxide concentrations ranging from 1.2 to 8.5 mg/m^3 were then compared with those of controls. Workers exposed to an average boron oxide concentration of 4.1 mg/m^3 reported significant increases in productive cough; eye, nose, and throat irritation; dryness of the mouth; and sore throat (Garabrant, Bernstein, Peters, and Smith 1984/Ex. 1–555).

OSHA is proposing permissible exposure limits of 10 mg/m^3 TWA, as total particulate, for boron oxide in the construction, maritime, and agriculture industries, retaining the 5 mg/m^3 respirable fraction TWA in construction and maritime, and proposing the respirable fraction limit in agriculture. The Agency preliminarily concludes that these limits will protect workers in the construction, maritime, and agriculture industries from the significant risk of upper-respiratory-tract and eye irritation associated with exposure to this substance. OSHA believes that these health effects constitute material impairments of health and that the proposed PELs are necessary to substantially reduce these risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

CALCIUM CARBONATE

CAS: 1317–65–3; Chemical Formula: $CaCO_3$

H.S. No. 1057

OSHA currently covers calcium carbonate in the construction and maritime industries under the Agency's generic 15-mg/m^3 total particulate limit. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 10 mg/m^3 for this substance, measured as total dust. In construction, maritime, and agriculture, OSHA is proposing a total particulate PEL of 15 mg/m^3 as an 8-hour TWA; OSHA is also retaining its 8-hour 5-mg/m^3 PEL for the respirable fraction in construction and maritime and proposing to extend this limit to agriculture. These are the limits recently established for calcium carbonate in general industry.

Calcium carbonate is an odorless, tasteless powder or crystalline solid that is found in limestone, chalk, marble, plant ashes, bones, and shells. This substance is used in human and animal medicine, as an ingredient in paint, rubber, plastics, dentifrices, ceramics, polishes, cosmetics, and many other products.

Calcium carbonate is a moderate skin irritant and a severe eye irritant (Sax and Lewis 1989, p. 677). The oral LD_{50} in rats is 6450 mg/kg (Sax and Lewis 1989, p. 677). Rabbits exposed dermally for 24 hours or ocularly for the same period developed moderate and severe irritation, respectively.

OSHA is proposing an 8-hour TWA PEL of 15 mg/m^3 for calcium carbonate (total particulate) in the construction, maritime, and agriculture industries, is retaining the 5-mg/m^3 respirable particulate limit in construction and maritime, and is proposing it in agriculture. OSHA believes that these limits are necessary to protect workers in construction, maritime, and agriculture from the significant risk of physical irritation associated with exposures to calcium carbonate in the workplace. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

CALCIUM SILICATE

CAS: 1344–95–2; Chemical Formula: None

H.S. No. 1061

In construction, maritime, and agriculture, OSHA has no specific limit for calcium silicate; the Agency regulates this substance in construction and maritime under its generic 8-hour TWA limit for particulates of 15 mg/m^3 (measured as total particulate). The ACGIH classifies calcium silicate as a nuisance dust and has an 8-hour TLV®–TWA of 10 mg/m^3. For construction, maritime, and agriculture, the proposed total particulate PEL is 15 mg/m^3, and the 5-mg/m^3 limit for the respirable fraction is retained in construction and maritime and proposed in agriculture. These are the limits recently established in general industry.

Calcium silicate is a white powder. Calcium silicate is used as an anticaking agent in table salt, foods, pharmaceuticals, and agricultural pesticides. It is also widely used as a replacement for asbestos in thermal insulation (ACGIH 1986, p. 92.1(89); Hawley's 1987, p. 205).

No health effects other than physical irritation have been reported in humans or animals as a result of exposure to calcium silicate. Calcium silicate is thus without long-term adverse health effects if exposures are kept under reasonable control.

OSHA is retaining the 8-hour TWA PEL of 15 mg/m^3, total particulate, and 5 mg/m^3, respirable fraction, for calcium silicate in construction and maritime and is proposing these limits in agriculture. The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant risk of physical irritation in the workplace. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

CALCIUM SULFATE

CAS: 7778–18–9; Chemical Formula: $CaSO_4$

H.S. No. 1062

OSHA currently regulates calcium sulfate in the construction and maritime industries under its generic total particulate limit of 15 mg/m^3. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 10 mg/m^3 (total dust) for this substance. OSHA is proposing 8-hour TWA PELs of 15 mg/m^3 (total particulate) and 5 mg/m^3 (respirable fraction) for calcium sulfate in construction, maritime, and agriculture. These are the limits recently established for this substance in general industry.

Calcium sulfate occurs in the form of a white odorless powder or white crystals. It is used as a soil conditioner, Portland cement retarder, in quick-setting cements, molds, and surgical casts, and as a food additive (ACGIH 1986, p. 93, Hawley's 1987, p. 209). Calcium sulfate dust is reported not to have been associated with lung disease in miners (Hunter 1975). Calcium sulfate thus appears to produce no adverse effects

beyond those associated with general physical irritation.

OSHA is proposing to retain its 8-hour TWA permissible exposure limits for calcium sulfate of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) in construction and maritime and to extend these limits to agriculture. The Agency preliminarily concludes that these limits are sufficient to prevent the significant risk of eye, skin, and other physical irritation in workers in these sectors and believes that these limits are necessary to reduce a significant risk. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

CELLULOSE
CAS: 9004–34–6; Chemical Formula: $(C_6H_{10}O_5)n$
H.S. No. 1076

OSHA currently regulates cellulose in construction and maritime under the Agency's generic 8-hour TWA total particulate limit of 15 mg/m³. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 10 mg/m³ (total dust) for this substance when it contains less than 1 percent quartz. The Agency is retaining the total particulate PEL of 15 mg/m³ as an 8-hour TWA and 5-mg/m³ limit for the respirable fraction of cellulose containing less than 1 percent quartz in construction and maritime and is proposing to extend both PELs to the agriculture sector. These are the limits recently established for cellulose in general industry.

Technical cellulose refers to that portion of the plant cell wall derived exclusively from glucose; it resembles cotton cellulose in its physical and chemical properties (ACGIH 1986/Ex. 1–3, p.113). Cellulose is used in paper, cotton products, packaging, as a source of ethanol, in medical equipment, insulation and soundproofing, and as a fuel (ACGIH 1986, p. 113; Hawley's 1987, p. 236).

Inhalation of cellulose dust is not irritating or toxic in exposed humans if exposures are properly controlled (Schreiber 1974/Ex. 1–1096). In industry, cellulose dust occurs in combination with other substances, such as quartz dust, wood, cotton, flax, jute, and hemp fibers, and these substances have demonstrated toxicities that are unrelated to their cellulose content (ACGIH 1986/Ex. 1–3, p.113).

In construction, maritime, and agriculture, OSHA is proposing 8-hour TWA PELs for this substance of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) for cellulose dust containing less than 1 percent quartz. The Agency preliminarily concludes that these limits are necessary to protect exposed workers in these sectors from the significant risks of eye, skin, and other physical irritation. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

2–CHLORO–6–TRICHLOROMETHYL PYRIDINE (NITRAPYRIN)
CAS: 1929–82–4; Chemical Formula: $C_6H_3Cl_4N$
H.S. No. 1082

OSHA has no specific limit for 2-chloro-6-trichloromethyl pyridine (nitrapyrin) in the construction or maritime industries, although the Agency's generic total particulate limit of 15 mg/m³ as an 8-hour TWA applies. There is no PEL in agriculture. The 1987–1988 ACGIH TLV®–TWA for nitrapyrin is 10 mg/m³ and the TLV®–STEL is 20 mg/m³. There is no NIOSH REL for this substance, but NIOSH concurs (Ex. 8–47, Table N1) with the limits being proposed. OSHA is proposing a total particulate limit of 15 mg/m³ and a respirable particulate limit of 5 mg/m³ for nitrapyrin in the construction, maritime, and agriculture industries. These are the limits recently established for this substance in general industry.

Nitrapyrin is a crystalline substance. It is used as an additive in fertilizer to control nitrification and to prevent loss of nitrogen in soil (ACGIH 1986, p. 428). Agricultural workers are thus exposed to nitrapyrin.

Nitrapyrin's very low vapor pressure makes hazardous inhalation exposures unlikely in the workplace. The oral LD_{50} in rats is 940 mg/kg, and the dermal LD_{50} in rabbits is 850 mg/kg (RTECS 1991). Torkelson (as cited in ACGIH 1986/Ex. 1–3, p. 428) has reported feeding dogs and rats 15 mg/kg nitrapyrin daily for 93 days. He observed no adverse effects in appearance, behavior, growth, food consumption, body and organ weight, mortality, or blood chemistry, and no tissue or organ changes.

OSHA is proposing 8-hour TWA PELs of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) for this dust in the construction, maritime, and agriculture industries; OSHA preliminarily finds that these limits will be protective against the significant risk of nitrapyrin's physical irritant effects. The Agency believes physical irritation is a material health impairment. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

CLOPIDOL (COYDEN)
CAS: 2971–90–6; Chemical Formula: $C_7H_7Cl_2NO$
H.S. No. 1095

OSHA has no specific limit for clopidol in the construction, maritime, and agriculture industries; however, OSHA's generic total particulate limit of 15 mg/m³ TWA applies in construction and maritime. The ACGIH has a TLV®–TWA of 10 mg/m³ and a TLV®–STEL of 20 mg/m³ for clopidol. NIOSH has no REL for this substance. OSHA is proposing a 15-mg/m³ limit (total particulate) and 5-mg/m³ limit (respirable fraction), both as 8-hour TWAs, for clopidol in the construction, maritime, and agriculture industries. These are the limits recently established for this substance in general industry.

Clopidol is a white to light brown powder used as an anticoccidiostat in poultry (Hazardous Substance Fact Sheet 1986, p. 1; ACGIH 1986, p. 141). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The oral LD_{50} for clopidol in rats, rabbits, and guinea pigs is greater than 8 g/kg (Dow Chemical Company 1973c, as cited in ACGIH 1986/Ex. 1–3, p. 141). Long-term (two-year) studies of rats and dogs fed at levels of 15 mg/kg and 5 mg/kg per day, respectively, showed no adverse effects. Similarly, there were no adverse effects on fertility, gestation, viability, or lactation in rats and rabbits, and no increase in teratogenicity (Dow Chemical Company 1973c, as cited in ACGIH 1986/Ex. 1–3, p. 141). The chronic toxicity of clopidol is reported to be low (ACGIH 1986/Ex. 1–3, p. 141).

OSHA is proposing 8-hour TWA PELs of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) for clopidol in construction, maritime, and agriculture. OSHA preliminarily concludes that these limits will protect workers in the construction, maritime, and agriculture industries from the significant risk of eye, skin, and other physical irritation associated with exposure to clopidol. OSHA considers physical irritation a material health impairment. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

CRAG HERBICIDE (SESONE)
CAS: 136–78–7; Chemical Formula: $C_8H_7Cl_2NaO_3S$
H.S. No. 1102

In construction and maritime, OSHA currently applies an 8-hour TWA limit of 15 mg/m³ for the total particulate of crag herbicide; this is the Agency's generic total particulate limit for all particulates. There is no limit in agriculture. The ACGIH has a total-dust TLV®–TWA of 10 mg/m³ for this colorless, odorless,

noncombustible solid. The proposed PELs for crag herbicide are 10 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction) in the construction, maritime, and agriculture industries. NIOSH concurred (Ex. 8–47, Table N4) with OSHA in the selection of these limits when the Agency recently established them in general industry.

Crag herbicide is a colorless, odorless, noncombustible solid used commercially as an herbicide (ACGIH 1986, p. 519). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

An early study reported an oral LD_{50} in rats of 1500 mg/kg for this herbicide (Smyth 1956/Ex. 1–759). At high concentrations, crag herbicide is a gastrointestinal irritant (NIOSH 1984, as cited in ACGIH 1986/Ex. 1–3, p. 519). Rats fed a diet containing 60 mg sesone/100 gm of diet experienced minor liver damage; when fed 20 mg sesone/100 gm of diet for two years, rats showed no adverse effects (ACGIH 1986/Ex. 1–3, p. 519). In 1984, NIOSH reported the oral LD_{50} in rats to be 730 mg/kg (NIOSH 1984, as cited in ACGIH 1986/Ex. 1–3, p. 519). In addition to physical irritation, crag herbicide can affect the central nervous system and cause convulsions in experimental animals. Acutely poisoned animals showed muscular tremor, nystagmus, and convulsions before death. Autopsy of these animals showed evidence of pulmonary hemorrhage and mild liver and kidney damage. A 5 percent solution of crag herbicide dropped into rabbits' eyes caused corneal necrosis, and 0.01 ml of a 5 percent suspension in acetone caused edema and necrosis of the skin in rabbits (Carpenter et al. 1961).

There are no reported incidents of human poisoning associated with the use of sesone.

For construction, maritime, and agriculture, OSHA is proposing 8-hour TWA PELs for crag herbicide (total particulate) of 10 mg/m³ and 5 mg/m³ (respirable particulate). OSHA preliminarily concludes that these limits will protect workers in the construction, maritime, and agriculture industries from eye, skin, gastrointestinal, and other forms of irritation caused by exposure to crag herbicide. The Agency believes that these effects constitute material health impairments. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

DICYCLOPENTADIENYL IRON (FERROCENE)

CAS: 102–54–5; Chemical Formula: $C_{10}H_{10}Fe$

H.S. No. 1133

In construction and maritime operations, OSHA currently covers dicyclopentadienyl iron (ferrocene) under its generic 8-hour TWA total particulate limit of 15 mg/m³. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 10 mg/m³ for this substance. There is no NIOSH REL. OSHA proposes a PEL for dicyclopentadienyl iron of 10 mg/m³ (total particulate) as an 8-hour TWA in the construction, maritime, and agriculture industries and a PEL of 5 mg/m³ (respirable fraction) as an 8-hour TWA. NIOSH (Ex. 8–47, Table N4) supports the selection of these PELs, which were recently established for dicyclopentadienyl iron in general industry.

Dicyclopentadienyl iron is a bright orange crystalline solid that smells like camphor. It is used as a combustion catalyst, as an antiknock additive for gasoline, and in polymers (Hazardous Substance Fact Sheet 1986, p. 1; ACGIH 1986, p. 195).

Available evidence in animals suggests that dicyclopentadienyl iron has a moderate order of oral toxicity but a high order of intravenous and intraperitoneal toxicity. In rats, the oral LD_{50} is 1320 mg/kg (RTECS 1991). In rats, 500 mg/kg is the LD_{50} by the intraperitoneal route of administration (RTECS 1991), but subacute oral toxicity tests have shown no fatalities when 10 feedings of 200 mg/kg were given to rats over a two-week period (E.I. du Pont de Nemours and Co., Inc. 1955, as cited in ACGIH 1986/Ex. 1–3, p. 195). Daily oral administration of this substance at unspecified doses caused hemosidenosis; at a dose of 300 mg/kg for one week, dogs showed decreased hemoglobin, decreased packed cell volume, and decreased erythrocyte count (HSDB 1991). Ferrocene is mutagenic in mammalian test systems (RTECS 1991).

OSHA is proposing 8-hour TWA limits of 10 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction) for dicyclopentadienyl iron in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that these limits will substantially reduce the significant risk of material health impairments, in the form of mutagenic and other effects, that are potentially associated with occupational exposure to this substance. The Agency considers these effects material health impairments and believes that these PELs are necessary to substantially reduce these risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

EMERY

CAS: 112–62–9; Chemical Formula: Al_2O_3

H.S. No. 1155

OSHA currently covers emery in construction and maritime under the Agency's generic 15-mg/m³ total particulate limit for all particulates. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 10 mg/m³, total dust, for emery containing less than 1 percent quartz. OSHA is proposing a total particulate PEL of 10 mg/m³ as an 8-hour TWA and a respirable fraction PEL of 5 mg/m³ for emery containing less than 1 percent quartz in the construction, maritime, and agriculture sectors. These are the limits recently established in general industry.

Emery is impure corundum (aluminum oxide) and is found in certain mineralogical deposits. Natural crystalline deposits of emery are used as abrasives; fine emery is used in lens grinding and in lapidary work (ACGIH 1986, p. 229; Hawley's 1987, p. 316).

Emery dust inhalation is believed to have contributed to a case of pneumoconiosis in France, although it is questionable whether this incident was caused by emery dust alone or by the silica impurities in the dust (Archives des Maladies Professionelles de Medecin du Travail et de Securité Sociale 1970, as cited in ACGIH 1986/Ex. 1–3, p. 229). Exposure to emery dust containing less than 1 percent silica produces little, if any, effect on the health of exposed workers; it does not affect the lungs or produce organic disease at commonly encountered levels (ACGIH 1986/Ex. 1–3, p. 229).

Rats exposed to aluminum oxide (emery is impure aluminum oxide) developed lipoid pneumonia (Stacy, King, Harrison et al. 1959/Ex. 1–761), and humans so exposed have reported skin and respiratory tract irritation.

OSHA is proposing an 8-hour TWA PEL of 10 mg/m³ TWA, total particulate, and an 8-hour TWA PEL of 5 mg/m³, respirable particulate, for emery in construction, maritime, and agriculture. OSHA preliminarily concludes that these limits will prevent the significant risk associated with exposures to emery in the workplace; these risks include skin and upper respiratory tract irritation and, perhaps, other respiratory effects, all of which constitute material health impairments. Promulgation of these limits will also make OSHA's

PELs for this substance consistent across all regulated sectors.

FERBAM

CAS: 14484–64–1; Chemical Formula: $[(CH_3)_2NCS_2]_3Fe$

H.S. No. 1176

In construction and maritime, OSHA currently applies its generic particulate limit of 15 mg/m³ as an 8-hour TWA (total particulate) to ferbam. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 10 mg/m³ for this substance. In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA total particulate PEL of 10 mg/m³ for ferbam as well as an 8-hour TWA respirable fraction PEL of 5 mg/m³. NIOSH concurred (Ex. 8–47, Table N1) with these limits when the Agency recently established them for ferbam in general industry.

Ferbam is an odorless, black solid used as a fungicide on fruits, nuts, vegetables, ornamental crops, and in household applications (ACGIH 1986, p. 269; HSDB 1991). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Ferbam has been reported to have an oral LD_{50} of more than 17 mg/kg in rats, but rabbits and guinea pigs demonstrated less sensitivity to this substance (Hodge, Maynard, Downs, and Blanchet 1952/Ex. 1–861). Dogs showed no adverse effects when fed 25 mg/kg of ferbam daily for six months.

In addition to physical irritation, ferbam can cause skin sensitization, gastrointestinal effects, and reproductive effects in experimental animals. The offspring of pregnant rats exposed to high but sublethal doses of ferbam showed evidence of embryo/fetotoxicity (IARC 1982, Vol. 12, pp. 121–129). Ten of 20 rats died after ingesting a diet containing 0.5 percent ferbam for 30 days; autopsies revealed minor lung, liver, kidney, and bone marrow abnormalities in a few of these animals (Hodge et al. 1952).

Inhalation of ferbam affects the upper respiratory tract in humans in the manner typical of airborne exposures to workplace dusts (Hodge, Maynard, Downs, and Blanchet 1952/Ex. 1–861). The dust of ferbam is irritating to the eyes, nose, throat, and skin (Grant 1986, p. 431). Large oral doses of ferbam can cause gastrointestinal disturbances in humans (AMA Council on Pharmacy and Chemistry 1955, from Proctor, Hughes, and Fischman 1988, p. 257). Ferbam dust can be irritating to the skin and mucous membranes and may cause skin sensitization (Gosselin, Smith, and Hodge 1984, p. II–311).

OSHA is proposing a total particulate PEL for ferbam of 10 mg/m³ as an 8-hour TWA and a respirable fraction PEL of 5 mg/m³ as an 8-hour TWA. The Agency preliminarily concludes that these limits are necessary to prevent the significant health and safety risks associated with exposures to ferbam in the construction, maritime, and agriculture industries. These risks include skin, eye, and upper respiratory tract irritation, which OSHA considers material health impairments. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

GLYCERIN (MIST)

CAS: 56–81–5; Chemical Formula: $CH_2OHCHOHCH_2OH$

H.S. No. 1188

In construction, maritime, and agriculture, OSHA has no specific limit for glycerin mist, although this substance has been regulated in construction and maritime under the Agency's generic total particulate limit. The ACGIH has a TLV®–TWA of 10 mg/m³ (total particulate) for glycerin. OSHA is proposing a total particulate PEL of 10 mg/m³ and a respirable fraction PEL of 5 mg/m³ for glycerin mist in construction, maritime, and agriculture. These are the limits recently established for glycerin in general industry.

Glycerin is an oily, hygroscopic liquid with a warm, sweet taste. Glycerin is widely used to manufacture plastics, cosmetics, foodstuffs, confectioneries, explosives, pharmaceuticals, perfumes, inks, lubricants and softeners, and antifreeze mixtures (ACGIH 1986, p. 286; Hawley's 1987, p. 568).

Glycerin was long considered to be nontoxic; however, there are indications that the mist may be injurious to the kidneys at very high exposure levels (Campanacci 1965/Ex. 1–1047). In addition to physical irritation and kidney effects, glycerin can affect the central nervous system, reproductive system, and gastrointestinal tract in experimental animals. Mice exposed to 8800 mg/kg glycerin experienced convulsions and skin effects (RTECS 1991). Male rats given 2-day intratesticular doses of 280 mg/kg and a 1-day dose of 1600 mg/kg showed changes in fertility (Wiebe and Barr 1984). Rabbits suffered mild skin irritation at a dermal dose of 500 mg glycerin for 24 hours and experienced mild eye irritation after 126 mg of glycerin was put into their eyes (RTECS 1991).

An oral dose of 1428 mg/kg glycerin caused headaches and gastrointestinal effects in humans (RTECS 1991). Although glycerin's toxicity is generally considered to be low, large doses can cause hemolysis, hemoglobinuria, renal failure, convulsions, and paralysis in humans (Ex. 8–47). Oral doses of glycerin administered for therapeutic purposes can cause side effects such as headache, dizziness, vomiting, diarrhea, and fever (Gosselin, Smith, and Hodge 1984, p. II–226).

OSHA is proposing 8-hour TWA limits of 10 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) for glycerin mist in construction, maritime, and agriculture. The Agency preliminarily concludes that these limits will provide protection for workers in these sectors against the significant risks of glycerin exposure, which include possible kidney damage and blood effects. OSHA believes that these effects constitute material health impairments and that the proposed limits are necessary to substantially reduce these risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

GRAPHITE, SYNTHETIC

CAS: None; Chemical Formula: None

H.S. No. 1191A

OSHA currently has no specific limit for synthetic graphite in construction and maritime, although this substance is covered under the Agency's generic total particulate limit. There is no PEL in agriculture, and NIOSH has no REL. The ACGIH has a TLV®–TWA of 10 mg/m³ for graphite as total dust. OSHA is proposing PELs of 10 mg/m³ and 5 mg/m³ for the total and respirable particulate, respectively; these are the limits recently established for synthetic graphite in general industry.

Synthetic graphite is a crystalline form of carbon made from the high-temperature treatment of coal or petroleum products; it has the same properties as natural graphite. Synthetic graphite is used in place of natural graphite in refractories and in electrical uses (ACGIH 1986, p. 291).

Meiklejohn reported in 1958 that synthetic graphite injected intraperitoneally in mice produced effects characteristic of those of the inert dusts (Meiklejohn 1958).

In humans, exposure to natural graphite has long been associated with the development of pneumoconiosis (Koopman 1924/Ex. 1–131; Ruttner, Bovet, and Aufdermauer 1952/Ex. 1–661; Pendergrass, Vorwald, Mishkin et al. 1967/Ex. 1–77). Lister (1961/Ex. 1–422) and Lister and Wimborne (1972/Ex. 1–423) reported fibrotic changes in the

lungs of a worker who had been engaged for 17 years in the production and milling of synthetic graphite. Other reports of lung injury caused by exposure to graphite have not distinguished between the form of the graphite (i.e., natural or synthetic) causing the injury; in addition, exposures to impurities, such as quartz silica, were involved in many of the reported cases (ACGIH 1986/Ex. 1–3, p. 291). In the prior rulemaking, NIOSH (Ex. 8–47) commented that, in its opinion, it is not appropriate to distinguish between the natural and synthetic forms of graphite and noted that the Lister and Wimborne (1972/Ex. 1–423) study described above suggests that synthetic graphite dust exposure "is capable of producing pneumoconiosis." NIOSH believes that a 2-mg/m³ 8-hour TWA PEL is appropriate for synthetic graphite because this is the limit set for coal dust (respirable) to protect against pneumoconiosis (Ex. 8–47). However, because the primary objective of this proposed rule is to make OSHA's limits consistent across all industry sectors, OSHA intends as a first step to propose in construction, maritime, and agriculture the limits recently established for graphite in general industry. At the time of the first PEL update, OSHA will consider the recent toxicological literature on graphite to evaluate whether a lower limit should be considered.

At present, however, OSHA is proposing an 8-hour TWA total particulate limit for synthetic graphite of 10 mg/m³ and an 8-hour TWA respirable fraction limit of 5 mg/m³ to protect workers in construction, maritime, and agriculture against the significant health risks associated with graphite exposures in the workplace. OSHA preliminarily concludes that these limits are necessary to substantially reduce the risks of graphite-induced respiratory disease, which constitutes a material impairment of health. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

GYPSUM

CAS: 7778–18–9; Chemical Formula: $CaSO_4 \cdot H_2O$

H.S. No. 1192

The current OSHA limit for gypsum in general industry, construction, and maritime is an 8-hour TWA of 15 mg/m³. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 10 mg/m³, measured as total particulate. OSHA is proposing to extend the 8-hour TWA PELs for gypsum of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) to workplaces in agriculture. These are the limits recently established for gypsum in general industry.

Gypsum is found either as colorless or white crystals. Gypsum finds use in soil treatment, the manufacture of Plaster of Paris and Portland cement, in the production of heavy chemicals, in water clarification, and in animal feed. There is also a food and pharmaceutical-grade gypsum that is used as a source of calcium (ACGIH 1986, p. 93).

The ACGIH (1986/Ex. 1–3) states that gypsum does not "produce significant organic disease or toxic effect when exposures are kept under reasonable control." Exposures in excess of the proposed limit may result in reduced visibility, deposits of gypsum dust in the eyes, ears, and nasal passages, and skin irritation.

In agriculture, OSHA is proposing 8-hour TWA PELs for gypsum of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction). The Agency preliminarily concludes that these limits will protect workers in this sector from the significant risk of eye, skin, and other forms of physical irritation caused by gypsum exposure. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

KAOLIN

CAS: None; Chemical Formula: $H_2Al_2Si_2O_8O \cdot H_2O$

H.S. No. 1230

OSHA's current limit for kaolin in construction and maritime is 15 mg/m³, measured as total particulate; this is the Agency's generic total particulate limit for all dusts and particulates. There is no PEL for agriculture. The ACGIH has a TLV®–TWA for kaolin of 10 mg/m³, measured as total dust. OSHA is proposing 8-hour TWA PELs for kaolin of 10 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction) in construction, maritime, and agriculture. These are the limits recently established for kaolin in general industry.

Kaolin takes the form either of a white powder or a white or yellow-white earthy mass. Kaolin is used for filler and coatings for paper and rubber; in refractories, ceramics, cements, fertilizers, chemicals, anticaking preparations, cosmetics, insecticides, and electrical insulators. It is also used as a catalyst carrier, a source of alumina, and an adsorbent for the clarification of liquids (Hawley's 1987, p. 669).

Exposure to excess amounts of kaolin dust may cause injury to the skin and mucous membranes (ACGIH 1986/Ex. 1–3). NIOSH (Ex. 8–47, Table N4) also notes that exposure to kaolin dust has been associated with respiratory effects (Lapenas and Gale 1983).

In construction, maritime, and agriculture, OSHA is proposing 8-hour TWA PELs of 10 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) for kaolin. The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant health risks associated with exposure to this substance. These risks include skin and mucous membrane injury, and, perhaps, irreversible respiratory effects, all of which constitute material health impairments. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

LIMESTONE

CAS: 1317–65–3; Chemical Formula: $CaCO_3$

H.S. No. 1232

In construction and maritime, the current generic particulate PEL for limestone is an 8-hour TWA of 15 mg/m³, measured as total particulate. There is no PEL in agriculture. The ACGIH has a 10-mg/m³ TWA for limestone (total particulate). OSHA is proposing 15 mg/m³ as the 8-hour TWA PEL for total limestone particulate and 5 mg/m³ as the 8-hour TWA for the respirable fraction in construction, maritime, and agriculture; these are the limits recently established for limestone in general industry.

Limestone is rock formed by the accumulation of organic remains that consist of calcium carbonate and, less often, magnesium carbonate. Limestone finds use in metallurgy, building stone, agriculture, as a source of carbon dioxide, in cement, alkali manufacture, and in the removal of sulfur dioxide from stock gases and sulfur from coal (Hawley's 1987, p. 701).

Direct contact with limestone dust at unspecified levels has been associated with the development of severe eye irritation and moderate skin irritation (Sax and Lewis 1989). The application of 500 mg limestone to the skin of rabbits for 24 hours produced moderate irritation, and 750 mg instilled into the eyes of rabbits caused severe irritation (RTECS 1990). The oral LD_{50} in rats is 6450 mg/kg (RTECS 1990). In the earlier rulemaking, the American Iron and Steel Institute (Exs. 3–1123 and 8–22) argued that limestone dust produces effects that are "short-term and immaterial" (Ex. 8–22, pp. 29–30); however, OSHA does not agree that the physical irritant effects caused by exposure to dusts and particulates are not material impairments; such irritation affects the

skin, eyes, nose, upper respiratory tract, and mucous membranes adversely.

In construction and maritime, OSHA is retaining 8-hour TWA limits of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) for limestone; the Agency is also proposing to extend these limits to agriculture. OSHA preliminarily concludes that these limits will protect workers in these sectors from the significant risk of eye and skin irritation that may be experienced by employees exposed to limestone in the workplace. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

MAGNESITE

CAS: 546–93–0; Formula: $(MgCO_3)_4 \cdot Mg(OH)_2\ 5H_2O$ (approx.)

H.S. No. 1233

In construction and maritime, OSHA's current PEL for magnesite is 15 mg/m³, measured as total particulate; this is the Agency's generic limit for all dusts and particulates. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 10 mg/m³, also measured as total particulate. The proposed PELs for magnesite are 8-hour TWAs of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction); these limits would apply in construction, maritime, and agriculture. These are the limits recently established for this substance in general industry.

Magnesite occurs as a white powder. It is used to produce various grades of magnesium oxide, to make carbon dioxide, and as an ingredient in refractories (Hawley's 1987, p. 718).

Magnesite is considered by both OSHA and the ACGIH to be one of the dusts that "do not produce significant organic disease or toxic effect when exposures are kept under reasonable control" (ACGIH 1986/Ex. 1–3). Exposure to excess levels of magnesite in the workplace causes skin or mucous membrane irritation resulting either from contact with the magnesite itself or from the rigorous cleansing procedures necessary for removing the dust.

In construction and maritime, OSHA is retaining its 8-hour TWA PEL of 15 mg/m³ TWA for magnesite, measured as total particulate, and its 8-hour TWA PEL of 5 mg/m³ measured as the respirable fraction; the Agency is proposing to extend these limits to agriculture. OSHA preliminarily concludes that these limits will protect workers in these sectors from the significant risk of skin, mucous membrane, and other physical irritation. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

MAGNESIUM OXIDE (FUME)

CAS: 1309–48–4; Chemical Formula: MgO

H.S. No. 1234

In construction and maritime, OSHA's current limit for magnesium oxide (as fume) is 15 mg/m³ as an 8-hour TWA, the Agency's generic limit for particulates. There is no PEL in agriculture. The ACGIH has a TLV®–TWA limit of 10 mg/m³ for magnesium oxide fume. OSHA is proposing 8-hour TWA PELs of 10 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) for magnesium oxide fume in construction, maritime, and agriculture. These are the limits recently established for this substance in general industry.

Magnesium oxide is a very fine white, odorless powder. Magnesium oxide fume is produced during the burning of magnesium. Magnesium oxide in powder form is used in ceramics, pharmaceuticals, as an acid neutralizing agent, as a food additive, and in fertilizers (ACGIH 1986, p. 351; Hawley's 1987, p. 721).

Slight reactions (not further specified) have been reported in human subjects after exposures of less than 10 minutes to freshly generated MgO fume at concentrations of from 400 to 600 mg/m³ (Drinker, Thomson, and Finn 1927/Ex. 1–356). Animal and human studies of magnesium oxide fume exposure have shown toxicities less marked than but similar to those attributable to zinc oxide fume (Drinker and Drinker 1928/Ex. 1–314). The symptoms of exposure include those of metal fume fever (fever, chills, muscular pain, nausea, and vomiting) and leukocytosis, symptoms analogous to those caused by exposure to zinc oxide fume. NIOSH notes that exposure to magnesium oxide may also cause chronic respiratory disease (Ex. 8–47, p. 12).

In construction, maritime, and agriculture, OSHA is proposing PELs of 10 mg/m³ TWA (total particulate) and 5 mg/m³ TWA (respirable fraction) for magnesium oxide fume. OSHA preliminarily concludes that these limits will substantially reduce the significant risks of metal fume fever, leukocytosis, and, perhaps, chronic respiratory disease associated with exposure to magnesium oxide fume in the workplace. OSHA believes that these health effects constitute material health impairments and that the proposed PELs will substantially reduce these risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

MALATHION

CAS: 121–75–5; Chemical Formula: $C_{10}H_{19}O_6PS_2$

H.S. No. 1235

In construction and maritime, OSHA currently has a 15-mg/m³ total particulate limit (8-hour TWA) for malathion, with a skin notation. OSHA has no PEL in agriculture. The ACGIH TLV®–TWA for this substance is 10 mg/m³, also with a skin notation, and the NIOSH REL is 15 mg/m³. OSHA has no PEL in agriculture. OSHA is proposing an 8-hour TWA PEL of 10 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction) in the construction, maritime, and agriculture sectors. These are the limits recently established for malathion in general industry. NIOSH (Ex. 8–47, Table N4) concurs with the selection of these limits.

Malathion is a noncombustible, yellow to deep brown liquid with a skunk-like odor. Malathion finds use as an insecticide for fruits, vegetables, and ornamental plants (Clayton and Clayton 1981, p. 4825). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In addition to physical irritation, malathion causes effects related to cholinesterase inhibition, including headaches, labored breathing, vomiting, tremors, and cardiovascular effects. The oral LD_{50}s for mice and rats are 507 mg/kg and 370 mg/kg, respectively (RTECS 1991). Malathion is a widely used organophosphorus insecticide having a relatively low level of toxicity; some authors have determined that malathion is approximately 1/100th as toxic as parathion (Johnson, Fletcher, Nolan, and Cassaday 1952/Ex. 1–149). Rats fed malathion at a concentration of 100 ppm for two years exhibited no toxic effects (Hazleton and Holland 1953/Ex. 1–126). Several occupational and research studies involving human volunteers have shown that malathion exposure produced no changes in blood cholinesterase or other effects (Rider, Mueller, Swader et al. 1959/Ex. 1–189; Hayes, Mattson, Short, and Witter 1960/Ex. 1–90; Culver, Caplan, and Batchelor 1956/Ex. 1–177). Fatalities have been reported in the Japanese and Indian literature, but these deaths have always involved extremely high doses of malathion (Chabra 1970/Ex. 1–151; Horiguchi 1973/Ex. 1–221).

In the prior rulemaking, one commenter (Ex. 3–678) questioned the need for a skin notation for a substance

with a dermal LD_{50} of 200 mg/kg or less in animal tests when there was no evidence of systemic effects in humans as a result of skin contact (Ex. 3–678, p. 3). OSHA explained to this commenter that its policy is to use a dermal LD_{50} in rabbits of 1000 mg/kg as an indicator of dermal toxicity; this is the Hazard Communication standard's upper cutoff for a toxic, rather than highly toxic, substance administered by the dermal route (see Section VI.C.16 of this preamble for a discussion of OSHA's reasoning on this issue). In addition, OSHA believes that evidence that a substance has caused systemic toxicity in humans exposed via the dermal route is sufficient reason to retain a skin notation; in the case of malathion, OSHA has received reports of exposed workers whose blood cholinesterase levels were reduced after dermal exposure to this substance. OSHA is therefore retaining the skin notation for malathion in the proposed rule.

In construction, maritime, and agriculture, OSHA is proposing PELs of 10 mg/m³ TWA (total particulate) and 5 mg/m³ TWA (respirable particulate) for malathion, with a skin notation. The Agency preliminarily finds that exposure to malathion poses a significant risk of material health impairment in the form of cholinesterase inhibition. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

MARBLE

CAS: 1317–65–3; Chemical Formula: None

H.S. No. 1239

In construction and maritime, OSHA currently has no specific limit for marble dust, although this substance is currently covered under the generic total particulate limit of 15 mg/m³. There is no PEL in agriculture. The ACGIH has established an 8-hour TLV®–TWA of 10 mg/m³ for marble dust containing less than 1 percent quartz (measured as total dust). OSHA is proposing to retain its 8-hour TWA limits for marble dust of 15 mg/m³ as total particulate containing less than 1 percent quartz and 5 mg/m³ as the respirable fraction in construction and maritime, and to extend both limits to agriculture. These are the limits recently established for marble in general industry.

Marble dust, a metamorphic form of calcium carbonate dust, is an odorless and tasteless powder or crystal. Marble is used as a source of carbon dioxide in laboratory experiments and as building stone, monumental stone, ashlar, veneer panelling, wainscotting, and tiling (Hawley's 1987, p. 732, Parmeggiani 1983, p. 1284).

There are no toxicity data in animals for marble dust. Exposure to this substance causes physical irritation of the eyes, nose, mucous membranes, and skin (Genium MSDS 1977).

In the proposed rule, OSHA is retaining its former total particulate limit for marble of 15 mg/m³, as well as the respirable particulate limit of 5 mg/m³ in construction and maritime and is proposing to extend these limits to agriculture. OSHA preliminarily finds that these limits will protect exposed workers in these sectors against the significant risk posed by the physical irritant properties of marble. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

METHOXYCHLOR

CAS: 72–43–5; Chemical Formula: $C_{16}H_{15}C1_3O_2$

H.S. No. 1246

In construction and maritime, OSHA currently applies its generic 15-mg/m³ TWA limit for particulates to methoxychlor. There is no limit in agriculture. The ACGIH recommends a limit of 10 mg/m³ TWA for methoxychlor. OSHA is proposing a 10 mg/m³ limit for the total particulate of methoxychlor and a 5 mg/m³ limit for the respirable fraction in construction, maritime, and agriculture. NIOSH (Ex. 8–47, Table N4) concurs with the selection of these limits, which are identical to those recently established in general industry.

Methoxychlor is a white crystalline solid used as an insecticide on fruit and shade trees, vegetables, dairy and beef cattle, home gardens, and farm buildings (except poultry houses) (Farm Chemicals Handbook 1990, p.C191; ACGIH 1986, p. 364). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The reported oral LD_{50} for methoxychlor in rats is 6000 mg/kg (Lehman 1954, as cited in ACGIH 1986/ Ex. 1–3, p.364). Lehman also determined that 100 ppm for two years is the lowest dietary level producing no effect in rats; this corresponds to a level of 350 mg/man/day (Lehman 1954, as cited in ACGIH, 1986/Ex. 1–3, p. 364). Results of another dietary study indicated that rats fed 200 ppm methoxychlor for two years were not affected in terms of growth or survival (Hodge, Maynard, and Blanchet 1952/ Ex. 1–488). Tegeris and co-workers (1966/Ex. 1–389) reported that dogs fed 1 g/kg daily for 6 months showed weight loss; most animals died within nine weeks when the dietary level was increased to 2 g/kg daily (Tegeris, Earl, Smalley, and Curtis 1966/Ex. 1–389). Morgan and Hickenbottom (1978/Ex. 1–351) reported that male Holtzman rats fed 10, 40, 160, or 640 mg/kg for 24 hours showed no liver abnormalities. Extrapolating from animal data, Lehman (1954) estimated the dose levels that would produce toxic effects in humans as follows: the fatal oral dose would be 450 grams; adverse health effects would occur at 6430 mg/kg orally; and 2414 mg/kg is the level at which dermal effects would be predicted to occur (Lehman 1954). Methoxychlor has been tested in carcinogenicity bioassays and the results have not provided sufficient evidence to determine the carcinogenicity of methoxychlor in animals (Sax and Lewis 1989).

In construction, maritime, and agriculture, OSHA is proposing 8-hour TWA limits for methoxychlor of 10 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction). The Agency believes that these limits are necessary to reduce the significant health risks of systemic toxicity, which constitutes a material impairment of health within the meaning of the Act. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

MOLYBDENUM (INSOLUBLE COMPOUNDS)

CAS: 7439–98–7; Chemical Formula: Insoluble compounds

H.S. No. 1278

In construction and maritime, OSHA currently has a limit of 15 mg/m³ TWA for the insoluble compounds of molybdenum, which include molybdenum metal dust and the dioxide; this is the Agency's generic limit for all particulates. There is no limit in agriculture. The ACGIH recommends a TLV®–TWA of 10 mg/m³, measured as molybdenum, for these substances. NIOSH has no REL. OSHA is proposing PELs of 10 mg/m³ TWA (total particulate) and 5 mg/m³ (respirable particulate) for the insoluble compounds of molybdenum, measured as molybdenum, in construction, maritime, and agriculture. These are the limits recently established for these compounds in general industry.

Molybdenum is a silver-white metal or a dark gray or black powder. The soluble molybdenum compounds include molybdenum trioxide, ammonium molybdate, the hexammonium salt of molybdic acid, and dihydrated sodium molybdate. Molybdenum trioxide is used as a source of molybdenum

compounds, in agriculture, pigments, ceramic glazes, and as a catalyst. Ammonium molybdate is used in pigments, as an analytical reagent, and in coal technology. The hexammonium salt of molybdic acid is used as an intermediate in the manufacture of pigments, as a component of fertilizers, and as a reagent in chemistry. Sodium molybdate finds use in the manufacture of pigments, as a bath additive for metal finishing, and as a micronutrient for plants and animals (Merck 1983, p. 79, 1238; Hawley's 1987, p. 68, 795).

Mogilvskaya (1950, as cited in ACGIH 1986/Ex. 1–3, p. 415) reported that the dust of molybdenum metal and molybdenum dioxide caused irritation of the mucosal surfaces in white mice after an intensive dusting for one hour; in a similar 30-day exposure, the metal and the dioxide proved only minimally poisonous. Molybdenum trioxide at 100 mg/kg/day was lethal to rats, and exposure to this substance by inhalation caused eye and nose irritation in these animals and was subsequently lethal (HSDB 1991). Molybdenum trioxide was also weakly tumorigenic (lung tumors) in Strain A mice after injection (Shimkiu et al. 1978). Rats given 1.2 g/kg ammonium molybdate orally showed trembling, signs of colic, incoordination, and dyspnea (Browning 1969).

The insoluble compounds of molybdenum are generally acknowledged to have a low order of toxicity; however, there is some evidence that respiratory effects have been caused by exposure to these compounds (Browning 1961b, Friberg and Lener 1986, Stokinger 1981d). Exposure to molybdenum trioxide is known to cause severe irritation of the eyes and mucous membranes (Parmeggiani 1983). Three of 19 workers exposed to molybdenum and molybdenum trioxide developed pneumoconiosis after 4 to 7 years of exposure to concentrations ranging from 1 to 19 mg/m^3 (Parmeggiani 1983).

In construction, maritime, and agriculture, OSHA is proposing PELs for the insoluble compounds of molybdenum, measured as molybdenum, of 10 mg/m^3 TWA (total particulate) and 5 mg/m^3 TWA (respirable particulate). The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant health risks of exposure to the insoluble compounds of molybdenum, which include eye, nose, and skin irritation, and may include chronic respiratory effects. OSHA believes that these effects constitute material health impairments and that the proposed limits are necessary to substantially reduce the risk of these effects. In addition, promulgation of these limits will make OSHA's PELs for these substances consistent across all regulated sectors.

PARTICULATES (NOT OTHERWISE REGULATED)

CAS: None; Chemical Formula: None
H.S. No. 1294

In construction and maritime, OSHA currently covers all otherwise unregulated particulates under a single 8-hour TWA PEL of 15 mg/m^3 (measured as total particulate) and a single 8-hour TWA PEL of 5 mg/m^3 (measured as the respirable fraction). The ACGIH has a TLV®–TWA of 10 mg/m^3 (as total dust) for all not-otherwise-classified particulates having a quartz content of less than 1 percent.

OSHA is proposing to extend the permissible exposure limits (PELs) for Particulates Not Otherwise Regulated (PNORs) that were recently retained in general industry, which are 8-hour TWAs of 15 mg/m^3 (total particulate) and 5 mg/m^3 (respirable fraction) to workplaces in the agricultural sector. OSHA is retaining these limits for workplaces in construction and maritime but changing the name given to this group of substances from "nuisance dusts" to particulates not otherwise regulated and making it clear that these limits apply to organic as well as inorganic dusts. OSHA has established PELs for particulates not otherwise regulated based on their ability to cause physical irritation of the eyes, skin, mucous membranes, and upper respiratory tract. In the earlier rulemaking, OSHA did not consider or discuss other adverse health effects caused by exposure to the PNORs. The following discussion summarizes more recent information and studies on adverse health effects that result from exposure to the so-called "nuisance," or inert dusts. This information demonstrates that these dusts have health effects and that the term "nuisance" is indeed inappropriately applied to them.

The term "lung overload" denotes a series of general responses of the pulmonary system to the presence of large quantities of respirable, insoluble, and relatively benign dusts that are retained for extended periods of time in the lungs (Muhle et al. 1989). The mechanism thought to be responsible for this phenomenon is impaired clearance. Morrow (Fundamental and Applied Tox, 1988, Possible Mechanisms to Explain Dust Overloading of the Lungs, p. 369) hypothesizes that dust overloading reflects a breakdown in alveolar macrophage (AM)-mediated dust removal caused by the loss of AM mobility. The inability of dust-laden AMs to translocate to the mucociliary escalator correlates with the average composite particle volume per alveolar macrophage in the lung; when the distributed particulate volume exceeds a specific volume, the evidence suggests that AM-mediated particle clearance virtually ceases, causing particle-laden macrophages to remain in the alveolar region. A number of theories have been advanced to explain how these particle-laden cells become immobilized.

Even dusts thought to be benign can cause lung overload, which appears to be dependent on the amount of dust in the lung rather than the particular type of dust. Dust overload occurs when the amount of dust present overwhelms the ability of the macrophages (a type of white blood cell) to engulf and digest or move the dust particles. A well-functioning phagocyte engulfs dust and other foreign particles, and either digests them or moves them toward the bronchus and up the trachea so that they can be spit out or swallowed. When dust overload occurs, clearance of these particle-laden macrophages is reduced, and they remain deep in the lung. These retained particles then further reduce the ability of the lung to function normally.

To investigate lung response to high molecular weight polymers (considered biologically inert), Muhle et al. 1989 (H. Muhle, R. Mermelstein, C. Dasenbrock, S. Takenaka, U. Mohr, R. Klipper, J. MacKenzie and P. Morrow, Lung response to test toner upon 2-year inhalation in rats, *Exp. Pathol.* 1989; 37:239–242) exposed rats to a special test toner (35% respirable) at 0, 1, 4, or 16 mg/m^3, to titanium dioxide (TiO_2) at 5 mg/m^3, or to silica (SiO_2) at 1 mg/m^3 for 6 hours/day, 5 days/week for up to 24 months. No changes in any pulmonary parameter were observed in the rats exposed to the toner at the low exposure level. In the toner mid-exposure group, a very slight to slight degree of fibrosis was detected in 20% of the rats at the termination of the study. In the toner high-exposure group, fibrosis of slight degree was observed in 65% of the animals and a moderate degree of fibrosis was seen in 18% of the rats. In the TiO_2-exposed rats, a 5% prevalence of lung fibrosis was observed. Based on the volume of TiO_2 retained in the lungs in this study and on comparison with published values, the fibrogenic potency of the test toner and TiO_2 were calculated to be comparable.

In the SiO_2 (positive control) group, a moderate degree of lung fibrosis was detected in 85% of the animals and a

slight degree of fibrosis was observed in 14% of these rats at the termination of the study. Lipoproteinaceous material in the alveoli was detected in the alveoli in many of the animals at final sacrifice, predominantly in the toner high-exposure and silica exposure groups. The authors concluded that the various lung changes observed in the toner high-exposure rats and to a lesser extent in the toner mid-exposure animals (including the proteinosis) are identical to the characteristic signs of lung overloading. The authors also stated that, since the observed pulmonary changes are not uniquely associated with a specific test toner, their findings are likely to be applicable to other toners and to other relatively non-toxic respirable dusts as well.

In a subsequent study, Muhle et al. (H. Muhle, B. Bellman, O. R. Klipper, J. MacKenzie, R. Mermelstein, Subchronic Inhalation Study of Toner in Rats, Inhalation Toxicology, 2:341–360, 1990, p. 341–359) exposed rats to special (35% respirable) test toner at 0, 1, 4, 16, or 64 mg/m³ for 6 hours/day, 5 days/week for 13 weeks. No exposure-related effects were seen in the test animals in terms of survival or causes of death, clinical chemistry or hematology parameters, or systemic or upper respiratory system effects. Exposure also caused no alteration in tumor frequency.

Significant pulmonary responses were seen in the rats exposed at the middle and high dose levels. At 16 mg/m³, some indications of retarded lung clearance were noted and, at 64 mg/m³, no appreciable clearance of toner was detected. Seventy-five days after the 90-day exposure interval, test toner was retained in the lungs of the rats in the 4, 16, or 64 mg/m³ groups in the ratio of 1: 10.3: 77, compared with the theoretical toner input ratio of 1: 4: 16.

The authors attributed the pulmonary changes observed at the two highest exposure levels to "lung overload." Based on the results of this study, the authors stated that both the maximum tolerated dose (MTD) and the maximum functionally tolerated dose (MFTD) of test toner were exceeded at the 64 mg/m³ exposure level.

At a symposium organized by the International Society of Aerosols in Medicine (ISAM) and Environmental Health Sciences Center (EHSC) of the University of Rochester and held in Rochester, New York (May 17 and 18, 1990), Mermelstein and Klipper, scientists from the Xerox Corporation, presented a poster session, entitled "Particle-Lung Interactions: 'Overload' Related Phenomena." These authors also presented a paper entitled, "Xerox exposure limit for respirable dust" (N.O.S.) (Paper 406) at the 1990 American Industrial Hygiene Conference (AIHC), May 21–25, in Orlando, FL. Mermelstein and Klipper conducted a chronic inhalation study of toner in rats (discussed above) and then performed a recovery study to determine the reversibility of the pulmonary changes observed in rats after both acute (8 days) and subchronic (13 weeks) exposure.

To simulate an acute overexposure situation, the rats were exposed to 40 mg/m³ (high level) of the test toner for 8 days. In the main recovery study, female rats were exposed either to 0 mg/m³ (control), 10 mg/m³ (low), or 40 mg/m³ (high) concentrations of toner for 13 weeks, followed by four 13 week observation intervals.

The general results of the recovery study paralleled those of the subchronic study. In the high-dose 8-day exposure group, a slight inflammatory response was seen at the end of exposure, but the lungs returned to normal within 90 days post-exposure. In the low dose 13-week exposure group, the slight inflammatory response observed returned to normal within 180 days but the slight decrease in clearance persisted through the 15 month post-exposure period. In the high dose 13-week exposure group, a moderate inflammatory response and a decrease in clearance, both symptoms of overloading, were observed. At the end of the 15-month post-exposure period, animals in this group continued to show a decrease in clearance; the authors described these effects as "extensive symptoms of overloading." In addition, very slight fibrosis was present in the lungs of 2 of the 5 animals in the high-dose subchronic group.

Researchers have attempted to correlate published accounts in the toxicologic literature that document "lung overload" in animals with the human experience. Cotes and Steel described the progressive nature of lung overload in coal miners (1987, Work-Related Lung Disorders, Blackwell Scientific Publications, Oxford, p. 178, as reported in Mermelstein and Klipper, Paper #406: 1990 AIHC):

> Coal miners and others who breathe air contaminated with coal dust retain some dust in their lungs. The dust accumulates in small foci or macules which constitute simple pneumoconiosis of coal workers * * *. Simple pneumoconiosis is not associated with respiratory symptoms, does not shorten life and in the absence of chest radiograph may not even be suspected. However, with passage of time, following heavy dust exposure one or more macules enlarge or a number coalesce to form progressive massive fibrosis (PMF); the lesions progressively disrupt the normal function of the lung, give rise to respiratory symptoms and reduce life expectancy. This sequence constitutes classical pneumoconiosis of coal workers.

Morrow et al. (Morrow, Muhle, Mermelstein, 1991, Chronic Inhalation Study Findings as a Basis for Proposing a New Occupational Dust Limit, Journal of the American College of Toxicology) assumes that there may be a human counterpart to the dust overloading seen in animals that occurs at the same milligram dust per gram lung concentration. Applying extrapolation modeling to determine a likely correlate value for humans, these authors conclude that an exposure limit of 1 mg/m³, measured as respirable dust, would be appropriate in industrial settings. Based on these considerations, Xerox Corporation has established an internal respirable dust limit of 0.4 mg/m³.

Pulmonary Alveolar Proteinosis

Pulmonary alveolar proteinosis (PAP), also referred to as phospholipidosis, is a syndrome that occurs as a result of the abnormal accumulation of surfactant phospholipids and proteins in the alveolar spaces of the lung. This condition was first described by Rosen et al. in 1958. Since then, numerous case reports have appeared in the literature. The symptoms of PAP are similar to those of other progressive restrictive lung diseases: nonproductive cough, progressive dyspnea on exertion, and fatigue. Depending on the severity of the disorder, weight loss, hemoptysis, chest pain, and fevers may be experienced. No single etiologic agent has been identified. In fact, the broad spectrum of etiologic agents and disease progression found in alveolar proteinosis suggest that it represents one mechanism by which the lung responds to a variety of insults (Rosen, et al., 1958; Fishman, Pulmonary Diseases and Disorders, 2nd Edition, 1988, p. 893).

The material that fills the alveolar spaces in PAP is granular, protein-lipid in nature, and stains positive with the PAS stain. In most respects, the material in the alveoli is qualitatively similar to normal surfactant. Because of the excess of this material in the alveolar spaces, however, profound alterations occur in gas exchange and exercise tolerance, and it is also likely that its presence contributes to a marked decline in alveolar antimicrobial defenses. Surfactant is a substance formed in the lungs that helps to keep the small air sacs expanded by virtue of its ability to reduce the surface tension. Most evidence suggests that PAP is due to decreased removal of alveolar surfactant rather than to an increase in its production (Fishman, Pulmonary Diseases and Disorders, 1988).

There are several steps in the intra-alveolar and intra-cellular regulation of the quantity of surfactant in the alveolar spaces. Interference with the cycle, particularly in the return phase, by diverse stimuli ranging from inorganic particulates, fibrogenic dusts, and wood products to viral infections could lead to a decrease in the uptake of the surfactant phospholipid from the alveolar spaces at the level of the surfactant apoproteins or the apoprotein-phospholipid product.

PAP has been associated with exposure to various occupational substances. Case reports have implicated coal, tin, and uranium, as well as cement, silica, bentonite, and alumina (Rosen Samuel H; Benjamin Castleman and Averill A. Liebow, "Pulmonary Alveolar Proteinosis," N Eng J Med, 258, 23;1123–1142, June, 1958; Rubin, Ava Gordon, L. Weisbrod and Douglas E. Sanders, "Pulmonary Alveolar Proteinosis: Relationship to Silicosis and Pulmonary Infection," Radiology, 135:35–41, April 1980; Gough, J., "Correspondence concerning silicosis and alveolar proteinosis, Brit Med J, 1:629, March 1967; Davidson, Jean M. and W.M. Macleod, "Pulmonary Alveolar Proteinosis," Brit J Dis Chest, 63:13–28, 1969; Miller, Roberta R., Andrew M. Churg, Marion Hutcheon and Stephen Lam, "Pulmonary Alveolar Proteinosis and Aluminum Dust Exposure," Am Rev Resp Dis, 130:312–315, 1984). Most case reports characterize the dust exposures involved in these cases only as "heavy." However, one case study involving alumina (aluminum oxide dust) did characterize the dust obtained by lung biopsy as consisting of spheres less than 1 micron in diameter.

Rubin et al., 1980, report on the clinical course and treatment of 13 cases of pulmonary proteinosis seen at Toronto General Hospital from 1974 to 1978. The authors report that PAP appears to be a disorder of the alveolar lining cells that is characterized by an increase in the number of cells resembling granular Type II pneumocytes and inadequate clearance of pulmonary lipoproteins, and particularly of palmitoyl lecithin (surfactant). Treatment for the disease is palliative and consists of pulmonary lavage.

Davidson and Macleod reviewed the literature on PAP in 1969; these authors substantiate Rosen's original claim that PAP is primarily a disease of males (Davidson and Macleod, 1969, Pulmonary Alveolar Proteinosis, Brit J Dis Chest, 63, 13). The male-to-female ratio among reported cases is 3 to 1. This ratio is most likely to be due to the fact that men rather than women generally work in dusty jobs. The authors also report that an analysis of the reported cases shows that nearly half of the affected individuals had been exposed to dusty environments or solvents.

Animal studies have demonstrated that PAP develops after exposure to a variety of mineral dusts and fumes. The condition has often been described in rats that have inhaled silica dust (Gross and de Treville, 1968; Heppleston et al., 1970, 1974; Heppleston, 1975).

Corrin and King (1970, Pathogenesis of experimental pulmonary alveolar proteinosis) exposed rats to pyro aluminum powder, pure quartz, quartz of 60% and 5% cristobalite content, or a non-fibrogenic feldspar. Alveolar proteinosis developed, in these animals and could be traced to the earliest stages of dust exposure. There was an increase in alveolar macrophages in all dust-exposed animals, especially in those who later developed proteinosis.

In the Muhle et al. study discussed earlier, lipoproteinaceous material was detected in the alveoli at the final sacrifice, predominantly in animals from the high toner and quartz exposure groups. A very slight to slight degree of lipoproteinosis was also seen in the high toner group animals, while lipoproteinosis of slight to moderate degree was observed in the quartz-exposed rats.

Researchers have concluded that there is an association between alveolar proteinosis and the development of fibrosis, although the nature of that relationship remains unclear. Hudson et al. (1974) suggest that the typical histological picture of PAP is an early stage of a disease in which the end stage is fibrosis (Hudson et al., Pulmonary Interstitial Fibrosis Following Alveolar Proteinosis, *Chest*, 65: June 6, 1974).

OSHA preliminarily concludes that there is ample evidence of dust overloading of the lung and pulmonary alveolar proteinosis to provide additional support for a total particulate PEL of 15 mg/m³ as an 8-hour TWA and a respirable dust PEL of 5 mg/m³, also as an 8-hour TWA, for all Particulates Not Otherwise Regulated. These limits are necessary to reduce the significant risk of dust overloading and PAP, as well as other dust-related effects such as physical irritation, reduced vision, etc. Consequently, OSHA is proposing to retain these limits in workplaces in construction and maritime and to propose them in agriculture. These limits cover both organic and inorganic PNORs. OSHA will continue to monitor the research in these areas and may elect to revisit these issues in the future to determine whether these limits are adequately protective.

OSHA believes that good industrial hygiene practice requires that exposures to particulates be controlled in the workplace to or below the 15-mg/m³ level as an 8-hour TWA to protect workers in construction, maritime, and agriculture from the broad range of adverse effects associated with exposure to these substances. In the past, these particulates were often called "nuisance" or "inert" substances. These terms are misleading, however, because exposures to these substances in the workplace may cause serious and sometimes disabling effects. Further, good industrial hygiene and public health practice require that workplace exposures to particulates be maintained below the level associated with physical irritation, accidents, and respiratory effects.

PENTAERYTHRITOL

CAS: 115–77–5; Chemical Formula: $C(CH_2OH)_4$

H.S. No. 1305

In construction and maritime, OSHA currently has no separate limit for pentaerythritol, although this substance is covered by an 8-hour TWA PEL of 15 mg/m³ TWA, the Agency's generic total particulate limit in these sectors. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 10 mg/m³ for pentaerythritol (total dust) containing less than 1 percent quartz. For construction, maritime, and agriculture, OSHA is proposing PELs of 10 mg/m³ TWA (total particulate) and 5 mg/m³ (respirable particulate) for this substance. These are the limits recently established for pentaerythritol in general industry.

Pentaerythritol is an odorless, white crystalline solid. It is used in making insecticides, lubricants, alkyd resins, explosives, tall oil esters, pharmaceuticals, and paint swelling agents (ACGIH 1986, p. 462). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Rats exposed to pentaerythritol at 11,000 mg/m³ for 6 hours showed no ill effects from a single exposure, and rats, dogs, and guinea pigs exposed 6 hours daily for 90 days also showed no effects (Keplinger and Kay 1964/Ex. 1–743). The oral LD_{50}s in guinea pigs and mice were 11.3 and 22.5 g/kg, respectively; rats survived oral doses as high as 16 g/kg. At higher doses, animals displayed

diarrhea, tremors, ataxia, and loss of righting reflex (Keplinger and Kay 1964/Ex. 1–743). Daily applications of a saturated aqueous solution of technical pentaerythritol to rabbit skin produced no significant irritation; a single application of 10 g/kg aqueous paste on intact or abraded rabbit skin produced no evidence of percutaneous absorption (Keplinger and Kay 1964/Ex. 1–743; Hercules, Inc., as cited in ACGIH 1986/Ex. 1–3, p. 462). Instillation of a 50-percent aqueous suspension into the conjunctival sac of rabbits' eyes resulted in slight transient irritation (Hercules, Inc., as cited in ACGIH 1986/Ex. 1–3, p. 462).

Human volunteers are reported to have eliminated 85 percent of dietary pentaerythritol unchanged in the urine within 30 hours. A slight and transient increase in apparent blood sugar that was proportional to the ingested dose appeared in these subjects soon after administration (Berlow, Barth, and Snow 1958, as cited in ACGIH 1986/Ex. 1–3, p. 462).

In construction, maritime, and agriculture, OSHA is proposing 8-hour TWA PELs of 10 mg/m³ (total particulate) for pentaerythritol and 5 mg/m³ (respirable fraction). The Agency believes that these limits will protect employees in these sectors from the significant risks of physical irritation potentially associated with exposure to pentaerythritol at higher levels. OSHA believes that physical irritation constitutes a material impairment of health within the meaning of the Act. Promulgation of these limits will also make OSHA's PELs for this substance consistent across all regulated sectors.

PERLITE

CAS: None; Chemical Formula: None
H.S. No. 1310

In construction, maritime, and general industry, OSHA covers perlite under its generic total particulate limit of 15 mg/m³. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 10 mg/m³ for perlite as total dust containing less than 1 percent quartz. OSHA is proposing TWA PELs of 15 mg/m³ for perlite as total particulate containing less than 1 percent quartz and 5 mg/m³ for perlite as the respirable fraction. These are the limits recently established for perlite in general industry.

Perlite is a natural volcanic glass; it is essentially an amorphous mineral consisting of fused sodium-potassium-aluminum silicate. Perlite has a wide variety of uses, which include use as an abrasive; in plaster and tile; in fertilizer; in concrete construction aggregates; as a filter aid, pipe insulator, soil conditioner, cleaner base, and insulation board filler; in refractory products; and as tile mortar aggregate (Clayton and Clayton 1981, p. 3027; ACGIH 1986, p. 467).

Perlite is reported to have a free-silica content varying from zero to 3 percent (Anderson, Selvig, Baur et al. 1956 and the Perlite Institute, both as cited in ACGIH 1986/Ex. 1–3, p. 467). In its processed crude and expanded forms, perlite is reported to have a measurable quartz content of 0.4 percent quartz and 0.2 percent cristobalite (Sheckler 1977, as cited in ACGIH 1986/Ex. 1–3, p. 467). There are no published reports of adverse physiologic effects from exposure to perlite dust.

In construction, maritime, and agriculture, OSHA preliminarily finds that perlite is nontoxic when airborne total particulate concentrations are maintained at levels of 15 mg/m³ or below (as an 8-hour TWA) and when its quartz content is limited to a level below 1 percent crystalline silica. For these reasons, OSHA is proposing an 8-hour TWA PEL of 15 mg/m³ for total perlite dust containing less than 1 percent quartz and a 8-hour TWA PEL of 5 mg/m³ for the respirable fraction in these sectors. OSHA preliminarily concludes that these limits will protect workers from the significant risk of eye, skin, and other forms of physical irritation associated with exposure to this substance. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

PICLORAM

CAS: 1918–02–1; Chemical Formula: $C_6H_3Cl_3N_2O_2$
H.S. No. 1328

In construction and maritime, OSHA currently has no specific limit for picloram, although this substance has been covered by the Agency's generic total particulate limit of 15 mg/m³ (8-hour TWA). There is no PEL in agriculture. The 1987–1988 ACGIH TLV®s for this substance are 10 mg/m³ as an 8-hour TWA and 20 mg as a 15-minute STEL (both for total dust). In construction, maritime, and agriculture, OSHA is proposing a 10-mg/m³ 8-hour TWA PEL for picloram (total dust) and a 5-mg/m³ 8-hour TWA for the respirable fraction. These are the limits recently established in general industry.

Picloram is a white powder with an odor like that of chlorine. This substance is used as an herbicide and defoliant. It is used as a growth regulator on apricots, figs, and cherries and is used to control various weeds in the western states (ACGIH 1986, p. 489; HSDB 1986). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Picloram has low acute oral toxicity, with LD_{50} values of 3.75 g/kg for rats, 1.5 g/kg for mice, and 2.0 g/kg for rabbits (NIOSH 1979b, as cited in ACGIH 1986/Ex. 1–3, p. 489). Two-year feeding studies revealed no ill effects in albino rats and beagle dogs from ingestion of doses up to and including 150 mg/kg/day (McCollister and Leng 1969, as cited in ACGIH 1986/Ex. 1–3, p. 489). At 225 mg/kg/day, rats displayed moderate liver and kidney changes and, in females, slight body weight loss after 90 days. These authors (McCollister and Leng 1969, as cited in ACGIH 1986/Ex. 1–3, p. 489) also reported no fertility, reproduction, or lactation effects in albino rats fed at levels of up to 3000 ppm (0.3 percent) in a three-generational study. Although maternal toxicity in rats was reported at picloram dietary levels of 750 or 1000 mg/kg administered during days 6 through 15 of gestation, neither teratogenic nor neonatal effects were observed when subtoxic or maternally toxic doses of picloram were administered during organogenesis (Thomson et al. 1972, as cited in ACGIH 1986/Ex. 1–3, p. 489). The National Cancer Institute (NCI) (1977d) found a dose-related increase in benign liver tumors in female rats only and concluded that "under the conditions of the bioassay, the findings are suggestive of the ability of the compound to induce benign tumors in the livers of female Osborne-Mendel rats." Based on these results, NIOSH (Ex. 8–47, Table N4) concluded that "picloram is not a nuisance particulate and is not without toxic effects." OSHA notes that picloram must therefore be added to the list of substances formerly believed to be inert but subsequently shown to be toxic.

Based on this evidence, OSHA is proposing 8-hour TWA limits of 10 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction) for picloram in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that these total and respirable particulate limits will minimize the significant risk of material health impairment in the form of systemic effects, such as liver and kidney damage, that are potentially associated with exposure to this substance at higher levels. OSHA believes that these effects constitute material health impairments and that the proposed limits are necessary to substantially reduce these risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

PLASTER OF PARIS
CAS: 7778–18–9; Chemical Formula: $CaSO_4$
H.S. No. 1331

In construction and maritime, OSHA's current 8-hour TWA exposure limit for Plaster of Paris is 15 mg/m³ (total particulate). The ACGIH has a 10-mg/m³ TLV®–TWA for Plaster of Paris, measured as total dust. OSHA is retaining its current limit in construction and maritime and is proposing to extend this limit to agriculture, as well as the 5-mg/m³ limit for the respirable fraction. These are the limits recently established for this substance in general industry.

Plaster of Paris is a fine, white powder used in wall plasters and wallboard, in the building industry for tiles and blocks, in moldings and statuary, and in the paper industry (Merck 1983, p. 234).

Where occupational exposures to Plaster of Paris have been limited, no toxic effects or organic diseases of the lungs have occurred. Exposure to excessive levels of dust in the workplace may result in reduced visibility or injury to the skin or mucous membranes from the dust itself, or in damage to the skin from the rigorous skin-cleansing procedures required to remove the dust (ACGIH 1986/Ex. 1–3).

In construction and maritime, OSHA is retaining both the 8-hour TWA of 15 mg/m³ (total particulate) and the 8-hour TWA PEL of 5 mg/m³ (respirable particulate) for Plaster of Paris and is proposing to extend these limits to agriculture. The Agency preliminarily concludes that these limits will protect workers in these industries from the significant risks of skin, eye, and other forms of physical irritation associated with exposure to this substance. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

PORTLAND CEMENT
CAS: 65997–15–1; Chemical Formula: None
H.S. No. 1333

In construction and maritime, OSHA currently has a limit of 50 mppcf (approximately 15 mg/m³) for Portland cement containing less than 1 percent crystalline silica. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 10 mg/m³ for Portland cement as total dust containing less than 1 percent quartz. OSHA is proposing PELs of 10 mg/m³, measured as total particulate, for Portland cement in the construction, maritime, and agriculture industries and of 5 mg/m³, measured as the respirable fraction. These are the limits recently established in general industry.

Portland cement refers to a class of cements that consist of odorless gray powders containing less than 1 percent crystalline silica. Portland cement is insoluble in water and contains tri-and dicalcium silicate, in addition to varying amounts of alumina, tricalcium aluminate, and iron oxide. Portland cement is used to manufacture mortar for building blocks, stone and pre-cast items and as a moisture sealant for the exterior of concrete blocks (NIOSH/OSHA Occupational Health Guideline 1981, p. 3).

Intraperitoneal injection of Portland cement in guinea pigs produced an absorptive reaction, which is an effect typical of inert particulates. Portland cement is eventually eliminated from the tissue and is generally not considered harmful when ingested (Miller and Sayers 1941/Ex. 1–595).

In a study of industrial exposures, Gardner and associates (1939/Ex. 1–589) found no evidence of Portland-cement-related pneumoconiosis in 2,278 workers who had been heavily exposed to this substance for prolonged periods of time (Gardner, Durkan, Brumfiel, and Sampson 1939/Ex. 1–589). Conflicting reports of pneumoconiosis (Parmeggiani 1951, as cited in ACGIH 1986/Ex. 1–3, p. 494; Prosperi and Barsi 1957/Ex. 1–1093) are attributed to the presence of silica in the inhaled dust rather than to exposure to Portland cement itself (ACGIH 1986/Ex. 1–3, p. 494). Cement dermatitis does occur among exposed workers, however, as a consequence of the alkaline, abrasive, and hygroscopic properties of the wet cement, which cause irritation of the skin (Schwartz, Tulipan, and Birmingham 1957a/Ex. 1–1168).

In the construction, maritime, and agriculture industries, OSHA is proposing 8-hour TWA PELs of 10 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction) for Portland cement containing less than 1 percent quartz. The Agency preliminarily concludes that these limits will protect workers in these sectors against the significant risks associated with on-the-job exposures to Portland cement dust. These risks include eye, skin, and mucous membrane irritation, and may include more severe respiratory effects, all of which constitute material health impairments. Revising the total particulate limit to 10 mg/m³ as an 8-hour TWA will also simplify employee exposure monitoring for Portland cement, since gravimetric rather than impinger methods can then be used. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

ROUGE
CAS: None; Chemical Formula: None
H.S. No. 1351

In construction and maritime, OSHA currently has no specific limit for rouge, although rouge is covered under the Agency's generic total particulate standard of 15 mg/m³ as an 8-hour TWA. There is no PEL in agriculture. The ACGIH has an 8-hour TLV®–TWA limit of 10 mg/m³ for rouge as total dust containing less than 1 percent quartz. OSHA is proposing 10 mg/m³ as the 8-hour TWA PEL for the total particulate of rouge and 5 mg/m³ as the 8-hour TWA for the respirable fraction of rouge in construction, maritime, and agriculture. These are the limits recently established for these substances in general industry.

Rouge is a high-grade red pigment, composed mainly of ferric oxide, that is used as a polishing agent for glass, jewelry, etc., as an ingredient in pigments and magnetic tapes, and in metallurgy (ACGIH 1986, p. 325; Hawley's 1987, p. 1016).

There is evidence showing that exposure to hematite dust (ferric oxide) increases the risk of lung cancer in hematite miners, and this human evidence is consistent with the results of two recent animal studies: Warshawsky, Bingham, and Niemeier (1984), which showed that intratracheal administration of ferric oxide and exposure to benzo(a)pyrene (BaP) "enhances the metabolic activation of BaP"; and Niemeier, Mulligan, and Rowland (1986), which also found that ferric oxide has co-carcinogenic potential. In this rulemaking, OSHA's principal concern is to make the Agency's limits consistent across all regulated sectors; OSHA will consider rouge's carcinogenicity in the future in connection with the first PEL update rulemaking.

Accordingly, OSHA is proposing an 8-hour TWA PEL of 10 mg/m³ for the total particulate of rouge and an 8-hour TWA PEL of 5 mg/m³ for the respirable fraction in the construction, maritime, and agriculture industries. OSHA preliminarily concludes that these limits will protect workers in these sectors from the significant risks of physical irritation associated with workplace exposure to higher levels of rouge. These effects include eye, nose, and upper respiratory irritation and, perhaps, other more serious chronic diseases, all of which constitute material health impairments within the meaning of the Act. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

SILICON
CAS: 7440–21–3; Chemical Formula: Si
H.S. No. 1359

In construction and maritime, OSHA currently has no specific limit for silicon; however, silicon is covered under OSHA's generic particulate limits of 15 mg/m³ TWA (total particulate) and 5 mg/m³ (respirable fraction). There is no limit in agriculture. The ACGIH has a 10-mg/m³ 8-hour TLV®–TWA for silicon, measured as total dust. In construction, maritime, and agriculture, OSHA is proposing a particulate PEL for silicon of 10 mg/m³ as an 8-hour TWA, as well as the 5-mg/m³ respirable fraction limit. These are the limits recently established for this substance in general industry.

Silicon is a black to gray, lustrous, needle-like crystal that is used in the manufacture of semiconductors, transistors, for making alloys such as ferrosilicon and silicon copper, and as a reducing agent in high-temperature reactions (ACGIH 1986, p. 526(86)).

The evidence of silicon's toxicity in animals is conflicting. An early study by McCord, Fredrick, and Stolz (1937/Ex. 1–640) reported no response in guinea pigs and rats injected intraperitoneally with silicon. A more recent study (Schepers 1971/Ex. 1–570) demonstrated pulmonary lesions in rabbits administered an intratracheal dose of 25 mg silicon dust.

OSHA is proposing an 8-hour TWA PEL of 10 mg/m³ (total particulate) and an 8-hour TWA PEL of 5 mg/m³ (respirable fraction) for silicon in the construction, maritime, and agriculture sectors. The Agency preliminarily concludes that these limits will reduce the significant health risks potentially associated with exposure to this substance at higher levels. These risks include eye, skin, mucous membrane and other forms of physical irritation and may include chronic respiratory effects. OSHA believes that these effects constitute material health impairments and that the proposed PELs will substantially reduce the risk of these health effects. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

SILICON CARBIDE
CAS: 409–21–2; Chemical Formula: SiC
H.S. No. 1360

In construction and maritime, OSHA currently covers silicon carbide under its generic 15-mg/m³ total particulate limit. There is no limit in agriculture. The ACGIH TLV®–TWA is 10 mg/m³ over 8 hours, measured as total dust. In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA total particulate PEL for silicon carbide of 10 mg/m³ and an 8-hour TWA respirable fraction limit of 5 mg/m³. These are the limits recently established for this substance in general industry.

Silicon carbide is a green to blue-black iridescent crystal. It is used for polishing glass and granite, smoothing bisque ware, sharpening stones, and as an abrasive; it also finds use in the manufacture of porcelain, shoe soles, furnace linings, and antiskid pavements, and in semiconductor technology (Merck 1983, p. 1220).

An animal study (Gardner 1923/Ex. 1–737) showed that, although exposure to silicon carbide alone produced no fibrosis of the lungs, exposure of guinea pigs infected with tuberculosis to silicon carbide (six hours/day, five days/week for one year) aggravated pulmonary tuberculosis to the extent that extensive fibrosis occurred. Guinea pigs exposed to silicon carbide dust and infected with the tubercle bacteria developed tuberculopneumoconiotic lesions (Gross, Westrick, and McNerney 1959/Ex. 1–697). Miller and Sayers (1941/Ex. 1–595) observed that silicon carbide dust administered by intraperitoneal injection to guinea pigs produced no reaction.

Bruusgaard (1949/Ex. 1–1143) found that X-rays of 10 out of 32 workers exposed to average levels of 34 mppcf of silicon carbide for 15 years or more demonstrated pulmonary changes; these 10 workers were also tuberculin-positive. Miller, Davis, Goldman, and Wyatts (1953/Ex. 1–40) described three cases of pulmonary reactions and hyperglobinemia in tungsten carbide industry workers; these authors concluded that exposure to silicon carbide was not a hazard unless the exposed workers already had pulmonary tuberculosis. However, a recent case study (Hayashi and Kajita 1988) reports the case of a 36-year-old abrasives plant worker who developed cough and sputum and showed apical lesions on x-ray; the lesions were excised and were found to consist primarily of silicon carbide. The authors concluded that silicon carbide was responsible for this worker's pneumoconiosis. Another study (Osterman, Greaves, Smith, Hammond, Robins, and Theriault 1989) has shown that workers in a silicon carbide factory with long-term exposure develop parenchymal changes leading to fibrosis.

In construction, maritime, and agriculture, OSHA is proposing a 10-mg/m³ TWA total particulate limit for silicon carbide and a 5-mg/m³ TWA respirable fraction limit. The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant risk of material health impairment in the form of physical irritation and, perhaps, of respiratory effects, that are associated with exposure to this particulate. These are the limits recently established for silicon carbide in general industry.

STARCH
CAS: 9005–25–8; Chemical Formula: $(C_6H_{10}O_5)_n$
H.S. No. 1369

In construction and maritime, OSHA's limit for starch is 15 mg/m³ as an 8-hour TWA, the Agency's generic limit for all particulates. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 10 mg/m³ for starch as total dust that contains no asbestos and less than 1 percent crystalline silica. OSHA is retaining its current total dust and respirable fraction limits for starch in construction and maritime and is proposing to extend them to agriculture. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

Starch is a white, odorless powder. Starch has many uses, including use as an adhesive, textile filler and sizing agent, gelling agent, food product thickener, baking powder filler, fabric stiffener, indicator in analytical chemistry, anticaking agent, polymer base, and in face powders (Hawley's 1987, p. 1090).

Exposure to high concentrations of starch dust may result in impaired vision or may cause injury to the mucous membranes or skin. Injury may also result from the vigorous skin-cleansing procedures necessary for the complete removal of starch (ACGIH 1986/Ex. 1–3).

OSHA is retaining both the 8-hour TWA total particulate PEL of 15 mg/m³ and the 5-mg/m³ respirable particulate limit for starch in construction and maritime and is proposing these limits in agriculture. The Agency preliminarily concludes that these limits will reduce the significant risk of eye, skin, and other physical irritation that may result from exposure to high levels of starch in the workplace. In addition, promulgation of these limits will make OSHA's PELs for starch consistent across all regulated sectors.

SUCROSE
CAS: 57–50–1; Chemical Formula: $C_{12}H_{22}O_{11}$
H.S. No. 1374

In construction and maritime, OSHA's 8-hour TWA limit for sucrose is 15 mg/m³ as total particulate, the Agency's generic limit for all particulates. There is no limit in agriculture. The ACGIH includes sucrose among the group of

particulates that "do not produce significant organic disease or toxic effect when exposures are kept under reasonable control" (ACGIH 1986/Ex. 1–3) and has a TLV®–TWA limit of 10 mg/m³ for sucrose as total particulate containing no asbestos and less than 1 percent quartz. OSHA is retaining the 8-hour TWA PEL of 15 mg/m³ total particulate and the 8-hour TWA PEL 5 mg/m³ respirable fraction for sucrose in construction and maritime and is proposing these limits in agriculture. These are the limits recently established for sucrose in general industry.

Sucrose is found in the form of white crystals. It is used as a sweetening agent and food, as a starting material in fermentation, as a pharmaceutical preservative, and as an antioxidant, a demulcent, a glycerol substitute, a granulation agent, and an excipient for tablets (Merck 1983, p. 1273).

Exposure to excess levels of sucrose dust can cause skin and eye irritation, interference with vision, and distraction from the task at hand. The LD_{50} in rats is 29, 700 mg/kg (RTECS 1991). Acutely poisoned animals showed prostration, diarrhea, cyanosis, convulsions, and stupor before death (HSDB 1991).

OSHA is retaining its 8-hour total particulate TWA of 15 mg/m³ and its 8-hour TWA respirable fraction limit of 5 mg/m³ for sucrose in construction and maritime and is proposing to extend these limits to agriculture. The Agency preliminarily concludes that these limits will protect exposed workers in these sectors from the significant risk of physical irritation associated with sucrose exposure. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

2,4,5-T (TRICHLOROPHENOXYACETIC ACID)

CAS: 93–76–5; Chemical Formula: $C_8H_5Cl_3O$

H.S. No. 2148

In general industry, construction, and maritime, OSHA's permissible exposure limit for 2,4,5-T is 10 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 10 mg/m³ for 2,4,5-T. NIOSH has no REL for this substance but concurs (Ex. 8–47) with the limit being proposed. OSHA is proposing an 8-hour TWA limit of 10 mg/m³ for 2,4,5-T in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

2,4,5-T, which is 2,4,5-trichlorophenoxyacetic acid, is an odorless, colorless to tan solid. This substance is a phenoxy acid herbicide that is used to control woody plants (ACGIH 1986, p. 549; NIOSH/OSHA Occupational Health Guideline 1981, p. 1). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

2,4,5-T causes irritation, chloracne, and systemic effects in humans and central nervous system and teratogenic effects in animals. In early studies of 2,4,5-T, dioxin was a common contaminant; the role of dioxin must therefore always be considered in evaluating early reports of 2,4,5-T's toxicity (Proctor, Hughes, and Fischman 1988, p. 490). The oral LD_{50} values in rats, mice, dogs, guinea pigs, and hamsters are 300 mg/kg, 242 mg/kg, 100 mg/kg, 381 mg/kg, and 425 mg/kg, respectively (RTECS 1991). The dermal LD_{50} in rats is 1535 mg/kg (RTECS 1991). A single oral 2,4,5-T dose of 100 mg/kg given to pigs caused signs of anorexia, vomiting, diarrhea, and ataxia; at autopsy, hemorrhagic enteritis and congestion of the liver and kidney were seen Björklund and Erne 1966, in IARC 1977, Vol. 15, p. 280). In groups of rats maintained on diets containing 2,4,5-T, changes were observed in male and female animals receiving 100 mg/kg; these changes included a reduction in weight gain, a decrease in food intake, elevated serum alkaline phosphatase levels, and, in males, increased serum glutamic-pyruvic transaminase levels (IARC 1977, Vol. 15, p. 281). Dogs fed daily doses of 20 mg/kg 2,4,5-T died 11 to 75 days after dosing began (Drill and Hiratzka 1953, in IARC 1977, Vol. 15, p. 282). A dose of 100 mg/kg given subcutaneously to pregnant mice on the 6th through the 15th days of gestation produced an increased incidence of cleft palate in the offspring (Moore and Courtney 1971, in Proctor, Hughes, and Fischman 1988, p. 490). In hamsters administered oral doses of 100 mg/kg/day 2,4,5-T on days 6 to 10 of gestation, this substance caused signs of feticidal and teratogenic effects (Collins and Williams 1971, in Proctor, Hughes, and Fischman 1988, p. 490). A significant increase in the occurrence of cleft palate and embryotoxic effects was seen in mice after the oral administration of 35 to 130 mg/kg 2,4,5-T during days 6 to 15 of gestation (Roll 1971, in IARC 1977, Vol. 15, p. 285). Rats, rabbits, and monkeys appear to be relatively resistant to 2,4,5-T's teratogenic effects (Proctor, Hughes, and Fischman 1988, p. 490).

The effects most frequently reported in workers exposed to 2,4,5-T include chloracne, liver disorders, neurological and behavioral changes, fat metabolism disorders, and porphyria cutanea tarda (IARC 1977, Vol. 15, p. 288). However, the role of dioxin in the etiology of these disorders should not be overlooked. In 1964, an outbreak of chloracne affected 60 workers at a 2,4,5,-T factory of the Dow Chemical Company in Michigan (Firestone 1977, in IARC 1977, Vol. 15, p. 289). In 1969, employees of the same Michigan factory were reexamined and of 73 workers, 13 were found to have chloracne. Several of these workers had hyperpigmentation or facial hypertrichosis and one worker showed mild porphyrinuria. About 30 percent of these workers suffered from gastrointestinal symptoms such as nausea, vomiting, diarrhea, abdominal pain, and bloody stools; approximately 10 percent had symptoms including lower extremity weakness, headache, and decreased auditory acuity (Poland et al. 1971, in IARC 1977, Vol. 15, p. 289). In a factory in the USSR where 2,4,5-T was produced, 128 workers had skin lesions, and 69 of those examined had acne; many with severe lesions also suffered liver impairment. In 18 of these workers, a neurasthenic syndrome was observed (Telegina and Bikbulotova 1970, in IARC 1977, Vol. 15, p. 290). Among 228 people affected in an accident at a 2,4,5-T-producing factory in West Virginia, symptoms included chloracne, melanosis, muscular aches and pain, fatigue, nervousness, and cold intolerance (Firestone 1977, in IARC 1977, Vol. 15, p. 290). Eleven male volunteers showed no clinical effects after ingesting 5 mg/kg of 2,4,5-T; however, most did report having a metallic taste in the mouth which lasted 1 to 2 hours after ingestion (Gehring et al. 1973; Kohli et al. 1974a, in Hayes 1982, p. 530). A study of chemical application workers using 2,4,5-T in New Zealand revealed no significant differences in the rates of congenital defects, stillbirths, or miscarriages when compared with controls (Smith et al. 1981, in Proctor, Hughes, and Fischman 1988, p. 490). The International Agency for Research on Cancer has determined that the evidence for the carcinogenicity of 2,4,5-T in humans and animals is inadequate (IARC 1982, Suppl. 4, pp. 235–238).

Based on this evidence in humans and animals, OSHA preliminarily concludes that 2,4,5-T potentially causes irritation, neurological effects, and liver damage in exposed individuals. OSHA preliminarily finds that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed limit of 10 mg/m³ for 2,4,5-T in agriculture is necessary to significantly reduce these

risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for 2,4,5-T consistent across all regulated sectors.

TEMEPHOS

CAS: 3383–96–8; Chemical Formula: $C_{16}H_{20}O_6P_2S_3$

H.S. No. 1383

There is no specific limit for temephos in construction or maritime, although this substance is covered under OSHA's generic particulate limit of 15 mg/m³. There is no PEL in agriculture. The ACGIH limit is 10 mg/m³ (total dust) as an 8-hour TWA. In construction and maritime, OSHA is proposing an 8-hour TWA of 10 mg/m³ (total particulate) and is retaining the 5-mg/m³ limit for the respirable fraction of temephos dust; the Agency also proposes to extend both limits to agriculture. NIOSH (Ex. 8–47, Table N4) concurs with the selection of these PELs, which were recently established in general industry.

Temephos may be a white crystalline solid or a viscous brown liquid. Temephos is used as an insecticide for the control of mosquito larvae, flies, and midges (Clayton and Clayton 1981, p. 4807; ACGIH 1986, p. 557). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In rats and mice, temephos has an acute oral LD_{50} of 400 mg/kg or greater. Various animal species tolerated doses of 10 mg/kg without clinical effect and 1 mg/kg without effect on cholinesterase activity (Gaines, Kimbrough, and Laws 1967/Ex. 1–553). Laws, Morales, Hayes, and Joseph (1967/Ex. 1–562) revealed that human volunteers consuming oral doses of temephos at levels of either 256 mg/person/day for five days or 64 mg/person/day for four weeks evidenced no detectable effects on erythrocyte or plasma cholinesterase levels. Murphy and Cheever (1972/Ex. 1–567) reported that 1 mg of temephos per liter of drinking water produces no effect. These authors found that rat liver carboxylesterases were at least 30 times more sensitive to inhibition from temephos than were rat cholinesterases. Assuming that human liver carboxylesterases are proportionately more sensitive than cholinesterases, it is estimated that significant inhibition of these carboxylesterases could occur as a result of consuming 2 liters of drinking water containing 1 mg/L of temephos. Although nonspecific liver carboxylesterase is not critical for normal physiologic function, adverse effects on this enzyme could increase the susceptibility of exposed individuals to chemicals and drugs that contain carboxylesterase linkages (ACGIH 1986/Ex. 1–3, p. 557).

OSHA is proposing an 8-hour TWA limit of 10 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction) for temephos in construction, maritime, and agriculture. The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant risk of cholinesterase inhibition and reduction in carboxylesterase activity, which together constitute material health impairments within the meaning of the Act and are potentially associated with exposure to this substance. In addition, promulgation of these limits will make OSHA's PELs for temephos consistent across all regulated sectors.

4,4'-THIOBIS (6-TERT-BUTYL-n-CRESOL)

CAS: 96–69–5; Chemical Formula: $C_{22}H_{30}O_2S$

H.S. No. 1391

In construction and maritime, OSHA is currently covering 4,4'-thiobis under the Agency's generic total particulate limit of 15 mg/m³ TWA. There is no PEL in agriculture. The ACGIH limit is 10 mg/m³ as an 8-hour TWA, the limit established by the ACGIH for all of the nuisance dusts. OSHA proposes a 10-mg/m³ total particulate TWA limit and a 5-mg/m³ respirable fraction TWA PEL for 4,4'-thiobis in the construction, maritime, and agriculture industries, and NIOSH concurs (Ex. 8–47, Table N4) with the selection of these limits. These are the limits recently established for this substance in general industry.

4,4'-Thiobis is a light gray to tan powder with a slightly aromatic odor. This substance is an antioxidant for polyolefins, polyethylenes, and polypropylenes (ACGIH 1986, p. 570).

The intraperitoneal LD_{50} in mice is 50 mg/kg (RTECS 1991). The lethal oral dose in rats is approximately 5 g/kg (RTECS 1991). In a 30-day study, rats fed diets of 500 ppm 4,4'-thiobis exhibited normal weight gain; those rats fed five times this amount developed enlarged livers and had a reduced rate of weight gain (Lefaux 1968/Ex. 1–814). In a 90-day study reported by the same author, rats fed 50 ppm showed no toxic effects, but male rats fed 500 ppm ate and grew at a slightly lower rate. No pathologic changes were observed in the 500-ppm group. A dose of 5 g/kg of 4,4'-thiobis proved lethal to rats, with the predominant symptom being gastroenteritis.

In construction, maritime, and agriculture, OSHA is proposing exposure limits of 10 mg/m³ TWA (total particulate) and 5 mg/m³ TWA (respirable fraction) for 4,4'-thiobis. The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant risk of material health impairment, in the form of eye, skin, and other physical irritation, which is associated with exposure to this substance. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

TITANIUM DIOXIDE

CAS: 13463–67–7; Chemical Formula: TiO_2

H.S. No. 1396

In construction and maritime, OSHA's current PEL for titanium dioxide is 15 mg/m³ as an 8-hour TWA; this is the Agency's generic exposure limit for particulates. There is no PEL in agriculture. A 10-mg/m³ 8-hour TWA, measured as total dust, has been established by the ACGIH. The Agency is proposing 8-hour TWA PELs of 10 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) for titanium dioxide in construction, maritime, and agriculture; these are the limits recently established in general industry.

Titanium dioxide is a white crystalline solid. Titanium dioxide is used in paints, paper, rubber, plastics, cosmetics, floor coverings, glassware and ceramics, printing inks, and welding rods (ACGIH 1986, p. 576; Hawley's 1987, p. 1159).

Miller and Sayers (1941/Ex. 1–595) reported that titanium dioxide injected intraperitoneally in guinea pigs remained in the tissues at the injection site but did not produce a proliferative response. A study by Grandjean, Turrian, and Nicod (1956/Ex. 1–638) in which rats were administered 50 mg of titanium dioxide intratracheally showed pigmented dust deposits in the lungs. In addition, evidence of infection appeared in the alveoli of one rat and diffuse fibrosis was found in the lungs of another test animal. No nodule formation was observed (Grandjean, Turrian, and Nicod 1956/Ex. 1–638). Another study by Dale (1973/Ex. 1–624) revealed thickening of the walls of the alveoli in the lungs of rabbits injected with titanium dioxide dust; however, the lungs of these animals had returned to normal by three months post-treatment. Feeding studies of rats and mice at doses of 2.5 percent or 5 percent titanium dioxide for 103 weeks revealed no signs of carcinogenicity in either species (National Cancer Institute 1979d/Ex. 1–947).

In the prior rulemaking, NIOSH (Tr. p. 3–95) testified that exposure to this substance is associated with "a risk of cancer. . . . The incidence of tumors in

animals exposed to titanium dioxide (Lee, Trochimowicz, and Reinhardt 1985) meets the . . . criteria for . . . [a] potential occupational carcinogen." Because OSHA's primary objective in the proposed rulemaking is to make the Agency's PELs for individual substances consistent across all regulated sectors, OSHA is proposing for construction, maritime, and agriculture the same limits as those recently established for titanium dioxide in general industry. In the first PEL update project, however, OSHA will evaluate the toxicologic literature on this substance to determine whether further reduction in the PEL is warranted in all sectors.

Accordingly, OSHA is proposing at this time 8-hour TWA PELs of 10 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) for titanium dioxide in the construction, maritime, and agriculture industries. OSHA preliminarily concludes that these limits will protect workers in these sectors from the significant health risks associated with exposure to titanium dioxide at higher levels. These risks include material impairments of health in the form of eye, skin, and other physical irritation, and, perhaps, of carcinogenicity. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

VEGETABLE OIL MIST (EXCEPT CASTOR OIL, CASHEW NUT, OR SIMILAR IRRITANT OILS)

CAS: None; Formula: None

H.S. No. 1423

OSHA has no limit for vegetable oil mist in construction, maritime, or agriculture. The ACGIH has established a 10-mg/m³ 8-hour TLV®–TWA for all nuisance particulates. OSHA is proposing a 10 mg/m³ 8-hour TWA PEL for total particulate and a 5 mg/m³ 8-hour TWA PEL for the respirable fraction in the construction, maritime, and agriculture sectors. NIOSH concurs (Ex. 8–47, Table N4) with the proposed limits. These are also the limits recently established in general industry.

Vegetable oil is a pale yellow, oily liquid. Vegetable oils are used in paints, shortenings, food products, rubber softeners, dietary supplements, and as pesticide carriers (Hawley's 1987, p. 1219).

Oil mist presents the same safety and health hazards as all of the physical irritants. Occupational exposure to vegetable oil mist is associated with interference with vision, eye tearing, and skin and other forms of physical irritation.

In construction, maritime, and agriculture, OSHA is proposing 8-hour TWA limits of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) for vegetable oil mist (except castor oil, cashew nut, or similar irritant oils). The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant risks of physical irritation described above. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

ZINC STEARATE

CAS: 557–05–1; Chemical Formula: $Zn(C_{18}H_{35}O_2)_2$

H.S. No. 1434

In construction and maritime, OSHA currently covers zinc stearate under its generic total particulate limit of 15 mg/m³ TWA. There is no PEL in agriculture. The ACGIH has established an 8-hour TWA of 10 mg/m³ for zinc stearate, measured as total dust. OSHA is proposing 8-hour TWA PELs of 10 mg/m³ (total dust) and 5 mg/m³ (respirable fraction) for zinc stearate in the construction, maritime, and agriculture industries. NIOSH concurred (Ex. 8–47, Table N4) that these PELs were appropriate when OSHA established them recently in general industry.

Zinc stearate is a white hydrophobic powder. Zinc stearate is used in cosmetics, lacquers, and ointments. It is also used as a dusting powder, lubricant, mold-release agent, filler, heat and light stabilizer, in the manufacture of tablets and dietary supplements (Hawley's 1987, p. 1257; ACGIH 1986, p. 646).

A single intratracheal administration of 50 mg zinc stearate killed 50% of all exposed rats (Ueda et al. 1984). A report in *Folia Medica* (Volita and Noro 1957, as cited in ACGIH 1986/Ex. 1–3, p. 646) documented the case of a worker exposed to zinc stearate dust for 30 years who died from extensive fibrosis of the lungs. More recent studies have revealed incidences of pulmonary fibrosis associated with encephalopathy that stemmed directly from exposure to aluminum dust, which is frequently coated with stearic acid (British Journal of Industrial Medicine 1962, as cited in ACGIH 1986/Ex. 1–3, p. 646). Observations of long-term worker exposures to this dust in the rubber industry revealed no adverse effects of exposure (B.F. Goodrich Rubber Company, private communication, as cited in the ACGIH 1986/Ex. 1–3, p. 646).

In construction, maritime, and agriculture, OSHA is proposing a 10-mg/m³ TWA limit for zinc stearate (measured as total particulate) and a 5-mg/m³ TWA limit (measured as the respirable fraction). The Agency preliminarily concludes that these limits will prevent the significant health risks associated with workplace exposures to zinc stearate dust at higher levels. OSHA believes that the pulmonary effects potentially associated with exposure to zinc stearate constitute material impairments of health within the meaning of the Act and that the proposed limits are necessary to reduce this risk. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

ZINC OXIDE DUST

CAS: 1314–13–2; Chemical Formula: ZnO

H.S. No. 1438

In construction, maritime, and agriculture, OSHA has no exposure limit specifically for zinc oxide dust. The ACGIH has a TLV®–TWA of 10 mg/m³ for zinc oxide, measured as total dust. In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA of 10 mg/m³ (measured as total particulate) and an 8-hour TWA of 5 mg/m³ (measured as the respirable fraction). These are the limits recently established in general industry.

Zinc oxide dust is a white or pale yellow powder. Zinc oxide is used as a pigment and reinforcing agent in rubber, and ointments, in plastics, ceramics, floor tile, glass, as a food additive, dietary supplement, and in seed treatment, cosmetics, copier machines, photography and paints (Hawley's 1987, p. 1255; Merck 1983, p. 1457).

In contact with the skin of rabbits, zinc oxide causes mild irritation; in the eyes, it also causes a mild reaction (RTECS 1991). The LD_{50} in mice is 7950 mg/kg (RTECS 1991). According to Turner and Thompson (1926/Ex. 1–1124), exposure to finely divided zinc oxide dust can produce symptoms similar to those of metal fume fever. Beeckmans and Brown (1963/Ex. 1–775) reported that catalytically active zinc oxide dust is more toxic when treated with ultraviolet light. Metal fume fever, the principal health effect of zinc oxide exposure, causes chills, fever, dry cough, and substernal pain (Rohrs 1957). Leukocytosis occurs in severe attacks (McCord 1960). Several recent studies suggest that exposure to zinc oxide dust causes respiratory effects (Gupta, Pandey, Misra, and Viswanathan 1986; Lam, Conner, Rogers et al. 1985; and NIOSH 1975d). The PEL for zinc oxide may have to be reduced further if toxicological review at the time of OSHA's first PEL update indicates that further reduction is warranted.

In construction, maritime, and agriculture, OSHA is proposing limits of

10 mg/m³ TWA (total particulate) and 5 mg/m³ TWA (respirable particulate) for zinc oxide. The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant risk of material health impairment in the form of physical irritation and, perhaps, of respiratory effects. The Agency believes that the proposed PELs are necessary to reduce these risks. In addition, promulgation of these limits will make OSHA's PELs for zinc oxide consistent across all regulated sectors.

Preliminary Conclusions

OSHA's current generic 8-hour TWA particulate standard (29 CFR 1910.1000, Table Z–3) in construction and maritime was adopted from the 1970 ACGIH TLV®–TWA of 15 mg/m³ for total dust and 5 mg/m³ for respirable dust. At the time, the ACGIH considered the 15-mg/m³ value to be "an acceptable limit of good hygienic practice," based on the then-prevailing "lack of knowledge" of any adverse exposure-related effects at levels below this value (Documentation of the Threshold Limit Values and Biological Exposure Indices, ACGIH 1966/Ex. 1–13). Shortly after OSHA adopted the ACGIH's 1970 limit, the ACGIH revised its limit downward to 10 mg/m³ for total dust and 5 mg/m³ for respirable dust. In justifying this reduction, the ACGIH noted that the lower levels would "result in appreciable improvement of working conditions in plants where the old limit of 15 mg/m³ formerly prevailed" (Documentation of the Threshold Limit Values for Substances in Workroom Air, 3rd ed., p. 190, ACGIH 1971).

In this proposed rule, OSHA has preliminarily determined that it is appropriate to set a 10-mg/m³ total particulate limit and to retain the 5-mg/m³ respirable fraction limit for those particulates demonstrated to have, in addition to physical-irritant properties, specific adverse health effects. This is the policy OSHA followed in the recent air contaminants rulemaking for general industry. These substances will also be identified separately in Tables Z, Construction; Z, Shipyards; Z, Longshoring & Marine Terminals; and Z, Agriculture. For the substances in this section that are physical irritants but for which other health effects have not specifically been identified, OSHA is retaining the 8-hour TWA total particulate limit of 15 mg/m³ and the 8-hour TWA respirable fraction limit of 5 mg/m³ in construction and maritime and is proposing to extend these limits to agriculture. For those particulates not otherwise regulated, which includes all workplace particulates, both organic and inorganic that are not separately identified on Tables Z, Construction; Z, Shipyards; Z, Longshoring & Marine Terminals; and Z, Agriculture, OSHA is proposing a generic total particulate limit of 15 mg/m³. For all particulates, OSHA is retaining the 5-mg/m³ TWA limit for the respirable fraction in construction and maritime and is proposing this limit in agriculture. Promulgation of these limits will make OSHA's PELs for these substances consistent across all regulated sectors.

11. Substances for Which Proposed Limits Are Based on Analogy to Related Substances

Introduction

OSHA is proposing limits for 96 substances on the basis of their toxicologic and structural similarities to other chemical substances that create significant risks of systemic toxicity, ocular effects, kidney or liver damage, and other similarly adverse health effects. For many of these substances, OSHA has not previously had permissible exposure limits (PELs) in construction and maritime; for others, limits are currently in place in these sectors. The Agency has no PELs for any of these substances in agriculture. OSHA is proposing to establish PELs in construction, maritime, and agriculture that are identical to their limits in general industry. Promulgation of these PELs would thus make OSHA's limits for these substances consistent across all OSHA-regulated sectors.

Table C11–1 shows these substances, their CAS and HS numbers, and their 1987–1988 ACGIH TLV®s and NIOSH RELs. The table also shows the current limits for these substances in construction and maritime and the limits OSHA is proposing for them in construction, maritime, and agriculture.

TABLE C–11–1.—SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON ANALOGY TO RELATED SUBSTANCES

H.S. number/chemical name	CAS No.	Current OSHA PEL in construction and maritime *	1987–1988 ACGIH TLV®**	NIOSH REL ***	Proposed OSHA PEL in construction, maritime, and agriculture *
1003 Acetic anhydride	108–24–7	5 ppm TWA	5 ppm Ceiling		5 ppm Ceiling
1009 Acrylic acid	79–10–7		10 ppm TWA		10 ppm TWA, Skin
1015 Aluminum (alkyls)	7429–90–5		2 mg/m³ TWA		2 mg/m³ TWA
1018 Aluminum (soluble salts)	7429–90–5		2 mg/m³ TWA		2 mg/m³ TWA
2006 sec-Amyl acetate	626–38–0	125 ppm TWA	125 ppm TWA		125 ppm TWA
1040 Boron tribromide	10294–33–4	1 ppm TWA	1 ppm Ceiling		1 ppm Ceiling
1043 Bromine pentafluoride	7789–30–2	0.1 ppm TWA	0.1 ppm TWA		0.1 ppm TWA
2022 sec-Butyl acetate	105–46–4	200 ppm TWA	200 ppm TWA		200 ppm TWA
2023 tert-Butyl acetate	540–88–5	200 ppm TWA	200 ppm TWA		200 ppm TWA
1048 Butyl acrylate	141–32–2		10 ppm TWA		10 ppm TWA
1055 o-sec-Butylphenol	89–72–5		5 ppm TWA, Skin		5 ppm TWA, Skin
1059 Calcium hydroxide	1305–62–0		5 mg/m³ TWA		5 mg/m³ TWA
1060 Calcium oxide	1305–78–8	5 mg/m³ TWA	2 mg/m³ TWA		5 mg/m³ TWA
1074 Carbonyl fluoride	353–50–4		2 ppm TWA		2 ppm TWA
			5 ppm STEL		5 ppm STEL
1075 Catechol (pyrocatechol)	120–80–9		5 ppm TWA		5 ppm TWA, Skin
1081 1-Chloro-1-nitropropane	600–25–9	20 ppm TWA	2 ppm TWA		2 ppm TWA
1098 Cobalt carbonyl (as Co)	10210–68–1		0.1 mg/m³ TWA		0.1 mg/m³ TWA
1099 Cobalt hydrocarbonyl (as Co)	16842–03–8		0.1 mg/m³ TWA		0.1 mg/m³ TWA
2042 Cresol (all isomers)	1319–77–3	5 ppm TWA, Skin	5 ppm TWA, Skin	2.3 ppm TWA	5 ppm TWA, Skin
2043 Crotonaldehyde	123–73–9; 4170–30–3	2 ppm TWA	2 ppm TWA		2 ppm TWA
2049 Demeton (Systox)	8065–48–3	0.1 mg/m³ TWA, Skin.	0.1 mg/m³ TWA, Skin.		0.1 mg/m³ TWA, Skin
1118 Diazinon	333–41–5		0.1 mg/m³ TWA, Skin.		0.1 mg/m³ TWA, Skin
1121 1,1-Dichloro-1-nitroethane	594–72–9	10 ppm Ceiling	2 ppm TWA		2 ppm TWA

TABLE C–11–1.—SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON ANALOGY TO RELATED SUBSTANCES—Continued

H.S. No./chemical name	CAS No.	Current OSHA PEL in construction and maritime *	1987–1988 ACGIH TLV®**	NIOSH REL ***	Proposed OSHA PEL in construction, maritime, and agriculture *
1125 p-Dichlorobenzene	106–46–7	75 ppm TWA	75 ppm TWA; 110 ppm STEL		75 ppm TWA; 110 ppm STEL
1128 Dichloromonofluoromethane	75–43–4	1000 ppm TWA	10 ppm TWA		10 ppm TWA
1135 Diethyl ketone	96–22–0		200 ppm TWA		200 ppm TWA
1138 Diethylene triamine	111–40–0		1 ppm TWA, Skin		1 ppm TWA
2071 Dinitrobenzene (all isomers)	528–29–0; 99–65–0; 100–25–4	1 mg/m³ TWA, Skin	1 mg/m³ TWA, Skin		1 mg/m³ TWA, Skin
2072 Dinitrotoluene	25321–14–6	1.5 mg/m³ TWA, Skin.	1.5 mg/m³ TWA, Skin.	(†)	1.5 mg/m³ TWA, Skin
1148 Dipropyl ketone	123–19–3		50 ppm TWA		50 ppm TWA
1150 Diquat	85–00–7		0.5 mg/m³ TWA		0.5 mg/m³ TWA
1152 Disulfoton	298–04–4		0.1 mg/m³ TWA		0.1 mg/m³ TWA, Skin
1154 Divinyl benzene	1321–74–0		10 ppm TWA		10 ppm TWA
1156 Endosulfan	115–29–7		0.1 mg/m³ TWA, Skin.		0.1 mg/m³ TWA, Skin
2074 EPN	2104–64–5	0.5 mg/m³ TWA, Skin.	0.5 mg/m³ TWA, Skin.		0.5 mg/m³ TWA, Skin
1181 Fonofos	944–22–9		0.1 mg/m³ TWA, Skin.		0.1 mg/m³ TWA, Skin
1182 Formamide	75–12–7	20 ppm TWA	20 ppm TWA 30 ppm STEL		20 ppm TWA; 30 ppm STEL
1186 Germanium tetrahydride	7782–65–2		0.2 ppm TWA		0.2 ppm TWA
1212 Indene	95–13–6	10 ppm TWA	10 ppm TWA		10 ppm TWA
1214 Iodoform	75–47–8		0.6 ppm TWA		0.6 ppm TWA
2097 Isobutyl acetate	110–19–0	150 ppm TWA	150 ppm TWA. 187 ppm STEL		150 ppm TWA
1219 Isobutyl alcohol	78–83–1	100 ppm TWA	50 ppm TWA		50 ppm TWA
1220 Isooctyl alcohol	26952–21–6		50 ppm TWA, Skin		50 ppm TWA, Skin
1229 n-Isopropylaniline	768–52–5		2 ppm TWA, Skin		2 ppm TWA, Skin
1231 Ketene	463–51–4	0.5 ppm TWA	0.5 ppm TWA 1.5 ppm STEL		0.5 ppm TWA 1.5 ppm STEL
1244 Methacrylic acid	79–41–4		20 ppm TWA		20 ppm TWA, Skin
1247 4-Methoxyphenol	150–76–5		5 mg/m³ TWA		5 mg/m³ TWA
1250 Methyl acetylene-propadiene mixture (MAPP)		1000 ppm TWA	1000 ppm TWA 1250 ppm STEL		1000 ppm TWA 1250 ppm STEL
2108 Methylal (dimethoxymethane)	109–87–5	1000 ppm TWA	1000 ppm TWA		1000 ppm TWA
2110 Methylamine	74–89–5	10 ppm TWA	10 ppm TWA		10 ppm TWA
1256 Methyl demeton	8022–00–2		0.5 mg/m³ TWA, Skin.		0.5 mg/m³ TWA, Skin
2109 Methylene bisphenyl isocyanate (MDI)	101–68–8	0.02 ppm Ceiling	0.02 ppm Ceiling	0.005 ppm TWA 0.02 ppm Ceiling (10-min).	0.02 ppm Ceiling
1257 Methyl ethyl ketone peroxide (MEKP)	1338–23–4		0.2 ppm Ceiling		0.7 ppm Ceiling
1258 Methyl formate	107–31–3	100 ppm TWA	100 ppm TWA. 150 ppm STEL		100 ppm TWA 150 ppm STEL
1259 Methyl iodide	74–88–4	5 ppm TWA, Skin	2 ppm TWA, Skin, A2.	(†)	2 ppm TWA, Skin
1260 Methyl isoamyl ketone	110–12–3	100 ppm TWA	50 ppm TWA	50 ppm TWA	50 ppm TWA
1262 Methyl isopropyl ketone	563–80–4		200 ppm TWA		200 ppm TWA
1265 Methyl parathion	298–00–0		0.2 mg/m³ TWA, Skin.	0.2 mg/m³ TWA	0.2 mg/m³ TWA, Skin
1268 Methylcyclohexane	108–87–2	500 ppm TWA	400 ppm TWA		400 ppm TWA
1271 2-Methylcyclopentadienyl manganse tricarbonyl Skin (as Mn).	12108–13–3		0.2 mg/m³ TWA, Skin.		0.2 mg/m³ TWA, Skin
1279 Monocrotophos (Azodrin)	6923–22–4		0.25 mg/m³ TWA		0.25 mg/m³ TWA
1281 Morpholine	110–91–8	20 ppm TWA, Skin	20 ppm TWA 30 ppm STEL, Skin		20 ppm TWA 30 ppm STEL, Skin
1286 Nitric acid	7697–37–2	2 ppm TWA	2 ppm TWA 4 ppm STEL	2 ppm TWA	2 ppm TWA 4 ppm STEL
1287 p-Nitroaniline	100–01–6	6 mg/m³ TWA, Skin	3 mg/m³ TWA, Skin		3 mg/m³ TWA, Skin
2120 Nitromethane	75–52–5	100 ppm TWA	100 ppm TWA		100 ppm TWA
1292 Nitrotoluene; o-isomer, m-isomer, p-isomer	88–72–2, 99–08–1, 99–99–0	5ppm TWA, Skin	2 ppm TWA, Skin		2 ppm TWA, Skin
1293 Nonane	111–84–2		200 ppm TWA		200 ppm TWA
1299 Oxalic acid	144–62–7	1 mg/m³ TWA	1 mg/m³ TWA 2 mg/m³ STEL		1 mg/m³ TWA 2 mg/m³ STEL
1309 Perchloryl fluoride	7616–94–6	3 ppm TWA	3 ppm TWA 6 ppm STEL		3 ppm TWA 6 ppm STEL
1320 Phosdrin (Mevinphos)	7786–34–7	0.1 mg/m³ TWA, Skin.	0.01 ppm TWA 0.03 ppm STEL, Skin.		0.01 ppm TWA 0.03 ppm STEL, Skin
1323 Phosphorus oxychloride	10025–87–3		0.1 ppm TWA 0.5 ppm STEL		0.1 ppm TWA
1324 Phosphorus pentasulfide	1314–80–3	1 mg/m³ TWA	1 mg/m³ TWA 3 mg/m³ STEL		1 mg/m³ TWA 3 mg/m³ STEL

TABLE C–11–1.—SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON ANALOGY TO RELATED SUBSTANCES—Continued

H.S. No./chemical name	CAS No.	Current OSHA PEL in construction and maritime *	1987–1988 ACGIH TLV***	NIOSH REL ****	Proposed OSHA PEL in construction, maritime, and agriculture *
1326 Phthalic anhydride	85–44–9	2 ppm TWA	1 ppm TWA		1 ppm TWA
2132 Pindone (2-pivalyl-1,3-indandione)	83–26–1	0.1 mg/m³ TWA	0.1 mg/m³ TWA		0.1 mg/m³ TWA
1335 Propargyl alcohol	107–19–7	1 ppm TWA, Skin	1 ppm TWA, Skin		1 ppm TWA, Skin
1336 Propionic acid	79–09–4		10 ppm TWA		10 ppm TWA
			15 ppm STEL		
1338 n-Propyl acetate	109–60–4	200 ppm TWA	200 ppm TWA		200 ppm TWA
			250 ppm STEL		250 ppm STEL
1339 n-Propyl alcohol	71–23–8	200 ppm TWA	200 ppm TWA		200 ppm TWA
			250 ppm STEL, Skin		250 ppm STEL
2135 Propyleneimine	75–55–8	2 ppm TWA, Skin	2 ppm TWA, Skin, A2		2 ppm TWA, Skin
1344 Propylene oxide	75–56–9	100 ppm TWA	20 ppm TWA		20 ppm TWA
2141 Selenium hexafluoride (as Se)	7783–79–1	0.05 ppm TWA	0.05 ppm TWA		0.05 ppm TWA
1361 Silicon tetrahydride	7803–62–5		5 ppm TWA		5 ppm TWA
2144 Stibine	7803–52–3	0.1 ppm TWA	0.1 ppm TWA		0.1 ppm TWA
1379 Sulfuryl fluoride	2699–79–8	5 ppm TWA	5 ppm TWA		5 ppm TWA
			10 ppm STEL		10 ppm STEL
2149 TEDP (Sulfotep)	3689–24–5	0.2 mg/m³ TWA, Skin.	0.2 mg/m³ TWA, Skin.		0.2 mg/m³ TWA, Skin
2151 Tellurium hexafluoride (as Te)	7783–80–4	0.02 ppm TWA	0.02 ppm TWA		0.02 ppm TWA
2152 TEPP	107–49–3	0.05 mg/m³ TWA, Skin.	0.05 mg/m³ TWA, Skin.		0.05 mg/m³ TWA, Skin
2159 Thallium soluble compounds (as Tl)	7440–28–0	0.1 mg/m³ TWA, Skin.	0.1 mg/m³ TWA, Skin.		0.1 mg/m³ TWA, Skin
1393 Thionyl chloride	7719–09–7		1 ppm Ceiling		1 ppm Ceiling
1402 Tributyl phosphate	126–73–8	5 mg/m³ TWA	2.5 mg/m³ TWA		2.5 mg/m³ TWA
1404 Trichloroacetic acid	76–03–9		1 ppm TWA		1 ppm TWA
2162 1,1,2-Trichloroethane	79–00–5	10 ppm TWA, Skin	10 ppm TWA, Skin	(†)	10 ppm TWA, Skin
1411 Trimethylamine	75–50–3		10 ppm TWA		10 ppm TWA
			15 ppm STEL		15 ppm STEL
1420 n-Valeraldehyde	110–62–3		50 ppm TWA		50 ppm TWA
1432 m-Xylene-alpha, alpha'-diamine	1477–55–0		0.1 mg/m³ Ceiling, Skin.		0.1 mg/m³ Ceiling, Skin
1433 Xylidine	1300–73–8	5 ppm TWA, Skin	2 ppm TWA, Skin		2 ppm TWA, Skin

* OSHA's PELs do not currently apply in Agriculture; OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TLV®–TWA is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time. An A2 designation means that the ACGIH has designated the substance a suspected human carcinogen.

† NIOSH TWA limits are for 10 hour/day, 40 hour/week exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

†688 NIOSH considers this substance a potential occupational carcinogen and recommends that exposures be reduced to the lowest feasible concentration.

Description of the Health Effects

The health effects associated with occupational exposures to the diverse group of substances shown in Table C11–1 vary widely, ranging from sensory irritation, systemic toxicity, ocular effects, and neuropathy to renal and liver damage. This variation in target organs reflects the fact that the substances in this group have not been grouped on the basis of similarity in toxic effects, target organs, or mechanism of action; instead, they are considered together because the specific limits being proposed for them have been set on the basis of toxic effects observed after exposure to analogous chemicals. Table C11–2 shows these substances, along with their adverse health effects and the substances with which they share structural and toxicological similarities.

TABLE C11–2.—SUMMARY OF RATIONALE FOR PROPOSED LIMITS BASED ON ANALOGY TO RELATED SUBSTANCES

H.S. No./chemical name	Substance with analogous structure or activity	Associated health effects
1003 Acetic anhydride	Acetic acid	Sensory irritation.
1009 Acrylic acid	Acetic acid	Sensory irritation.
1015 Aluminum (alkyls)	Welding fumes	Pulmonary irritation.
1018 Aluminum (soluble salts)	Hydrogen chloride	Sensory irritation.
2006 sec-Amyl acetate	Amyl acetates	Sensory irritation.
1040 Boron tribromide	Hydrogen bromide	Sensory irritation.
1043 Bromine pentafluoride	Chlorine trifluoride	Systemic injury.
2022 sec-Butyl acetate	Acetates	Sensory irritation.
2023 tert-Butyl acetate	Acetates	Sensory irritation.
1048 Butyl acrylate	Methyl acrylate	Sensory irritation.
1055 o-sec-Butylphenol	Phenol and cresol	Respiratory, liver, and kidney effects.
1059 Calcium hydroxide	Sodium hydroxide	Sensory irritation.
1060 Calcium oxide	Sodium hydroxide	Sensory irritation.
1074 Carbonyl fluoride	Hydrolysis to hydrogen fluoride	Sensory irritation.
1075 Catechol	Phenol	Peripheral vaso-constriction, renal tubule oegeneration.

TABLE C11-2.—SUMMARY OF RATIONALE FOR PROPOSED LIMITS BASED ON ANALOGY TO RELATED SUBSTANCES—Continued

H.S. No./chemical name	Substance with analogous structure or activity	Associated health effects
1081 1-Chloro-1-nitropropane	Nitropropane	Damage to heart muscle, liver, and kidneys.
1098 Cobalt carbonyl	Nickel carbonyl	Systemic toxicity.
1099 Cobalt hydrocarbonyl	Nickel carbonyl	Systemic toxicity.
2042 Cresol (all isomers)	Phenol	Central nervous system effects.
2043 Crotonaldehyde	Acrolein	Sensory irritation, sensitization.
2049 Demeton	Parathion	Cholinesterase inhibition.
1118 Diazinon	Parathion	Cholinesterase inhibition.
1121 1,1-Dichloro-1-nitroethane	Related compounds	Systemic toxicity.
1125 p-Dichlorobenzene	o-Dichlorobenzene	Neurological effects, cataract formation.
1128 Dichloromonofluoromethane	Chloroform	Hepatotoxicity, cardiac sensitization.
1135 Diethyl ketone	Methyl propyl ketone	Narcosis.
1138 Diethylene triamine	Ethylamine	Irritation, sensitization.
2071 Dinitrobenzene (all isomers)		Aniline Cyanosis.
2072 Dinitrotoluene	Nitro- and Dinitrobenzenes	Methemoglobinemia.
1148 Dipropyl ketone	Methyl isobutyl ketone	Narcosis.
1150 Diquat	Paraquat	Ocular effects.
1152 Disulfoton	Parathion	Cholinesterase inhibition.
1154 Divinyl benzene	Styrene	Sensory irritation.
1156 Endosulfan	Aldrin, Dieldrin	Neurological effects.
2074 EPN	Parathion	Cholinesterase inhibition.
1181 Fonofos	Ethyl parathion	Cholinesterase inhibition.
1182 Formamide	Dimethyl formamide	Testicular toxicity, teratogenicity.
1186 Germanium tetrahydride	Stibine	Hemolytic effects.
1212 Indene	Naphthalene	Sensory irritation.
1214 Iodoform	Methyl iodide	Irritation, hepatotoxicity.
2097 Isobutylacetate	n-Butyl acetate	Sensory irritation.
1219 Isobutyl alcohol	n-Butyl alcohol	Irritation, narcosis.
1220 Isooctyl alcohol	Isoamyl alcohol	Sensory irritation.
1229 n-Isopropylaniline	Aniline, N,N-dimethylaniline	Hemolytic effects.
1231 Ketene	Phosgene	Sensory irritation.
1244 Methacrylic acid	Acrylic acid	Sensory irritation.
1247 4-Methoxyphenol	Hydroquinone	Ocular effects.
1250 Methyl acetylene-propadiene mixture	Methyl acetylene	Pulmonary effects.
2108 Methylal		Systemic toxicity.
2110 Methylamine	Ethylamine	Sensory irritation.
1256 Methyl demeton	Demeton	Ocular effects, respiratory effects, inner ear irritation.
2019 Methylene bisphenyl isocyanate (MDI)	Toluene diisocyanate	Sensitization.
1257 Methyl ethyl ketone peroxide (MEKP)	Benzoyl peroxide, hydrogen peroxide	Sensory irritation.
1258 Methyl formate	Methyl acetate	Sensory irritation.
1259 Methyl iodide	Methyl bromide	Central nervous system effects.
1260 Methyl isoamyl ketone	Methyl isobutyl ketone	Neuropathy.
1262 Methyl isopropyl ketone	Diethyl ketone, methyl propyl ketone	Narcosis, sensory irritation.
1265 Methyl parathion	Parathion	Cholinesterase inhibition.
1268 Methylcyclohexane	Heptane	Irritation.
1271 Methylcyclopentadienyl manganese tricarbonyl	Tetraethyl lead	Central nervous system effects, chronic lung effects.
1279 Monocrotophos (Azodrin)	Parathion	Cholinesterase inhibition.
1281 Morpholine	Ammonia	Kidney and liver degeneration, sensory irritation.
1286 Nitric acid	Hydrogen chloride, sulfuric acid	Sensory irritation.
1287 p-Nitroaniline	Aniline	Methemoglobin formation.
2120 Nitromethane	Nitroethane	Sensory irritation.
1292 Nitrotoluene (all isomers)	Aniline	Methemoglobin formation.
1293 Nonane	Octane	Narcosis.
1299 Oxalic acid	Sulfuric acid, phosphoric acid	Irritation, burns.
1309 Perchloryl fluoride	Fluoride	Fluorosis.
1320 Phosdrin (Mevinphos)	Parathion	Cholinesterase inhibition.
1323 Phosphorus oxychloride	Phosphorous trichloride	Sensory irritation, respiratory effects.
1234 Phosphorus pentasulfide	Phosphoric acid	Sensory irritation.
1326 Phthalic anhydride	Tetrachlorophthalic anhydride, maleic anhydride	Sensory irritation.
2132 Pindone	Warfarin	Inhibition of blood clotting.
1335 Propargyl alcohol	Allyl alcohol	Sensory irritation.
1336 Propionic acid	Acetic acid	Sensory irritation.
1338 n-Propyl acetate	Isopropyl acetate, n-butyl acetate	Sensory irritation.
1339 n-Propyl alcohol	Isopropyl alcohol	Sensory irritation.
2135 Propyleneimine	Ethyleneimine	Carcinogenicity.
1344 Propylene oxide	Ethylene oxide	Central nervous system depression, sensory irritation.
2141 Selenium hexafluoride	Ozone	Respiratory irritation.
1361 Silicon tetrahydride	Germane, stannane	Sensory irritation.
2144 Stibine	Arsine	Hemolysis.
1379 Sulfuryl fluoride	Hydrogen fluoride	Fluorosis.
2149 TEDP (Sulfotep)	Parathion	Cholinesterase inhibition.
2151 Tellurium hexafluoride	Ozone	Respiratory irritation.
2152 TEPP	Parathion	Cholinesterase inhibition.
2159 Thallium, soluble compounds	Other metals	Neurological effects.
1393 Thionyl chloride	Hydrogen chloride	Sensory irritation.
1402 Tributyl phosphate	Triphenyl phosphate	Narcosis, cholinesterase inhibition.
1404 Trichloroacetic acid	2,2-Dichloropropionic acid	Sensory irritation.
2162 1,1,2-Trichloroethane	Chloroform	Liver damage.

TABLE C11-2.—SUMMARY OF RATIONALE FOR PROPOSED LIMITS BASED ON ANALOGY TO RELATED SUBSTANCES—Continued

H.S. No./chemical name	Substance with analogous structure or activity	Associated health effects
1411 Trimethylamine	Dimethylamine	Sensory irritation.
1420 n-Valeraldehyde	Saturated aliphatic aldehydes	Sensory irritation.
1432 m-Xylene-alpha,alpha', diamine	Phenylenediamine	Allergic respiratory sensitization
1433 Xylidine	Aniline	Methemoglobin formation.

The use of structural analogy is a reasonable methodology for limit-setting because of the similarities in structure and activity between each substance in this group and at least one other toxic substance. Industrial hygienists and toxicologists frequently use this approach when dealing with lesser known substances either in the workplace or the laboratory. The limits for the substances in this group are being proposed based on dose-response information for other compounds that have a similar chemical structure or are known to have a similar mechanism of action. For example, limits are being proposed for a number of substances that are known cholinesterase inhibitors (including diazinon, disulfoton, and monocrotophos); however, since direct dose-response data are not available for these substances specifically, OSHA has proposed limits that are similar to the proposed limit for parathion, another cholinesterase inhibitor for which adequate dose-response data are available.

It is important to note that the setting of a limit on the basis of analogy to other substances does not mean that no information is available to demonstrate that the substance is toxic; acute animal data are available to demonstrate the toxicity of all of the substances for which limits are being proposed in this category, and, for several substances, there are case reports of human poisonings caused by exposure. Thus, the limits being proposed for these substances reflect much more than a theoretical consideration of chemical structure and physiologic reaction: the hazardous nature of exposure to every substance in this category has been demonstrated beyond doubt, although the precise level at which these effects will occur has not been established with certainty.

The following sections describe OSHA's preliminary findings for the substances in this group. They also discuss the material health impairments likely to occur among workers in construction, maritime, and agriculture as a consequence of occupational exposure to these substances.

ACETIC ANHYDRIDE

CAS: 108-24-7; Chemical Formula: $(CH_3CO)_2O$

H.S. No. 1003

The OSHA PEL for acetic anhydride in construction and maritime operations is 5 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV® of 5 ppm as a ceiling, based on analogy with acetic acid's (TLV=5 ppm ceiling) irritant potential. NIOSH has no REL but concurs (Ex. 8-47, Table N1) with the limit being proposed. In construction, maritime, and agriculture, OSHA is proposing a 5 ppm ceiling for acetic anhydride. This is the limit recently established for this substance in general industry.

Acetic anhydride is a colorless, mobile, strongly refractive liquid with a strong odor. Acetic anhydride is used in the manufacture of plastics and cellulose acetate fibers, in the synthesis of vinyl acetate, and as a dehydrating and acetylating agent in the production of insecticides, pharmaceuticals, dyes, explosives, perfumes, and aspirin (ACGIH 1986, p. 5).

In one study, rats inhaling 1000 ppm of acetic anhydride for 4 hours survived, but 2000 ppm was fatal (Smyth 1956/Ex. 1-759). In human studies, eye, nose, and throat irritation has been observed, and it has been suggested that bronchial and lung injury may occur as a consequence of exposure (Henderson and Haggard 1943j, as cited in ACGIH 1986/Ex. 1-3, p. 5). Skin burns and serious corneal injury have been reported in industrial settings when workers came into contact with the liquid (McLaughlin 1946/Ex. 1-641), and acetic anhydride is a marked lacrimator (Fairhall 1949b, as cited in ACGIH 1986/Ex. 1-3, p. 5).

In light of acetic anhydride's potential for acute toxicity, OSHA is proposing a 5-ppm ceiling limit in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors from the significant risk of ocular and respiratory effects associated with high, short-term exposures to acetic anhydride. OSHA believes that ocular and respiratory effects constitute material impairments of health. The proposed limit will substantially reduce these risks among workers exposed to this substance in these sectors. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ACRYLIC ACID

CAS: 79-10-7; Chemical Formula: $CH_2{=}CHCO_2H$

H.S. No. 1009

OSHA has no permissible exposure limit for acrylic acid in construction, maritime, or agriculture operations. The ACGIH has an 8-hour TLV®-TWA of 10 ppm. NIOSH has no REL. OSHA is proposing an 8-hour TWA of 10 ppm, with a skin notation, for acrylic acid in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Acrylic acid is a colorless, corrosive liquid with a distinctive acrid odor. Acrylic acid is known to polymerize explosively with amines, ammonia, oleum, and chlorosulfonic acid, and it is incompatible with strong alkalis and pure nitrogen. Occupational exposure to acrylic acid usually occurs when the chemical is used in the form of methyl, ethyl, or butyl esters in the manufacture of acrylic resins. It is also used in coatings, paints, adhesives, polishes, general finishes, and in binders (ACGIH 1986, p. 14.1(87)).

Data indicate that the oral LD_{50} in rats is between 0.25 and 0.5 mg/kg (Dow Chemical Company 1977f, as cited in ACGIH 1986/Ex. 1-3, p. 14), and the skin absorption LD_{50} in rabbits is 0.95 ml/kg (Smyth, Carpenter, Weil et al. 1962/Ex. 1-441). Another study indicates that rabbits given acrylic acid orally showed no ill effects at a level of 0.025 mg/kg (Klimkina et al. 1969, as cited in ACGIH 1986/Ex. 1-3, p. 14), and Gage (1970/Ex. 1-318) reports that rats exposed to 80 ppm for 6 hours daily for 20 days showed no adverse effects.

Case reports indicate that acute exposures to acrylic acid in workers have caused skin burns, eye burns, and upper respiratory tract effects (ACGIH 1986/Ex. 1-3, p. 14). NIOSH (Ex. 8-47, Table N2) believes that the PEL for acrylic acid should be lower than the limit being proposed, based on recent studies demonstrating degeneration of the nasal mucosa, changes in pulmonary function, and skin absorption in animals exposed to this substance (Miller, Ayres, Jersey, and Mckenna 1981 and Silver,

Leith, and Murphy 1981, both as cited in ACGIH 1986/Ex. 1–3, p 14.1). OSHA is aware of the recent literature on acrylic acid; however, the primary objective of this rulemaking is to make the Agency's limits consistent across sectors. In a future PEL update rulemaking, OSHA will evaluate the evidence for acrylic acid to determine whether a reduction in the PEL is warranted.

At the present time, however, OSHA preliminarily concludes that an 8-hour TWA PEL of 10 ppm and a skin notation are necessary to protect workers in construction, maritime, and agriculture from the significant risk of nasal and eye irritation, both material health impairments that are potentially associated with exposure to acrylic acid. The Agency believes that this limit will substantially reduce this risk and prevent recurrences of the burns and irritation previously associated with occupational exposures to acrylic acid. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ALUMINUM (ALKYLS)
CAS: 7429–90–5
Chemical Formula: Al
H.S. No. 1015

ALUMINUM (SOLUBLE SALTS)
CAS: 7429–90–5
Chemical Formula: Al
H.S. No. 1018

OSHA has no permissible exposure limits in construction, maritime, or agriculture for either the soluble salts of aluminum or the aluminum alkyls. The ACGIH has an 8-hour TLV®–TWA limit of 2 mg/m³ for aluminum (soluble salts) and of 2 mg/m³ for the aluminum alkyls. There is no NIOSH REL, but NIOSH concurs (Ex. 8–47, Table N1) that the limits being proposed are appropriate. OSHA is proposing a 2 mg/m³ 8-hour TWA PEL for the aluminum alkyls and the soluble salts of aluminum in construction, maritime, and agricultural operations. This is the limit recently established for these substances in general industry.

Aluminum in its elemental form is a white, malleable, ductile metal (ACGIH 1986, p. 22). Aluminum alkyls are organic compounds containing aluminum; the decomposition products of the alkyls (such as aluminum trioxide) are mucous membrane and pulmonary irritants. The soluble salts (e.g., aluminum sulfate, aluminum trichloride) form hydrolyzed acid in contact with moisture.

OSHA's limits for the aluminum soluble salts were set for general industry workplaces on the basis of the amount of hydrolyzed acid, such as hydrochloric acid or sulphuric acid, in their acid compounds. For example, three mols of hydrogen chloride (HCl) hydrolize from one mol of aluminum chloride; since HCl has a PEL of 5 ppm, a PEL of 2 mg/m³ for aluminum chloride (which is a soluble salt of aluminum) would provide the same degree of protection from irritation as that provided by OSHA's limit for HCl. The acute toxicity of aluminum chloride is generally representative of the toxicity of all of the soluble salts of aluminum. For the aluminum alkyls, toxicity data are sparse. However, all of the nonhalogenated alkyls decompose into aluminum oxide fume, and the halogenated alkyls are even more irritating because of acid hydrolysis.

OSHA is proposing an 8-hour TWA limit of 2 mg/m³, for both the soluble salts of aluminum and the aluminum alkyls, in construction, maritime, and agriculture. The Agency preliminarily concludes that these limits will protect workers in these sectors against the significant risk of irritation and skin burns, which constitute material health impairments that are associated with exposure to these substances. In addition, promulgation of these limits will make OSHA's PEL for these substances consistent across all regulated sectors.

sec-AMYL ACETATE
CAS: 628–63–7; Chemical Formula: $CH_3COOCH(CH_3)C_3H_7$
H.S. No. 2006

In general industry, construction, and maritime, OSHA's permissible exposure limit for sec-amyl acetate is 125 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV® of 125 ppm (665 mg/m³) as an 8-hour TWA for this substance; NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 125 ppm for sec-amyl acetate in agriculture. This is the limit recently established for this substance in general industry.

sec-Amyl acetate is a colorless, volatile liquid with a mild, non-residual, fruity odor. This substance is used as a solvent for nitrocellulose and ethyl cellulose cements, coated papers, lacquers, leather finishes, nail enamels, plastic wood, textile sizing, printing compounds, washable wallpaper, and chlorinated rubber. sec-Amyl acetate also finds use in metallic paints, perfumes, and pearlescent coatings for artificial pearls (ACGIH 1986, p. 29; Hawley's 1987, p. 75; HSDB 1989).

sec-Amyl acetate is an irritant of the eyes, skin, and respiratory tract; at high concentrations, it causes narcosis. The lowest lethal concentration in guinea pigs is 10,000 ppm for 5 hours (RTECS 1990). Much of the toxicological literature does not distinguish between the isomers of amyl acetate; however, all of the industrially important amyl acetate isomers have similar toxicological effects (AIHA 1978; Pagnotto 1964). Guinea pigs exposed to a 10,000-ppm concentration of sec-amyl acetate for 5 hours showed signs of eye and nose irritation, became narcotized, and died (Clayton and Clayton 1981, p. 2274). When the concentration was lowered to 5000 ppm for 13 hours, guinea pigs developed the same signs and symptoms but subsequently recovered (Clayton and Clayton 1981, p. 2274).

Humans exposed to a 5000- to 10,000-ppm concentration of sec-amyl acetate for short periods of time (not further specified) experienced irritation of the eyes and nasal passages (van Oettingen, 1960); however, exposure to a 1000-ppm concentration for 1 hour caused toxic effects described as "serious" (Amor 1950). A 200-ppm concentration of sec-amyl acetate is reported to have caused eye, nose, and upper respiratory tract irritation in one individual exposed for an unspecified amount of time (RTECS 1990).

Based on this evidence in humans and animals, and by analogy to effects caused by exposure to n-amyl acetate, OSHA is proposing to establish an 8-hour TWA PEL for sec-amyl acetate of 125 ppm in agriculture; adoption of this limit would establish the same PEL for workplaces in all OSHA-regulated industry sectors. The Agency preliminarily concludes that occupational exposure to sec-amyl acetate causes irritation of the eyes and respiratory tract, and, at high concentrations, narcosis. Accordingly, OSHA believes that, in the absence of a permissible exposure limit, workers in agriculture are potentially at significant risk of these exposure-related effects and that the proposed PEL will substantially reduce these risks.

BORON TRIBROMIDE
CAS: 10294–33–4; Chemical Formula: BBr_3
H.S. No. 1040

In construction and maritime, OSHA has a limit of 1 ppm as an 8-hour TWA for boron tribromide. There is no limit in agriculture. The ACGIH has a 1-ppm TLV®-ceiling limit for boron tribromide. NIOSH has no REL for this substance. The proposed PEL in construction, maritime, and agriculture, with which NIOSH concurs (Ex. 8–47, Table N1), is a ceiling of 1 ppm. This is the limit recently established for boron tribromide in general industry.

Boron tribromide is a colorless, fuming liquid used primarily in the manufacture of diborane and high-purity boron (ACGIH 1986, p. 62).

Boron tribromide has a high potential for acute local irritation, and its potential for systemic toxicity is analogous with that of hydrogen bromide (HBr). On decomposition, one molecule of boron tribromide would be expected to produce three molecules of HBr (ACGIH 1986/Ex. 1–3, p. 62). Animals repeatedly exposed to boron tribromide develop pneumonia, and exposure to 100 ppm caused a uniformly high mortality rate in animals of six laboratory species (Stokinger, Spiegel et al. 1953, as cited in ACGIH 1986/Ex. 1–3, p. 63). Rats, rabbits, and mice exposed to 1.5, 3.4, or 12.8 ppm boron trifluoride developed pneumonitis and dental fluorosis, although the evidence of pneumonitis was described as "marginal" at the lowest level tested (Torkelson, Sadek, and Rowe 1961, as cited in ACGIH 1986/Ex. 1–3, p. 63).

Based on this evidence of boron tribromide's severe pulmonary toxicity at exposure levels of 3.4 ppm, OSHA is proposing a ceiling limit of 1 ppm for this substance in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors from the significant risk of serious pulmonary damage, a material health impairment that is associated with exposure to this substance at levels above the proposed PEL. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

BROMINE PENTAFLUORIDE

CAS: 7789–30–2; Chemical Formula: BrF_5

H.S. No. 1043

In construction and maritime, OSHA's limit for bromine pentafluoride is 0.1 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.1 ppm for this substance. NIOSH has no REL, but concurs (Ex. 8–47, Table N1) with the proposed limit. OSHA is proposing an 8-hour TWA of 0.1 ppm for this substance in agriculture. This is the limit recently established for this substance in general industry.

Bromine pentafluoride is a highly reactive pale yellow liquid at temperatures below 40.3 °C; above this temperature, it is a colorless, pungent, corrosive gas. It is used in organic synthesis and as an oxidizer in liquid rocket propellants (Hawley's 1987, p. 170).

Bromine pentafluoride has been shown to be acutely toxic in animals. Animals exposed to bromine pentafluoride vapor at 500 ppm exhibited immediate symptoms of gasping, swollen eyelids, clouded corneas, tearing, salivation, and acute distress; these symptoms appeared after exposure for a period as short as three minutes. Exposures to 50 ppm were fatal after 30 minutes, and chronic exposures above 3 ppm resulted in severe nephrosis (in some animals), as well as marked hepatosis and severe respiratory involvement (The Matheson Co., Inc., as cited in ACGIH 1986/Ex. 1–3, p. 66). Bromine pentafluoride is toxicologically more active than free, elemental fluorine, and its toxicity appears to be closely related to that of chlorine trifluoride (Horn and Weir 1955/Ex. 1–592; Horn and Weir 1956, as cited in ACGIH 1986/Ex. 1–3, p. 66). Chlorine trifluoride has caused severe toxicity and some fatalities in dogs and rats exposed over a period of 6 months to an average concentration of 1.17 ppm for 6 hours daily (Horn and Weir 1955/Ex. 1–592).

OSHA is proposing a PEL of 0.1 ppm as an 8-hour TWA for bromine pentafluoride in agriculture to prevent the significant risk of serious systemic injury potentially associated with exposure to this substance at levels above the proposed limit. The Agency preliminarily concludes that this limit will substantially reduce this risk of systemic toxicity, which constitutes a material impairment of health, among agricultural workers. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

sec-BUTYL ACETATE

CAS: 105–46–4; Chemical Formula: $CH_3COOCH(CH_3)CH_2CH_3$

H.S. No. 2022

OSHA's PEL for sec-butyl acetate in general industry, construction, and maritime is 200 ppm as an 8-hour TWA; there is no PEL in agriculture. The ACGIH TLV®–TWA for sec-butyl acetate is 200 ppm as an 8-hour TWA. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 200 ppm for sec-butyl acetate in agriculture. This is the limit recently established for this substance in general industry.

sec-Butyl acetate is a colorless liquid with the fruity odor that is characteristic of acetates (ACGIH 1986, p. 73). sec-Butyl acetate is widely used as a solvent; some materials that contain this substance are thinners, nitrocellulose lacquers, nail enamels, and leather finishes (HSDB 1990).

In both humans and animals, sec-butyl acetate causes irritation of the eyes, mucous membranes, and respiratory tract; at high concentrations, it is believed to cause narcosis (ACGIH 1986, p. 73; Proctor, Hughes, and Fischman 1988, p. 105). By analogy with the effects of exposure to other acetates, overexposure to sec-butyl acetate causes the gradual onset of narcosis; the oral administration of acetates (form and dose not specified) caused a progressive loss of coordination in rabbits (Clayton and Clayton 1981, pp. 2268–2269). The vapor of sec-butyl acetate is reported to be less irritating to humans than the vapor of n-butyl acetate (ACGIH 1986, p. 73). Repeated contact of the skin with this substance causes irritation and dermatitis; possible allergic reactions have also been reported (Genium MSDS 1983, No. 519; New Jersey Fact Sheet 1985, p. 2).

Based on this evidence, OSHA preliminarily concludes that agricultural workers exposed to this substance at the levels permitted by the absence of a limit are at significant risk of experiencing irritation of the eyes, skin, mucous membranes, and upper respiratory tract. At very high concentrations, they may also be at risk of experiencing narcotic effects. The Agency believes that establishing a PEL of 200 ppm as an 8-hour TWA will protect workers in agriculture from these significant risks, which OSHA considers material health impairments. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

tert-BUTYL ACETATE

CAS: 540–88–5; Chemical Formula: $CH_3COOC(CH_3)_3$

H.S. No. 2023

In general industry, construction, and maritime, OSHA's permissible exposure limit for tert-butyl acetate is 200 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 200 ppm for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL in agriculture of 200 ppm for tert-butyl acetate. This is the limit recently established for this substance in general industry.

The principal use for tert-butyl acetate is as a gasoline additive; however, this substance is also used as a solvent (HSDB 1986). tert-Butyl acetate is a colorless liquid with a fruity, acetate-like odor (ACGIH 1986, p. 74).

tert-Butyl acetate is an irritant of the eyes, mucous membranes, and upper respiratory tract in both animals and humans; at high concentrations, this substance is believed to be a depressant

of the central nervous system (Proctor, Hughes, and Fischman 1988, p. 105; ACGIH 1986, p. 74). The vapors of tert-butyl acetate are reported to be less irritating to the throat than those of n-butyl acetate (ACGIH 1986, p. 74). The administration of excessive (dose not further specified) quantities of acetates initially causes eye, throat, and nose irritation, followed by the gradual onset of narcosis; these effects reverse when exposure ceases (Clayton and Clayton 1981, p. 2268). Human subjects exposed for 2 to 3 hours to butyl acetate concentrations (isomer not specified) of 400 to 600 ppm did not experience symptoms of narcosis, although eye irritation was experienced at a 200- to 300-ppm concentration (Clayton and Clayton 1981, p. 2271).

Based on this evidence, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing the eye, nose, and throat irritation associated with exposure to tert-butyl acetate and that they may also be at significant risk of experiencing central nervous system depression. The Agency believes that establishing an 8-hour TWA PEL of 200 ppm will substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

BUTYL ACRYLATE

CAS: 141–32–2; Chemical Formula: $H_2C{=}CHCO_2(CH_2)_3CH_3$

H.S. No. 1048

In construction, maritime, and agriculture, OSHA has no limit for butyl acrylate. The ACGIH's TLV®–TWA is 10-ppm. NIOSH has no REL. The proposed PEL, with which NIOSH concurs (Ex. 8–47, Table N1), is 10 ppm as an 8-hour TWA; this limit would apply in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Butyl acrylate is a colorless, flammable liquid. Butyl acrylate is used in the manufacture of polymers and resins and is also used in paint formulations (ACGIH 1986, p. 75).

Butyl acrylate is a skin and eye irritant and is toxic to animals. The LC_{50} for a 4-hour exposure is 1000 ppm (Carpenter, Weil, and Smith 1974/Ex. 1–304). In rabbits, the dermal LD_{50} for butyl acrylate is approximately 1800 mg/kg, compared with 1235 mg/kg for methyl acrylate (Smyth, Carpenter, and Weil 1951/Ex. 1–439). Butyl acrylate has also been found to be mildly irritating to the skin and to produce corneal necrosis in the unwashed eyes of rabbits (Holland 1974, as cited in ACGIH 1986/Ex. 1–3, p. 75).

OSHA is proposing an 8-hour TWA PEL of 10 ppm for butyl acrylate, based on the similarity of the toxicological response of butyl acrylate to methyl acrylate, for which OSHA also has a 10-ppm TWA limit. The Agency preliminarily concludes that this limit is necessary to reduce the significant risk of skin irritation and corneal necrosis, which constitute material health impairments that are associated with exposure to butyl acrylate. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

o-sec-BUTYLPHENOL

CAS No. 89–72–5; Chemical Formula: $C_2H_5(CH_3)CHC_6H_4OH$

H.S. No. 1055

In construction, maritime, and agriculture, OSHA has no limit for o-sec-butylphenol. The ACGIH has a 5-ppm 8-hour TLV®–TWA, with a skin notation. NIOSH has no REL, but concurs (Ex. 8–47, Table N1) with the proposed limit. The proposed PEL for o-sec-butylphenol in the construction, maritime, and agriculture industries is an 8-hour TWA of 5 ppm, with a skin notation. This is the limit recently established for this substance in general industry.

o-sec-Butylphenol is a colorless liquid. It is used as a chemical intermediate for preparing resins, plasticizers, surface active agents, and other products (ACGIH 1986, p. 84).

Animal studies indicate that contact with o-sec-butylphenol causes irritation of the skin, eyes, and respiratory tract and may result in skin burns. A Dow Chemical Company study (1977i, as cited in ACGIH 1986/Ex. 1–3, p. 84) showed that the oral and skin absorption LD_{50}s for guinea pigs ranged between 0.6 and 2.4g/kg. Prolonged contact of o-sec-butylphenol with the skin of these animals resulted in burns, whereas direct application to the eyes did not cause corneal injury. The oral LD_{50} for rats is 2700 mg/kg (RTECS 1990), and rats exposed to saturated air levels of this chemical survived for seven hours (Dow Chemical Company 1977i, as cited in ACGIH 1986/Ex. 1–3, p. 84). The intravenous LD_{50} for mice is 6 mg/kg (RTECS 1990). Acute workplace exposures to o-sec-butylphenol have resulted in mild respiratory irritation and skin burns (ACGIH 1986/Ex. 1–3, p. 84).

OSHA is proposing an 8-hour TWA limit of 5 ppm for o-sec-butylphenol, with a skin notation, in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that this limit is necessary to protect workers in construction, maritime, and agriculture from the significant risks of eye and respiratory tract irritation and skin burns potentially associated with exposure to this substance. OSHA believes that eye and mucous membrane irritation and skin burns constitute material health impairments within the meaning of the Act. Promulgation of this limit will also make OSHA's PEL for this substance consistent across all regulated sectors.

CALCIUM HYDROXIDE

CAS: 1305–62–0; Chemical Formula: $Ca(OH)_2$

H.S. No. 1059

In construction, maritime, and agriculture, OSHA has no limit for calcium hydroxide; the ACGIH has a TLV®–TWA of 5 mg/m³. There is no NIOSH REL, but NIOSH concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing a PEL of 5 mg/m³ as an 8-hour TWA for calcium hydroxide in the construction, maritime, and agriculture industries. This is the limit recently established for this substance in general industry.

Calcium hydroxide is a soft, white, odorless, crystalline powder with an alkaline, bitter taste. It is used in building and paving materials, in lubricants, in drilling fluids, in pesticides, and in the manufacture of paper. There are many other industrial uses for this substance (ACGIH 1986, p. 91).

Calcium hydroxide is a moderate to severe caustic and irritant when it comes into contact with the skin, eyes, or mucous membranes of the upper respiratory tract (ACGIH 1986/ Ex. 1–3, p. 92; Sax and Lewis 1989, p. 682). The oral LD_{50} in rats is reported to be 7.34 g/kg (Smyth, Carpenter, Weil et al. 1969/ Ex. 1–442). Industrial experience with this substance has not shown a high incidence of adverse health effects, although Sax (1984) reports that it is known to cause dermatitis (p. 621). Calcium hydroxide is also mutagenic (Sax 1984).

In the prior rulemaking, one commenter argued that calcium hydroxide should be regulated as a nuisance dust, i.e., should have a PEL of 10 mg/m³. In response, OSHA stated that the Agency does not agree that calcium hydroxide is a biologically inert substance. Instead, OSHA agrees with Sax (1984), who reports that, "in the form of dust, it [calcium hydroxide] is considered to be an important industrial hazard." OSHA believes that a PEL of 5 mg/m³, half that of the inert particulate limit, is appropriate for this well-known eye, skin, and upper respiratory tract

irritant. OSHA is proposing an 8-hour TWA limit for calcium hydroxide of 5 mg/m³ to protect workers in construction, maritime, and agriculture from the significant risk of skin, eye, and mucous membrane irritation, which OSHA considers material impairments of health. The Agency preliminarily concludes that this limit will reduce these risks substantially. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CALCIUM OXIDE
CAS: 1305–78–8; Chemical Formula: CaO
H.S. No. 1060

In general industry, construction, and maritime, OSHA's 8-hour TWA permissible exposure limit for calcium oxide is 5 mg/m³. There is no limit in agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. The ACGIH TLV® for calcium oxide, which was set on the basis of analogy with sodium hydroxide, a widely recognized sensory irritant, is 2 mg/m³ as an 8-hour TWA. OSHA is proposing a 5-mg/m³ 8-hour TWA PEL for calcium oxide in agriculture. This action will make OSHA's PEL for calcium oxide consistent across all regulated sectors.

Calcium oxide (lime) is a white or grayish-white powder. It is produced when limestone is calcined to drive off carbon dioxide. Calcium oxide is used as a refractory material; as a flux in steelmaking; as a binding agent in building, pulp and paper manufacture, sugar refining, and leather tanning; as the raw material for chlorinated lime bleaching powder, and as a soil treatment in agriculture (Parmeggiani 1983).

The amount of information that has been published specifically about calcium oxide's toxicological effects in animals or humans is limited. In direct contact with tissues, calcium oxide can result in burns and severe irritation because of its high reactivity and alkalinity. The major complaints of workers exposed to lime consist of irritation of the skin and eyes, although inflammation of the respiratory passages, ulceration and perforation of the nasal septum, and even pneumonia have been attributed to inhalation of the dust (ACGIH 1986/Ex. 1–3, p. 92). The Pennsylvania Department of Health reported that strong nasal irritation occurred as a consequence of exposure to a mixture of calcium-oxide-containing dusts at a concentration of approximately 25 mg/m³, but that exposure to concentrations of 9 to 10 mg/m³ produced no observable irritation (Wands 1981a, in Clayton and Clayton 1981, p. 3054). By comparison, exposure to airborne sodium hydroxide at a concentration of between 0.005 and 0.7 mg/m³ produced burning/redness of the nose, throat, or eyes in workers engaged in cleaning operations (Hervin and Cohen 1973/Ex. 1–945, as cited in NIOSH 1976k/Ex. 1–965). Thus, the demonstrated effect level for sensory irritation caused by exposure to sodium hydroxide is below 1 mg/m³, while that for calcium oxide is above 9 mg/m³.

Based on evidence that exposure to calcium oxide at levels above 9 mg/m³ may cause tearing of the eyes and mucous membrane irritation, OSHA preliminarily concludes that the proposed 8-hour TWA limit of 5 mg/m³ is appropriate for agricultural workplaces. The Agency believes that this limit will protect exposed workers in agriculture from the significant risk of sensory irritation known to occur at concentrations of 9 to 10 mg/m³. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CARBONYL FLUORIDE
CAS: 353–50–4; Chemical Formula: COF_2
H.S. No. 1074

In construction, maritime, and agriculture, OSHA has no limit for carbonyl fluoride. The ACGIH has an 8-hour TLV®–TWA of 2 ppm and a 15-minute TLV®–STEL of 5 ppm for this substance. There is no NIOSH REL. The PELs OSHA is proposing for construction, maritime, and agriculture are an 8-hour TWA of 2 ppm and a 15-minute STEL of 5 ppm. NIOSH concurs (Ex. 8–47, Table N1) with these limits, which are identical to those recently established for this substance in general industry.

Carbonyl fluoride is a colorless gas. This substance is used in organic synthesis (Hawley's 1987, p. 223).

The 1-hour LC_{50} for carbonyl fluoride in rats is 360 ppm, and the 4-hour LC_{50} for the same species is 90 ppm (ACGIH 1986/Ex. 1–3, p. 111). Carbonyl fluoride hydrolyzes instantly on contact with moisture. The ACGIH (1986/Ex. 1–3, p. 14) reports that carbonyl fluoride is "about as toxic as hydrogen fluoride as a respiratory irritant gas."

Repeated exposure of animals to carbonyl fluoride is known to have metabolic effects; this substance inhibits the fluoride-sensitive enzyme succinic dehydrogenase via hydrolysis of carbonyl fluoride to hydrogen fluoride (Scheel, McMillan, and Phipps 1968/Ex. 1–364). Carbonyl fluoride is described as a "potent" irritant (Sax and Lewis 1989, p. 717).

OSHA is proposing an 8-hour TWA limit of 2 ppm and a 15-minute STEL of 5 ppm for carbonyl fluoride in construction, maritime, and agriculture. The Agency preliminarily concludes that both a TWA and a STEL are necessary to provide workers in these sectors with protection against the significant risks of marked irritation and metabolic effects. OSHA considers these effects material health impairments. In addition, promulgation of these limits will make OSHA's PELs for carbonyl fluoride consistent across all regulated sectors.

CATECHOL (PYROCATECHOL)
CAS: 120–80–9; Chemical Formula: $C_6H_4(OH)_2$
H.S. No. 1075

In construction, maritime, and agriculture, OSHA has no limit for catechol. The ACGIH has a TLV®–TWA of 5 ppm. There is no NIOSH REL but NIOSH concurs (Ex. 8–47, Table N1) with the limit being proposed. The proposed PEL for this substance in construction, maritime, and agriculture is 5 ppm as an 8-hour TWA. In addition, the Agency is proposing to add a skin notation to the PEL, in accordance with its policy on skin designations, discussed in Section IV.C.16.

Catechol is a colorless crystalline solid that sublimes readily and thus occurs in the vapor state at room temperature. This substance finds use as an antiseptic and in organic synthesis. It is also used in electroplating and photography, and as an ingredient in dyestuffs, specialty inks, antioxidants, and light stabilizers (Hawley's 1987, p. 984).

Catechol is approximately 1.1 to 2.2 times more toxic than phenol, depending on the route of exposure (Industrial Bio-Test Laboratories 1974, as cited in ACGIH 1986/Ex. 1–3, p. 112). The oral LD_{50} in rats is 300 mg/kg, or approximately half that of phenol. Percutaneous toxicity for catechol in rabbits is 800 mg/kg; catechol's dermal LD_{50} in rabbits of 0.8 g/kg places this substance in the category of "toxic" by the percutaneous route of administration, as discussed in Section VI.C.16. In addition, the Agency is concerned by reports of catechol-induced central nervous system effects (i.e., convulsions) in humans; this effect is said to occur as a result of skin absorption and to be "more marked" than the CNS effects produced by phenol (Deichmann and Keplinger 1981, in Clayton and Clayton 1981, p. 2586). OSHA is therefore proposing a skin notation for catechol to protect workers in these sectors from the serious CNS effects that may potentially occur from percutaneous absorption of this substance. Eye and nose irritation, as

well as muscular spasms and tremor, have been observed in rats at a concentration of 2800 mg/m³ catechol, indicating that the acute respiratory toxicity of catechol is approximately one-third that of phenol (Industrial Bio-Test Laboratories 1974, as cited in ACGIH 1986/Ex. 1–3, p. 112). In mice, catechol is easily absorbed through the skin and gastrointestinal tract (Forsyth and Quesnel 1957/Ex. 1–978). Additional data document a variety of dermal, respiratory, and systemic toxicities that are closely analogous to those of phenol in their metabolic actions (Harold, Nierenstein, and Roaf 1910/Ex. 1–1111; Dietering 1938/Ex. 1–1019; Cushny et al. 1940, as cited in ACGIH 1986/Ex. 1–3, p. 112). Exposure to catechol causes an increase in blood pressure, and, at high doses, kidney damage, eczematous dermatitis, and systemic illness (Harold, Nierenstein, and Roaf 1910/Ex. 1–1111; Dietering 1938/Ex. 1–1019; Cushny et al. 1940, as cited in ACGIH 1986/Ex. 1–3, p. 112).

Based on this evidence in humans and animals, OSHA is proposing an 8-hour TWA permissible exposure limit of 5 ppm for catechol, with a skin notation, in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant risks of dermal and upper respiratory tract irritation, convulsions, and other central nervous system effects, all of which constitute material impairments of health that are potentially associated with exposure to catechol. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

1-CHLORO-1-NITROPROPANE
CAS: 600–25–9; Chemical Formula: $CH_3CH_2CHClNO_2$
H.S. No. 1081

In construction and maritime, OSHA's time-weighted average permissible exposure limit for 1-chloro-1-nitropropane is 20 ppm. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 2 ppm for this substance (ACGIH 1986/Ex. 1–3). There is no NIOSH REL. For construction, maritime, and agriculture, OSHA is proposing a PEL of 2 ppm as an 8-hour TWA, and NIOSH concurs (Ex. 8–47, Table N1) with this limit, which was recently established for this substance in general industry.

1-Chloro-1-nitropropane is a flammable liquid (ACGIH 1986, p. 132.2). This substance finds use as a fungicide (Hawley's 1987, p. 676). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

1-Chloro-1-nitropropane is the most acutely toxic of the fungicides known as the chloronitropropanes. In an inhalation experiment, two rabbits were exposed for 6 hours to a concentration of 393 ppm, after which one rabbit died; at an average concentration of 2574 ppm, both rabbits died. Guinea pigs tested under the same conditions survived these exposures. The oral LD_{50} in rabbits is between 50 and 100 mg/kg (Machle, Scott, Treon et al. 1945/Ex. 1–349). Other members of this family of fungicides cause a lesser degree of skin and lung irritation but have higher ingestion toxicities (Patty 1963i, as cited in ACGIH 1986/Ex. 1–3, p. 132). Exposure to high concentrations (not further specified) of 1-chloro-1-nitropropane can cause heart muscle, liver, and kidney damage (Patty 1963i, as cited in ACGIH 1986/Ex. 1–3, p. 132).

OSHA is proposing an 8-hour TWA PEL of 2 ppm for 1-chloro-1-nitropropane in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect employees in these sectors from the significant risk of skin and upper respiratory tract irritation and of systemic toxicity, which constitute material health impairments within the meaning of the Act. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

COBALT CARBONYL (as Co)
CAS: 10210–68–1; Chemical Formula: $Co_2(CO)_4$
H.S. No. 1098

In construction, maritime, and agriculture, OSHA has no limit for cobalt carbonyl. The ACGIH has a TLV®–TWA of 0.1 mg/m³ (measured as cobalt) for this substance. There is no NIOSH REL. In construction, maritime, and agriculture, OSHA is proposing a PEL of 0.1 mg/m³ as an 8-hour TWA (measured as cobalt), and NIOSH concurs (Ex. 8–47, Table N1) with this limit. This is the limit recently established for this substance in general industry.

Cobalt carbonyl takes the form of orange or brown crystals (Weast 1984, p. B–89). It is formed by vacuum sublimation.

Sax and Lewis (1989, p. 938) report that the LC_{50} in mice is 27 mg/m³ for 2 hours. The oral LD_{50} in mice is 377.7 mg/kg; in rats, it is 753.8 mg/kg (Spiridonova and Shabalina 1973/Ex. 1–1098). Exposure to cobalt carbonyl causes chemical pneumonitis (Stokinger 1981e). The hazards of exposure to the metal carbonyls range from relatively low (for iron pentacarbonyl) to extremely serious (for nickel carbonyl) (Stokinger 1981e, in Clayton and Clayton 1981, pp. 1797–1806); the greater the toxicity of the metal and the more stable and volatile the carbonyl, the more hazardous the compound. Exposure to any of the metal carbonyls causes the same symptoms of nausea, dizziness, headache, substernal pain, coughing, and dyspnea (Stokinger 1981e). Evidence concerning any chronic effects of long-term exposure is lacking (ACGIH 1986/Ex. 1–3, p. 145).

OSHA is proposing an 8-hour TWA PEL of 0.1 mg/m³ for cobalt carbonyl in construction, maritime, and agriculture to protect against the significant risk of headache, nausea, and pulmonary effects, all of which are material impairments of health that are associated with occupational exposure to this substance. The Agency preliminarily concludes that this limit is necessary to substantially reduce these significant risks among workers in these sectors. In addition, promulgation of this limit will make OSHA's PEL for cobalt carbonyl consistent across all regulated sectors.

COBALT HYDROCARBONYL (as Co)
CAS: 16842–03–8; Chemical Formula: $HCo(CO)_4$
H.S. No. 1099

In construction, maritime, and agriculture, OSHA has no limit for cobalt hydrocarbonyl. The ACGIH has a TLV®–TWA of 0.1 mg/m³ (measured as cobalt) for this substance. There is no NIOSH REL. OSHA is proposing an 8-hour TWA PEL of 0.1 mg/m³ for cobalt hydrocarbonyl (measured as cobalt) in construction, maritime, and agriculture; NIOSH concurs (Ex. 8–47, Table N1) with this limit, which was recently established for cobalt hydrocarbonyl in general industry.

Cobalt hydrocarbonyl is a gas that behaves like a strong acid and decomposes rapidly in air (Clayton and Clayton 1981, pp. 1793–1796). Cobalt hydrocarbonyl is the catalyst for the OXO process that converts olefins to oxidized products (Clayton and Clayto 1981, p. 1795).

Cobalt hydrocarbonyl is approximately half as toxic as nickel carbonyl in terms of acute effects; in animals, it produces clinical signs and symptoms very similar to those produced by nickel carbonyl (OSHA PEL of 0.007 mg/m³) and iron pentacarbonyl (OSHA PEL of 0.8 mg/m³) (ACGIH 1986/Ex. 1–3, p. 145). These include headache, dizziness, and, after a delay in onset, liver, brain, and lung damage. The 30-minute LC_{50} in rats is

46.2 mg/m³ (RTECS 1990). There is no evidence of chronic toxicity or of carcinogenicity.

OSHA is proposing an 8-hour TWA limit of 0.1 mg/m³ for cobalt hydrocarbonyl in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect exposed employees in these sectors from the significant risk of pulmonary, brain, and liver damage, as well as acute effects such as headaches and dizziness, all of which constitute material health impairments. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

CRESOL (all isomers)

CAS: 1319–77–3; Chemical Formula: $CH_3C_6H_4OH$

H.S. No. 2042

In general industry, construction, and maritime, OSHA's permissible exposure limit for all cresol isomers is 5 ppm (22 mg/m³) as an 8-hour TWA; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 5 ppm, and a skin notation, for cresol. NIOSH has a REL for this substance of 10 mg/m³ (2.3 ppm) as a 10-hour TWA. OSHA is retaining its PEL and skin notation for cresol in construction and maritime and is proposing to extend the PEL and notation to agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Cresol is a colorless, yellowish, or pinkish liquid with a phenol-like odor. The commercially available substance is a mixture of the ortho-, meta-, and para-isomers, which derive from coal tar or petroleum. Cresol is used in the manufacture of herbicides and synthetic resins; as a disinfectant and fumigant; in photographic developers and explosives; as a textile scouring agent, surfactant, organic intermediate, and metal degreaser; and in synthetic food flavoring (ACGIH 1986, p. 148; Hawley's 1987, p. 320; HSDB 1991). The cresols have wide application in the agricultural industries (Clayton and Clayton 1981, p. 2597). When used in pesticidal applications and in accordance with directions on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Cresol causes eye, nose, and skin irritation, central nervous system effects, cardiovascular effects, and liver and kidney injury in humans and animals. In rats, the oral LD_{50} for ortho-cresol is 121 mg/kg; in mice, the oral LD_{50} is 344 mg/kg (RTECS 1990). The dermal LD_{50} in rabbits is 890 mg/kg for the ortho-isomer (RTECS 1990 "o-Cresol"). For meta-cresol, the oral LD_{50} values are 242 mg/kg and 828 mg/kg for rats and mice, respectively, and the dermal LD_{50} in rabbits is 517 mg (RTECS 1990 "m-Cresol"). The oral LD_{50} values for para-cresol in rats and mice are 207 mg/kg and 344 mg/kg, respectively, and the dermal LD_{50} in rabbits is 301 mg/kg (RTECS 1990 "p-Cresol"). Instilled into the eyes of rabbits, doses of cresol ranging from 103 to 105 mg in the eyes or of 517 to 524 mg placed in contact with the skin of rabbits for 24 hours caused severe irritation (RTECS 1990). Rats dermally exposed to cresol at a dose of 1.0 to 1.7 ml/kg for 1 to 2 hours developed skin discoloration and died (Campbell 1941, in Soap Sanit. Chem. 17:103–111, in NIOSH Criteria Document 1978, p. 59). Exposure to saturated airborne concentrations of cresol for 1 hour per day for 10 days caused eye and nose irritation and some deaths in mice (Campbell 1941, as above). A single oral dose of 1300 to 2700 mg/kg of p-cresol caused twitching, comas, and death in rats (Deichmann, Witherup 1944, in J. Pharmacol. Exp. Ther. 80:233–240). A group of rats exposed to concentrations of 0.05 or 0.0052 mg/m³ of tricresol (a mixture of the three isomers) for 3 months showed decreased weight gain, increased central nervous system excitability and oxygen consumption, histologic changes in the lungs and liver, and decreases in the gamma-globulin content of the serum in the high-dose group; no significant changes were seen in the low-dose group (Kurlyandskiy et al. 1975, in Gig. Sanit. 5:85–87). Guinea pigs inhaling 9.0 mg/m³ of o-cresol over a 4-month period showed changes in their ECGs, and rats exposed to the same concentration on this regimen developed both hematological and central nervous system changes (NIOSH Criteria Document 1978, p. 60).

In humans, acute exposure to cresol by any route can cause muscular weakness, gastroenteric disturbances, severe depression, collapse, lung edema, and injury to the pancreas and spleen. Fatal poisoning may occur by skin absorption if a large body area is involved (Windholz 1983, p. 369). Oral exposure to 8 g or more of cresol produces rapid circulatory collapse and is followed by death (Windholz 1983, p. 369). Eight of 10 workers exposed to 1.4 ppm o-cresol experienced upper respiratory tract irritation (NIOSH Criteria Document 1978, p. 57). Cresol is a severe skin irritant and a frequent cause of skin discoloration and dermatitis among exposed workers (Sittig 1985, pp. 283–284). Chronic effects from cresol exposure include skin problems, central nervous system changes, and possible liver and kidney damage (Rom 1983, p. 528). Seven workers exposed to cresol for 1.5 to 3 years at unspecified concentrations had headaches and frequent nausea and vomiting. Four of the workers experienced elevated blood pressure, impaired kidney function, a blood calcium imbalance, and marked tremors (NIOSH Criteria Document 1978, p. 117).

Based on this evidence in humans and animals, OSHA preliminarily concludes that cresol causes primary irritation, central nervous system effects, cardiovascular effects, and liver and kidney injury. OSHA believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed limit for cresol of 5 ppm as an 8-hour TWA, with a skin notation, is necessary to substantially reduce the risks of material health impairment among agricultural workers. In addition, promulgation of this limit will make the PEL for cresols consistent across all OSHA-regulated sectors.

CROTONALDEHYDE

CAS: 4170–30–3; 123–73–9 (trans-isomer); Chemical Formula: $CH_3CH{=}CHCHO$

H.S. No. 2043

In general industry, construction, and maritime, OSHA's permissible exposure limit for crotonaldehyde is 2 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 2 ppm for this substance; NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 2 ppm for crotonaldehyde in agriculture. This is the limit recently established for this substance in general industry.

Crotonaldehyde is a colorless, flammable liquid with a strong, pungent odor. It turns a pale yellow color in contact with air or light. The commercially available product consists more than 95 percent of the trans-isomer. Crotonaldehyde is primarily used as an intermediate in the manufacture of n-butanol; this substance is also used in pesticide compounds, in chemotherapeutic agents, as a warning agent in fuel gases, as a solvent, and to locate breaks and leaks in pipes (HSDB 1988; ACGIH 1986, p. 149; AIHA 1988). When used in pesticidal applications and in accordance with directions on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Crotonaldehyde is an irritant of the eyes, mucous membranes, respiratory system, and skin in both animals and humans. Central nervous system effects have been observed in exposed animals, and sensitization may occur in humans. The oral LD_{50} in rats is 206 mg/kg, the 2-hour LC_{50} in rats is 200 mg/m³, and the dermal LD_{50} in rabbits is 380 mg/kg (RTECS 1990; AIHA 1988). Death occurred in rats exposed to a 1650-ppm concentration of crotonaldehyde for 10 minutes; acutely poisoned animals developed respiratory distress, excitation, and terminal convulsions, and autopsy revealed bronchiolar damage (Rinehart 1967). A single exposure to a 10-ppm concentration of crotonaldehyde for 20 minutes produced a measurable change in pulmonary performance in rats (Rinehart 1967). In contact with the skin of rabbits, crotonaldehyde caused mild irritation (RTECS 1990). The damage caused to the eyes of rabbits by liquid crotonaldehyde is severe (Grant 1986, p. 284). The mortality observed among rats in a 13-week gavage study was attributed to the corrosive action of crotonaldehyde; stomach lesions included hyperplasia of the forestomach epithelium, forestomach hyperkeratosis, ulcers, moderate necrosis, and acute inflammation (Wolfe, Rodwin, French et al. 1987).

Volunteers exposed for 15 minutes to a 4.1-ppm concentration of crotonaldehyde reported irritation of the nose and upper respiratory tract, and lacrimation was experienced after 30 seconds of exposure to this concentration (Sim and Pattle 1957). In eight cases of corneal injury in workers handling crotonaldehyde, complete healing occurred within 48 hours of the incident (Grant 1986, p. 284). A laboratory worker who handled small amounts of crotonaldehyde reportedly developed a sensitization reaction to this substance (ACGIH 1986, p. 149), and patch testing of a textile worker confirmed sensitization to crotonaldehyde (Shmunes and Kempton 1980).

Based on this evidence, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing the eye, mucous membrane, respiratory system, and skin irritation associated with exposure to crotonaldehyde. The Agency believes that establishing an 8-hour TWA PEL of 2 ppm is necessary to substantially reduce this risk among agricultural workers. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DEMETON (Systox)

CAS: 8065-48-3; Chemical Formula: $C_8H_{19}O_3PS_2$

H.S. No. 2049

In general industry, construction, and maritime, OSHA's permissible exposure limit for demeton (also called Systox) is 0.1 mg/m³ as an 8-hour TWA; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 0.1 mg/m³, with a skin notation, for this substance; this limit was established by analogy with parathion. NIOSH has no REL for demeton but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing a PEL of 0.1 mg/m³ as an 8-hour TWA, and a skin notation, for demeton in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Demeton is a light brown to pale yellow oil with a sulfur-like odor; it is a mixture of two isomers. Demeton is a systemic insecticide and acaricide used against sap-feeding insects and mites (ACGIH 1986, p. 170; Hayes 1982, p. 389). When used in pesticidal applications and in accordance with directions on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Demeton causes cholinesterase inhibition, including respiratory and ocular effects, cyanosis, and central nervous system effects. The oral LD_{50} values in rats and mice are 1700 μg/kg and 7850 μg/kg, respectively (RTECS 1990). The dermal LD_{50} in rabbits is 24 mg/kg (RTECS 1990). Rats fed 50 ppm demeton in the diet became sick but recovered after 3 to 4 weeks despite continued feeding of demeton (Barnes and Denz 1954, in Hayes 1982, p. 389). A dietary level of 5 ppm (0.149 mg/kg/day) caused erythrocyte cholinesterase inhibition in dogs; plasma cholinesterase inhibition was marked at 5 ppm and slight at 2 ppm (Frawley and Fuyat 1957, in Hayes 1982, p. 389). A single exposure to a concentration of 18 mg/m³ demeton was fatal to all of six rats exposed; the deaths occurred from 50 to 90 minutes after the exposure (Deichmann and Rakoczy 1955, in ACGIH 1986, p. 170). Seventeen rats exposed to a 3 mg/m³ concentration of demeton showed no signs of illness after the first exposure; following the second exposure, however, tremors were observed, and the fourth exposure caused death in 10 of the 17 rats (Deichmann and Rakoczy 1955, in ACGIH 1986, p. 170). Doses of 7 to 10 mg/kg administered to pregnant mice during days 7 to 11 of pregnancy produced reproductive, developmental, and embryonic effects in mice (RTECS 1990).

In humans, several cases of demeton intoxication have been reported. The lowest lethal oral dose reported for humans is 171 μg/kg (RTECS 1990). The first signs and symptoms of demeton intoxication are respiratory and ocular effects, which may include a feeling of tightness in the chest, wheezing, laryngeal spasm, excessive salivation, miosis, blurred distant vision, tearing, rhinorrhea, and frontal headache (Proctor, Hughes, and Fischman 1988, p. 176). Ingestion of demeton causes symptoms, such as anorexia, nausea, vomiting, abdominal cramps, and diarrhea, which usually appear within 15 minutes to 2 hours (Proctor, Hughes, and Fischman 1988, p. 176). Absorption of demeton through the skin results in localized sweating and muscular fasciculations within 15 minutes to 4 hours after exposure (Proctor, Hughes, and Fischman 1988, p. 176). Five volunteers given oral doses of 7.125 mg/day (about 0.1 mg/kg/day) for 25 days showed a depression of plasma and erythrocyte cholinesterase activity of 39.8 and 15.9 percent, respectively (Moeller and Rider 1965; Rider et al. 1969, in Hayes 1982, p. 389). A 16-year-old boy exposed to demeton while spraying hops was unconscious for 6 hours; 5 weeks after treatment with atropine he continued to complain about difficulty in breathing, general weakness, and lack of coordination in walking. After 3 months, the boy still had autonomic nervous system disturbances (Klimkova-Deutschova 1959, in Hayes 1982, p. 390). Twelve of 14 workers exposed to demeton in the air during aerial spraying of cotton and wheat had decreased cholinesterase levels but no signs of poisoning; demeton levels in the air breathed by these workers approached a concentration of 1 mg/m³ (Kagan 1956, in Hayes 1982, p. 390). Levels ranging from 4.3 to 6 mg/m³ were reported to have caused a reduction in the serum cholinesterase activity of workers applying demeton to trees, but no other signs or symptoms of intoxication were reported (Kagan et al. 1958, in Hayes 1982, p. 390).

Based on this evidence in humans and animals, OSHA preliminarily concludes that demeton causes cholinesterase inhibition and concomitant poisoning. OSHA preliminarily finds that, in the absence of a limit, workers in agriculture are at significant risk of

experiencing these adverse health effects. The Agency believes that establishing an 8-hour TWA PEL of 0.1 mg/m³, and a skin notation, for demeton in agriculture is necessary to significantly reduce this risk of material health impairment. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

DIAZINON

CAS: 333–41–5; Chemical Formula: $C_{12}H_{21}N_2O_3PS$

H.S. No. 1118

OSHA has no limit for diazinon in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 0.1 mg/m³, with a skin notation. There is no NIOSH REL. OSHA is proposing a PEL for diazinon in construction, maritime, and agriculture of 0.1 mg/m³ as an 8-hour TWA, with a skin notation; NIOSH concurs (Ex. 8–47, Table N1) that this limit is appropriate. This is the limit recently established for this substance in general industry.

Pure diazinon is a colorless liquid, but the technical grade is pale yellow to dark brown in color and has a faint odor. Diazinon is an insecticide that is used against fire ants (Hawley's 1987, p. 364). When used in pesticidal applications and in accordance with directions on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Gaines (1960/Ex. 1–319) reports the acute oral LD_{50} for male and female rats to be 108 and 76 mg/kg, respectively. Other reports set the acute oral LD_{50}s in rats, guinea pigs, and rabbits at 76 to 150, 240 to 320, and 130 mg/kg, respectively (Association of American Pesticide Control Officials, Inc. 1969, as cited in ACGIH 1986/Ex. 1–3, p. 172). The dermal LD_{50} in rabbits is 400 mg/kg (RTECS 1990). Susceptibility to repeated doses is relatively consistent among species, with dogs showing signs of poisoning at 9.3 mg/kg per day and rats showing complete inhibition of red blood cell cholinesterase and marked inhibition of brain cholinesterase at 50 mg/kg/day (Bruce, Howard, and Elsea 1955/Ex. 1–585). Monkeys were poisoned at diazinon doses of 5 mg/kg/day (Woodard, Woodard, and Cronin 1968/Ex. 1–458). Feeding studies in rats have shown no chronic toxicity at 10, 100, or 1000 ppm. For many mammals, diazinon is less toxic than parathion (ACGIH TLV®–TWA of 0.1 mg/m³), although this is not true under some circumstances (ACGIH 1986/Ex. 1–3, p. 172).

In humans, Hayes (1963/Ex. 1–982) reports that two patients were poisoned by a dermal diazinon dosage of about 1.1 mg/kg; however, Gassman (1957/Ex. 1–901) reports no ill effects from an accidental ingestion of 30 mg/kg. One man received a dose of 250 mg/kg and recovered after treatment, which included gastric lavage (Bockel 1967, as cited in ACGIH 1986/Ex. 1–3, p. 172). In tests, Geigy (1966, as cited in ACGIH 1986/Ex. 1–3, p. 172) found that a series of doses of 0.05 mg/kg/day for 28 days produced plasma cholinesterase inhibition, and it has been suggested that the no-effect level for cholinesterase inhibition in humans is 0.02 mg/kg/day. Skin absorption of diazinon occurs readily, and overexposures are associated with weakness, headache, blurred vision, salivation, sweating, nausea, vomiting, diarrhea, abdominal cramps, slurred speech, and moist rales in the lungs (ACGIH 1986/Ex. 1–3, p. 172).

The Agency is proposing an 8-hour TWA PEL of 0.1 mg/m³, with a skin notation, for diazinon in construction, maritime, and agriculture. OSHA preliminarily concludes that these limits will protect exposed workers in these sectors from the significant risk of cholinesterase inhibition, weakness, headache, nausea, vomiting, as well as the other symptoms and signs of diazinon poisoning, which together constitute material health impairments that are associated with exposure to this substance. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

1,1–DICHLORO–1–NITROETHANE

CAS: 594–72–9; Chemical Formula: $CH_3CCl_2NO_2$

H.S. No. 1121

In construction and maritime, OSHA currently has a ceiling limit of 10 ppm for 1,1-dichloro-1-nitroethane. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 2 ppm for this substance. There is no NIOSH REL, but NIOSH concurs (Ex. 8–47, Table N1) with the proposed limit. OSHA is proposing an 8-hour TWA PEL of 2 ppm for 1,1-dichloro-1-nitroethane in the construction, maritime, and agriculture industries. This is the limit recently established for 1,1-dichloro-1-nitroethane in general industry.

1,1–Dichloro-1-nitroethane is a colorless liquid. It is used in organic synthesis and as a fumigant for produce. When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Toxicity data on 1,1-dichloro-1-nitroethane are largely derived from the 1945 studies conducted by Machle and co-workers (Ex. 1–349). These scientists reported that both rabbits and guinea pigs died from inhaling vapors at 100 ppm for 8 hours; at a concentration of 60 ppm, the animals survived a 2-hour exposure. Four-hour inhalation exposures at 34 ppm and 8-hour daily exposures at 25 ppm for a total of 204 hours also did not kill rabbits or guinea pigs. Skin and mucous membrane irritation were not produced at the 25-ppm exposure level. At survival concentrations, the primary targets of toxicity were the lungs, which showed edema, congestion, hemorrhage, and acute bronchitis. At lethal exposures, these investigators observed acute myocardial degeneration with interstitial edema, cloudy swelling of the liver with cellular degeneration, and tubular degeneration and interstitial edema of the kidney, as well as edema of the tufts of the glomeruli and kidney necrosis. The compound was also found to be a severe skin irritant when two applications were applied on two successive days (Machle, Scott, Treon et al. 1945/Ex. 1–349). The ACGIH (1986/Ex. 1–3, p. 186) states that dichloronitroethane is more toxic than the nonchlorinated nitroalkanes.

OSHA is proposing a PEL of 2 ppm TWA for 1,1-dichloro-1-nitroethane in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risk of irritation, lung injury, and liver and kidney damage, all material health impairments that are associated with exposure to this substance. In addition, promulgation of this limit will make OSHA's PEL for 1,1-dichloro-1-nitroethane consistent across all regulated sectors.

p-DICHLOROBENZENE

CAS: 106–46–7; Chemical Formula: $C_6H_4Cl_2$

H.S. No. 1125

In construction and maritime, OSHA has an 8-hour TWA limit of 75 ppm for p-dichlorobenzene. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 75 ppm and a TLV®–STEL of 110 ppm for this substance. NIOSH has no REL for p-dichlorobenzene. In construction and maritime, OSHA is retaining the 75-ppm TWA limit and proposing to add a STEL of 110 ppm for p-dichlorobenzene; the Agency is also proposing to extend these limits to agriculture. These are the limits recently established for p-dichlorobenzene in general industry.

p-Dichlorobenzene is a white, crystalline solid with a camphor-like

odor. It is used as a fumigant to control mildew and molds (ACGIH 1986, p. 187). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In animal studies, an injection of 0.005 gram of p-dichlorobenzene in rats caused slight liver necrosis (Cameron, Thomas, Ashmore et al. 1937/Ex. 1–471). The intraperitoneal LD_{50} for rats is 2562 mg/kg (Zupko and Edwards 1949/Ex. 1–878). The oral LD_{50} in mice is 2950 mg/kg (Domenjoz 1946, as cited in ACGIH 1986/Ex. 1–3, p. 179); in rats, the oral LD_{50} is 2512 mg/kg (Varshavskaya 1970, as cited in ACGIH 1986/Ex. 1–3, p. 179). Rabbits fed a daily dietary dose of 5 grams developed opacity of the lens in 3 weeks (Berliner 1939/Ex. 1–715); this finding was not confirmed, however, in repeated studies (Pike 1944/Ex. 1–658).

Reports of a human inhalation exposure to unspecified levels of p-dichlorobenzene describe swelling of the feet, ankles, and hands after day-long use of a mothproofing agent consisting of this substance (Clayton 1935/Ex. 1–306). Other reports describe cataracts caused by exposure to unspecified concentrations of the vapor of p-dichlorobenzene (Berliner 1939/Ex. 1–715). Petit and Champaix (1948, as cited in ACGIH 1986/Ex. 1–3, p. 179) report the case of a woman who experienced tingling of the hands, vertigo, and loss of weight from working for 18 months with a mixture of 90 parts p-dichlorobenzene and 10 parts hexachloroethane (airborne concentration not specified).

Based on this evidence, OSHA is retaining the 8-hour TWA PEL of 75 ppm TWA in construction and maritime and is proposing to add a STEL of 110 ppm for p-dichlorobenzene in these sectors; the Agency is also proposing to extend these limits to agriculture. OSHA preliminarily concludes that both a TWA and a STEL are necessary to protect workers in these sectors from the significant risk of eye damage, vertigo, and neuropathic effects, which constitute material impairments of health within the meaning of the Act. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

DICHLOROMONOFLUOROMETHANE
CAS: 75–43–4; Chemical Formula: $CHCl_2F$
H.S. No. 1128

In construction and maritime, OSHA has a limit of 1000 ppm as an 8-hour TWA for dichloromonofluoromethane (FC–21). There is no limit in agriculture. The ACGIH has a TLV®–TWA of 10 ppm for this substance; this limit is based on FC–21's similarity to chloroform in terms of hepatotoxic effects. There is no NIOSH REL. In construction, maritime, and agriculture, OSHA is proposing a TWA PEL for FC–21 of 10 ppm, and NIOSH concurs (Ex. 8–47, Table N1) with this limit. This is the limit recently established for FC–21 in general industry.

Dichloromonofluoromethane is a colorless gas. It is used as a solvent and refrigerant (Hawley's 1987, p. 379).

FC–21 is considered more toxic than the related difluorinated methanes. The major health hazards associated with exposure to this substance are liver damage, cardiac sensitization, and narcosis. Freon-21 has a 4-hour LC_{50} of 49,900 ppm in rats (Tappan and Waritz 1984, as cited in ACGIH 1986/Ex. 1–3, p. 187). Within an hour, exposure to 100,000 ppm killed rats and guinea pigs (Weigand 1971/Ex. 1–1102); other tests with guinea pigs and mice demonstrated that concentrations of 50,000 ppm and higher cause unconsciousness or death (Nuckolls 1935, as cited in ACGIH 1986/Ex. 1–3, p. 187; Booth and Bixby 1932/Ex. 1–1079). The clinical signs of overexposure include loss of coordination, tremors, narcosis, and prostration, as well as possible lung and liver changes (Tappan and Waritz 1984, as cited in ACGIH 1986/Ex. 1–3, p. 187).

Two-week exposures of rats to 10,000 ppm for 6 hours daily caused hepatic failure or marked liver damage (Trochimowicz, Moore, and Chiu 1977/Ex. 1–34). A series of 90-day exposures of rats and dogs to concentrations of 1000 or 5000 ppm dichloromonofluoromethane resulted in bilateral hair loss, cirrhosis, and excessive mortality in rats at both exposure levels; dogs exhibited weight loss at both levels, but mild liver changes were observed only at the 5000-ppm level (Trochimowicz, Lyon, Kelly, and Chiu 1977, as cited in ACGIH 1986/Ex. 1–3, p. 187). Another uncompleted study reported liver pathology in rats repeatedly exposed for 90 days at 500 ppm, and probable liver pathology from similar exposures to 200 ppm; no hepatic effects were observed after exposure to 50 ppm (Allied Chemical Company 1978, as cited in ACGIH 1986/Ex. 1–3, p. 187).

Two of 12 dogs exposed to 10,000 ppm FC–21 plus intravenous epinephrine developed serious arrhythmias (Mullin, as cited in ACGIH 1986/Ex. 1–3, p. 187). Dogs and monkeys (anesthetized) demonstrated tachycardia and hypotension after exposure to FC–21 at levels between 50,000 and 100,000 ppm; bronchoconstriction was observed at 25,000 ppm (Aviado and Smith 1975/Ex. 1–82; Belej and Aviado 1975/Ex. 1–462). Anesthetized mice exposed to a concentration of 100,000 ppm FC–21 showed arrhythmia and cardiac sensitization to epinephrine (Aviado and Belej 1974/Ex. 1–615). Pre-implantation loss has been reported in pregnant rats exposed to FC–21 at 10,000 ppm on days 6 through 15 of gestation (Belej and Aviado 1975/Ex.1–462).

OSHA is proposing a TWA limit of 10 ppm for dichloromonofluoromethane in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risks of hepatotoxic effects, cardiac sensitization, and narcosis associated with exposure to this substance. OSHA believes that these exposure-related effects constitute material impairments of health within the meaning of the Act. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DIETHYL KETONE
CAS: 96–22–0; Chemical Formula: $C_2H_5COC_2H_5$
H.S. No. 1135

OSHA has no limit for diethyl ketone in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 200 ppm for this substance. There is no NIOSH REL. The proposed PEL in construction, maritime, and agriculture is 200 ppm as an 8-hour TWA, and NIOSH concurs (Ex. 8–47, Table N1) with this limit. This is the limit recently established for diethyl ketone in general industry.

Diethyl ketone is a colorless liquid with an acetone-like odor. This substance is used in medicine and in organic synthesis (Hawley's 1987, p. 394).

The oral LD_{50} for diethyl ketone in rats is reported to be 2.14 g/kg. Four of six rats died when exposed to diethyl ketone for four hours at 8000 ppm (Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1–440). In general, the toxicities of the methyl ketones increase with increasing molecular weight; diethyl ketone is somewhat less toxic than is methyl propyl ketone (TLV®–TWA of 200 ppm) (NIOSH 1978f, as cited in ACGIH 1986/Ex. 1–3, p. 199). Like all of the ketones, exposure to diethyl ketone causes mucous membrane and eye and skin irritation.

In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA PEL of 200 ppm for diethyl ketone, the same limit being proposed for methyl propyl ketone. The Agency preliminarily concludes that this limit is

necessary to reduce the significant risk of eye and skin irritation, which are material health impairments that are associated with exposure to diethyl ketone. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DIETHYLENE TRIAMINE
CAS: 111–40–0; Chemical Formula: $(NH_2CH_2CH_2)_2$ NH
H.S. No. 1138

OSHA has no limit for diethylene triamine (DETA) in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 1 ppm, with a skin notation, for this substance. There is no NIOSH REL. OSHA is proposing a PEL of 1 ppm as an 8-hour TWA for diethylene triamine in construction, maritime, and agriculture, and NIOSH concurs (Ex. 8–47, Table N1) with this limit. This is the limit recently established for this substance in general industry.

Diethylene triamine is a yellow liquid that has an ammonia-like odor. This substance is used as a solvent, fuel component, and saponification agent (Hawley's 1987, p. 391).

The acute intraperitoneal LD_{50} values for DETA are reported to be 71 and 74 mg/kg for the mouse and rat, respectively (Hine, Kodama, Anderson et al. 1958/Ex. 1–511). In the rat, the reported oral and percutaneous LD_{50} values are the same (1080 mg/kg); the dermal LD_{50} for the rabbit is 1090 mg/kg (Smyth, Carpenter, and Weil 1949/Ex. 1–528). Exposure to 300 ppm of diethylene triamine vapor for 8 hours failed to kill any of a group of exposed rats (Savitt 1955/Ex.1–663).

Sutton (1963/Ex. 1–1101) has reported that DETA causes severe corneal injury; solutions of 15 to 100 percent caused lasting corneal damage. If improperly controlled, the vapor and liquid cause sensitization of the respiratory tract and skin (American Industrial Hygiene Association 1960, as cited in ACGIH 1986/Ex. 1–3, p. 197). Dernehl (Ex. 1–728) demonstrated such sensitization in a study reported in 1951.

OSHA is proposing an 8-hour TWA limit of 1 ppm for diethylene triamine in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risk of skin and respiratory tract irritation and sensitization, all of which constitute material health impairments that are associated with occupational exposure to diethylene triamine. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DINITROBENZENE (ALL ISOMERS)
CAS: 528–29–0; 99–65–0; 100–25–4;
Chemical Formula: $C_6H_4(NO_2)_2$
H.S. No. 2071

In general industry, construction, and maritime, OSHA's permissible exposure limit for all isomers of dinitrobenzene is 1mg/m3 as an 8-hour TWA; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 1 mg/m³, with a skin notation, for the dinitrobenzene isomers. NIOSH has no REL for dinitrobenzene but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 1 mg/m³ for dinitrobenzene (all isomers) in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

The ortho- and para-isomers of dinitrobenzene are white crystals; the meta isomer takes the form of pale yellow crystals. Dinitrobenzene is used as a camphor substitute in the production of celluloids, in the manufacture of dyes, and in organic synthesis (ACGIH 1986, p. 214; Merck 1983, p. 477). The industrial product is usually a mixture of the three isomers (Clayton and Clayton 1981, p. 2455).

Dinitrobenzene causes eye and respiratory tract irritation, cyanosis, aplastic anemia, and liver damage in humans and animals and, in animals, testicular damage (NIOSH 1989, in J. Appl. Toxicol. 9(3):199–202). The oral LD_{50} for m-dinitrobenzene is 83 mg/kg in rats, and the lowest lethal oral dose of p-nitrobenzene in cats is 29 mg/kg (RTECS 1990). There are no acute toxicity data for the ortho-isomer. Dinitrobenzene is known to be readily absorbed through the skin, although specific toxicity data are not available. Rats were given a single oral dose of the m-isomer (20 mg/kg) showed cyanosis and methemoglobinemia (NIOSH 1989). Four or five 20 mg/kg doses caused ataxia (NIOSH 1989). m-Dinitrobenzene given orally to rats as a 1 percent suspension in corn oil caused signs and symptoms such as a reduction in ambulatory motion, ataxia, weakness, dyspnea, rapid heartbeat, cyanosis, coma, and respiratory failure (Cody et al. 1981, in HSDB 1985). The effects on male and female rats of oral dosing with 200 mg/l m-dinitrobenzene included reduced growth rates, weight loss, reductions in hematocrit and hemoglobin values, enlarged spleens, testicular atrophy in males, and yellow pigmentation of liver cells (Cody et al. 1981, in HSDB 1985). Rats were given single oral doses (50 mg/kg) of each of the dinitrobenzene isomers; only the m- and p-isomers caused visible cyanosis within 2 hours and enlarged spleens observable at autopsy, and only the m-isomer caused a statistically significant decrease in testis weight (Blackburn, Gray, Lloyd, Sheard, & Foster 1988, in Toxicol. Appl. Pharmacol. 92(1):54–64). In another experiment, rats were given single oral 48 mg/kg doses of dinitrobenzene and their testes were examined histologically at intervals of 1 to 175 days thereafter. The authors concluded that acute exposure to dinitrobenzene causes rapid and extensive disruption of spermatogenesis as well as lesions and occlusion of the seminiferous tubules (Hess, Linder, Struder, & Perrcault 1988, in J. Androl. 9(5):327–342).

Most signs and symptoms of overexposure to dinitrobenzene in humans are attributable to the loss of the blood's oxygen-carrying capacity (Proctor, Hughes, and Fischman 1988, p. 215). The onset of symptoms may be delayed for up to 4 hours, and the first effect may be an increasingly intense headache (Hamblin 1963, in Proctor, Hughes, and Fischman 1988, p. 215). Symptoms of weakness and dizziness occur at methemoglobin concentrations of 40 percent; methemoglobin concentrations of 70 percent cause ataxia, dyspnea, tachycardia, nausea, vomiting, and drowsiness (Hamblin 1963, in Proctor, Hughes, and Fischman 1988, p. 215). Coma occurs at levels above 70 percent; the lethal level is in the range of 85 to 90 percent methemoglobin (Chemical Safety Data Sheet SD–21 1967, in Proctor, Hughes, and Fischman 1988, p. 215). Alcohol ingestion increases the toxic effects of dinitrobenzene (Proctor, Hughes, and Fischman 1988, p. 215). The principal clinical sign of dinitrobenzene intoxication is cyanosis, which occurs at a methemoglobin concentration of 15 percent or more (Gosselin, Smith, and Hodge 1984, p. II–213; Hamblin 1963, in Proctor, Hughes, and Fischman 1988, p. 215). Workers exposed to dinitrobenzene have reported symptoms including a burning sensation in the mouth, dry throat, thirst, and, with more intense exposure, somnolence, staggering gait, and coma (von Oettingen 1941, in Proctor, Hughes, and Fischman 1988, p. 215). In munitions factories, exposure to dinitrobenzene has been associated with toxic hepatitis and aplastic anemia; other symptoms reported in these cases included headache, burning pain and parethesias in the feet, ankles, hands, and forearms,

and visual effects (Grant 1986, p. 356; Ellenhorn and Barceloux 1988, in HSDB 1989). Workers exposed to m-dinitrobenzene dust in 1969 developed cyanosis and experienced slight to moderate anemia; 10 years later in a follow-up on the same workers, no long-term adverse effects were seen (Okubo and Shigeta 1982, in HSDB 1985). In chronically exposed workers, vision impairment appears to be common; symptoms included contracted visual fields and reduced visual acuity with central scotomas (Hubner 1918; Lewin and Guillery 1913, in Grant 1986, p. 356).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to dinitrobenzene potentially causes eye and respiratory tract irritation, anoxia, anemia, and liver damage. The Agency preliminarily finds that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. OSHA believes that establishing an 8-hour TWA PEL of 1 mg/m³, and a skin notation, for dinitrobenzene in agriculture is necessary to significantly reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DINITROTOLUENE (ALL ISOMERS)

CAS: 25321–14–6; Chemical Formula: $CH_3C_6H_3(NO_2)_2$

H.S. No. 2072

In general industry, construction, and maritime, OSHA's permissible exposure limit for dinitrotoluene is 1.5 mg/m³ as an 8-hour TWA, with a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 1.5 mg/m³, and a skin notation, for dinitrotoluene. The NIOSH REL for this substance is the lowest feasible level; however, NIOSH concurs (Ex. 8–47, Table N6A) with the limit being proposed. OSHA is proposing an 8-hour TWA limit of 1.5 mg/m³, and a skin notation, for dinitrotoluene in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

The commercial grade of dinitrotoluene is a mixture of the ortho-, meta- and para-isomers and is an oily liquid; the pure isomers are yellow crystals. Dinitrotoluene is used in the manufacture of explosives and dyes and in organic synthesis (ACGIH 1986, p. 216; Hawley's 1987, p. 422).

Dinitrotoluene causes methemoglobinemia, anemia, and liver damage in humans and animals. In animals, dinitrotoluene has also caused liver tumors and reproductive effects. The oral LD_{50}s in rats, mice, and guinea pigs are 268 mg/kg, 790 mg/kg, and 1300 mg/kg, respectively (RTECS 1990). Cats tolerated repeated doses of 2 or 4 ml of 1 percent dinitrotoluene in cod liver oil up to a total of 24 ml and showed no signs of toxic effects (Kuhls 1905, in ACGIH 1986, p. 216). When administered cutaneously at a dose of 0.3 g/kg, cats showed no signs of toxic effects, but cats given two doses of 5 g died in 8 hours (Zieger 1913, in ACGIH 1986, p. 216). After oral treatment at unspecified doses for 5 days, male rats showed significantly elevated cytochrome P–450-dependent enzyme activities (Dent and Graichen 1982, in HSDB 1988). Hepatotoxic effects have also been observed in dogs, mice, and rats orally exposed to dinitrotoluene (NIOSH CIB 44 1985). In rats (130/sex/group) administered dietary doses of 3.5 or 14 mg/kg/day, a decrease in mean body weight and a high incidence of hepatic neoplasms and hepatotoxicity were observed, in addition to an increased incidence of mammary fibroadenomas and subcutaneous sarcomas (Chemical Industry Institute of Toxicology Report 1982, in HSDB 1988). Oral administration of dinitrotoluene caused reproductive effects, such as testicular and ovarian atrophy, decreased fertility, and decreased sperm count among rats, mice, and dogs (NIOSH CIB 44 1985). Block et al. (1988) evaluated the effects of 2,4-dinitrotoluene on the rat testis. Adult Sprague-Dawley rats were fed diets containing 0.1 or 0.2 percent dinitrotoluene for 3 weeks. Among rats fed the higher dose, the authors reported finding marked changes in sertoli cell morphology, including swollen mitochondria and distended endoplasmic reticulum. In addition, reduced weights of the epididymides and decreased sperm reserves were observed. These results indicate that dinitrotoluene can induce testicular injury and interfere with late-stage spermatogenesis.

In humans, exposure to dinitrotoluene causes anemia, methemoglobinemia, and liver damage. The effects of methemoglobinemia may include headache, fatigue, nausea, vomiting, chest pain, and weight loss (Proctor, Hughes, and Fischman 1988, p. 218). At a methemoglobin concentration of 15 percent, cyanosis occurs, with blueness of the lips, nose, and earlobes as the first signs of overexposure to dinitrotoluene; at a methemoglobin level of 40 percent, there is weakness and dizziness; and at a 70-percent concentration, methemoglobinemia causes ataxia, dyspnea, tachycardia, nausea, vomiting, and drowsiness (Hamblin 1963, in Proctor, Hughes, and Fischman 1988, p. 218). Ingestion of alcohol and high ambient temperatures can increase the toxic effects of dinitrotoluene exposure (von Oettingen 1941; Linch 1974, in Proctor, Hughes, and Fischman 1988, p. 218). Severe burns of the skin and eyes, with permanent scarring, were caused by hot fumes of dinitrotoluene (Smejkal 1949, in Grant 1986, p. 416). A workman employed in a dinitrotoluene production facility for a year developed tingling and numbness in the toes and legs; after 2 years, the symptoms became worse and vision decreased, but these disorders improved within a year after the cessation of exposure (Hamilton and Nixon 1918, in Grant 1986, p. 416). Chronic exposure to dinitrotoluene has been reported to cause jaundice and anemia (Clayton and Clayton 1982, p. 2463). A medical survey conducted in Kentucky where workers were exposed to dinitrotoluene and toluenediamine revealed a significant reduction in sperm count among males, suggesting that these substances may adversely affect the male reproductive system (Ahrenholz 1980, in HSDB 1988).

Based on this evidence in humans and animals, OSHA preliminarily concludes that dinitrotoluene causes methemoglobinemia, anemia, and liver injury. OSHA preliminarily finds that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that establishing a permissible exposure limit of 1.5 mg/m³ as an 8-hour TWA for dinitrotoluene is necessary to significantly reduce the risks of these material health impairments. In addition, promulgation of this PEL for dinitrotoluene will make the PEL for this substance consistent across all OSHA-regulated sectors.

DIPROPYL KETONE

CAS: 123–19–3; Chemical Formula: $(CH_3CH_2CH_2)_2CO$

H.S. No. 1148

OSHA has no limit for dipropyl ketone in construction, maritime, or agriculture. The ACGIH has a TLV® of 50 ppm TWA for this substance. NIOSH has no REL. In construction, maritime, and agriculture, OSHA is proposing a PEL of 50 ppm as an 8-hour TWA, and NIOSH concurs (Ex. 8–47, Table N1) with this limit. This is the limit recently established for this substance in general industry.

Dipropyl ketone is a colorless liquid with a pleasant odor. It finds use as a solvent for nitrocellulose, raw and blown oils, resins, and polymers. It is

also used in lacquers and flavorings (Hawley's 1987, p. 431).

Dipropyl ketone has a moderate oral and inhalation toxicity. In rats, the oral LD_{50} is 3.35 g/kg, and the dermal LD_{50} in rabbits is 9.5 g/kg. Tests have indicated that rats inhaling 2000 ppm for 4 hours survived, but at 4000 ppm all animals died (Carpenter, Weil, and Smyth 1974/Ex. 1–304). Methyl isobutyl ketone (MIBK) has a similar acute toxicity (ACGIH 1986/Ex. 1–3, p. 221); OSHA is establishing a 50 ppm 8-hour TWA and a 75 ppm STEL for MIBK.

OSHA is proposing to establish an 8-hour TWA PEL of 50 ppm TWA for dipropyl ketone in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit is necessary to protect workers in these sectors from the significant risk of narcosis and irritation, both material health impairments that are associated with exposure to dipropyl ketone. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DIQUAT

CAS: 85–00–7; Chemical Formula: $C_{12}H_{12}Br_2N_2$

H.S. No. 1150

OSHA has no PEL for diquat in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 0.5 mg/m³ for this substance. The proposed PEL is 0.5 mg/m³ as an 8-hour TWA, and NIOSH concurs (Ex. 8–47, Table N1) with this limit. OSHA is proposing this limit for diquat in the construction, maritime, and agriculture industries. This is the limit recently established for this substance in general industry.

Diquat is a yellow crystalline solid that is used as a herbicide and as a plant growth regulator (Hawley's 1987, p. 431). When used in pesticidal applications and in accordance with directions on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In most species, the acute oral toxicity of diquat is similar to that of paraquat; the LD_{50} for diquat ranges from 100 to 400 mg/kg in rats, mice, rabbits, and dogs. The 24-hour percutaneous LD_{50} in rabbits is greater than 400 mg/kg; no skin irritation or other ill effects were demonstrated at this level (Clark and Hurst 1970/Ex. 1–135; Rowe and Wright 1965, as cited in ACGIH 1986/Ex. 1–3, p. 222). Rats fed 1000 ppm of diquat daily (about 50 mg/kg/day) for two years survived; reduced food intake and growth were the only consequences observed. At 500 ppm (about 25 mg/kg/day), the only ill effect observed was a pathologic change in the eye. A dietary level of 10 ppm (about 0.5 mg/kg/day) for 2 years did not induce cataract formation, but cataracts do occur at higher levels, with pathology observed at 500 ppm; one in four animals demonstrated complete corneal opacity in one or both lenses after 6 months at the 1000-ppm level. Cataract formation requires prolonged exposure and is not induced by single high-level exposures (ACGIH 1986/Ex. 1–3, p. 222).

Unlike paraquat, diquat does not produce lung damage in exposed humans or animals. Acute poisoning may produce nonspecific respiratory distress as well as other nonspecific signs of poisoning. In humans, accidental ingestion has produced less toxic reactions than those associated with paraquat ingestion (Orepoulos and McEvoy 1969/Ex. 1–429).

In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA PEL of 0.5 mg/m³ TWA for diquat. The Agency preliminarily concludes that this limit will protect workers in these sectors from the significant risk of ocular damage, which constitutes a material health impairment that is associated with chronic exposure to diquat. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DISULFOTON

CAS: 298–04–4; Chemical Formula: $C_8H_{19}O_2PS_3$

H.S. No. 1152

OSHA has no exposure limit for disulfoton in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 0.1 mg/m³ for this substance. There is no NIOSH REL. The proposed PEL for disulfoton in construction, maritime, and agriculture is 0.1 mg/m³ as an 8-hour TWA, with a skin notation. This is the limit recently established for this substance in general industry.

Pure disulfoton is an oily, colorless liquid; the technical grade is a brown liquid. This substance is a systemic insecticide and acaricide (Hawley's 1987, p. 435). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The acute toxicity of disulfoton is very high by all laboratory-tested routes of administration. For weanling rats, the intraperitoneal LD_{50} is reported to be 5.4 mg/kg; for adult rats, it is 9.4 mg/kg (Brodeur and Dubois 1963/Ex. 1–718). The acute dermal LD_{50} is 6 mg/kg for adult female rats and 25 mg/kg for adult male rats (Gaines 1969/Ex. 1–320). The acute oral LD_{50}s for male and female rats are reported as 6.8 mg/kg and 2.3 mg/kg, respectively (Brodeur and Dubois 1964/Ex. 1–1015). Rats have demonstrated an acquired tolerance for disulfoton (Brodeur and Dubois 1964/Ex. 1–1015).

Metabolically, disulfoton is highly fat-soluble, and the compound apparently interferes with mixed-function oxidase activity in the same manner shown to be the case for parathion; with respect to median lethal doses, parathion and disulfoton are similar (Stevens et al. 1973, as cited in ACGIH 1986/Ex. 1–3, p. 226).

OSHA is proposing an 8-hour TWA PEL for disulfoton of 0.1 mg/m³, with a skin notation, in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will prevent the significant risk of acute toxicity and metabolic injury, which are material impairments of health that are associated with exposure to this substance. The skin notation is included to protect workers in these sectors against the dermal toxicity that has been demonstrated in animal tests. Promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

DIVINYL BENZENE

CAS: 108–57–6; Chemical Formula: $C_6H_4(CHCH_2)_2$

H.S. No. 1154

OSHA has no limit for divinyl benzene in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 10 ppm, based on this substance's similarity to styrene. There is no NIOSH REL. In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA PEL of 10 ppm. NIOSH concurs (Ex. 8–47, Table N1) with this limit, which was recently established for this substance in general industry.

The commercial grade of divinyl benzene is a pale-straw-colored liquid; it contains all three isomers, but the meta isomer predominates. This substance is used as a polymerization monomer for various materials (Hawley's 1987, p. 437).

The oral LD_{50} in rats is reported to be 4.1 g/kg, and an acute inhalation study showed no ill effects from a single 7-hour exposure at 351 ppm. However, repeated or prolonged contact with the liquid may cause skin burns (Dow Chemical Company 1977j, as cited in ACGIH 1986/Ex. 1–3, p. 228).

Industrial experience indicates that irritation of the respiratory system, skin, and eyes can result from inhalation exposures to divinyl benzene, but there are no data concerning chronic exposures in humans.

OSHA is proposing a PEL of 10 ppm (8-hour TWA) for divinyl benzene in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risk of irritation to the respiratory tract, eyes, and skin; such irritation constitutes a material impairment of health within the meaning of the Act. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ENDOSULFAN
CAS: 115–29–7; Chemical Formula: $C_9H_6Cl_6O_3S$
H.S. No. 1156

OSHA has no permissible exposure limit for endosulfan in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 0.1 mg/m³, with a skin notation. There is no NIOSH REL. The proposed PEL is 0.1 mg/m³ as an 8-hour TWA, with a skin notation; NIOSH concurs (Ex. 8–47, Table N1) with this limit. This is the limit recently established for this substance in general industry.

Technical endosulfan is a tan, semi-waxy solid mixture; it may have a slight odor similar to that of sulfur dioxide. This substance is used as an insecticide (Hawley's 1987, p. 462). When used in pesticidal applications and in accordance with directions on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Endosulfan is similar in its acute oral toxicity to the related insecticides aldrin and dieldrin (TLV®–TWAs of 0.25 mg/m³), except that it is slightly more toxic than these substances in female laboratory animals. In rats, the oral LD_{50} for endosulfan is 43 mg/kg for males and 18 mg/kg for females (*Farm Chemicals Handbook* 1974/Ex. 1–1147a). The dermal LD_{50}s in male and female rats are 130 mg/kg and 74 mg/kg, respectively (*Farm Chemicals Handbook* 1974/Ex. 1147a). The respiratory LC_{50} for male rats is 50 mg/kg for 4 hours of exposure (Association of American Pesticide Control Officials, Inc. 1969, as cited in ACGIH 1986/Ex. 1–3, p. 230).

In laboratory tests of chronic exposure, rats tolerated oral doses of up to 3.2 mg/kg/day for 3 months without injury (Gaines 1975, as cited in ACGIH 1986/Ex. 1–3, p. 230), and dogs tolerated doses up to 0.75 mg/kg for 1 year (Ely, MacFarlane, Galen, and Hines 1967/Ex. 1–414). A 2-year dietary level at 10 ppm (approximately 0.5 mg/kg/day) in rats was associated with a statistically insignificant decline in female survival rates and a reduction in testis weights in males. At 5.0 mg/kg/day, histopathologic findings showed renal tubular damage and some hydropic changes in rat livers (Czech 1958, as cited in ACGIH 1986/Ex. 1–3, p. 230).

Inhalation of endosulfan dust by humans has been associated with slight nausea, confusion, excitement, flushing, and dry mouth (State of California, Department of Industrial Relations/Ex. 1–8). Nine employees who had been working with 50-percent water-wettable endosulfan powder for only a few days had convulsions (Association of American Pesticide Control Officials, Inc. 1969, as cited in ACGIH 1986/Ex. 1–3, p. 230).

OSHA preliminarily concludes that exposure to endosulfan poses a significant risk of systemic poisoning and renal and testicular damage, and the Agency therefore is proposing a PEL of 0.1 mg/m³ TWA for endosulfan in construction, maritime, and agriculture, with a skin notation; these effects constitute material impairments of health within the meaning of the Act. OSHA preliminarily finds that this limit will substantially reduce the significant risk associated with exposure to this substance at the levels permitted by the absence of a limit. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

EPN (Ethyl-p-nitrophenylthionobenzenephosphate)
CAS: 2104–64–5; Chemical Formula: $C_{14}H_{14}NO_4PS$
H.S. No. 2074

In general industry, construction, and maritime, OSHA's permissible exposure limit for EPN is 0.5 mg/m³ as an 8-hour TWA; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 0.5 mg/m³, with a skin notation, for EPN. The limit for EPN was set based on analogy with parathion, which appears to be about five times more toxic than EPN but causes similar effects (ACGIH 1986, p. 234). NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing to establish an 8-hour TWA PEL of 0.5 mg/m³, with a skin notation, for EPN in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

EPN is a light yellow solid or brown crystalline substance. It is used as an acaricide and as an insecticide in cotton insect pest control (ACGIH 1986, p. 234; Hawley's 1987, p. 466). When used in pesticidal applications and in accordance with directions on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

EPN is an anticholinesterase agent and neurotoxin in humans and animals. The oral LD_{50} in rats is 7 mg/kg; in dogs, it is 20 mg/kg (RTECS 1990). Dermal LD_{50}s in rats and rabbits are 25 mg/kg and 30 mg/kg, respectively (RTECS 1990). Administration of a single dermal dose of 22.5 to 225 mg/kg or of 0.5 to 2.0 mg/kg/day for 90 days caused delayed neurotoxicity in cats; these animals developed leg weakness and ataxia. At autopsy, histological changes were seen in the central and peripheral nervous systems of subchronically exposed animals (Abou-Donia 1983). Although a dietary level of 50 ppm for 1 year (equivalent to 210 mg/person/day) caused no observable effects in rats, 180 ppm (equivalent to 630 mg/person/day) caused damage to the adrenal cortex tissue within 52 weeks (Lehman 1952, in ACGIH 1986, p. 234). In a study in which rats were maintained on a dietary EPN level of 25 ppm for 13 weeks, some cholinesterase inhibition was observed; the same results were seen in rats given 5 ppm (0.24 mg/kg/day) or more for 13 weeks (DuBois et al. 1956; Suzuki 1973, in Hayes 1982, p. 410).

In humans, ingestion, inhalation, or dermal absorption of EPN produces signs and symptoms of anorexia, nausea, vomiting, abdominal cramps, diarrhea, chest tightness, wheezing, bronchial secretions, excessive salivation, cyanosis, slurred speech, confusion, convulsions, and coma (Proctor, Hughes, and Fischman 1988, p. 226). A 73-year-old man who ingested a dose of EPN estimated to be about 1429 mg/kg showed typical signs of anticholinesterase poisoning, including muscle spasms and coma; the patient died after 14 days (Hosaka and Yamaura 1976, in Hayes 1982, pp. 410–411). In studies with volunteers who ingested EPN doses beginning at 3 mg/person/day, the toxicity threshold appeared to be 9 mg/person/day (≈0.13 mg/kg/day) (Rider et al. 1959; Moeller and Rider 1959, 1962a, in Hayes 1982, p. 410). Ten volunteers ingested 6 mg/person/day of EPN for 47 days without observable effects on their cholinesterase activity (Rider et al. 1959; Moeller and Rider 1959, 1962a, in Hayes 1982, p. 410). An accidental release of EPN at a manufacturing facility caused muscle weakness and cerebellar effects in exposed workers (Froines 1979, in NIOSH Testimony on Neurotoxic Chemicals).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to EPN causes cholinesterase inhibition. OSHA preliminarily finds that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that establishing a PEL of 0.5 mg/m³ as an 8-hour TWA, and a skin notation, for EPN in agriculture is necessary to substantially reduce these risks of material health impairment. In addition, establishing this limit for EPN in agriculture will make OSHA's PEL for this substance consistent across all OSHA-regulated sectors.

FONOFOS

CAS: 944–22–9; Chemical Formula: $C_{10}H_{15}OPS_2$

H.S. No. 1181

OSHA has no limit for fonofos in the construction, maritime, or agriculture industry. The ACGIH has a TLV®–TWA of 0.1 mg/m³, with a skin notation, for this substance. There is no NIOSH REL. The proposed PEL is an 8-hour TWA of 0.1 mg/m³, with a skin notation; NIOSH concurs (Ex. 8–47, Table N1) with this limit. This is the limit recently established for this substance in general industry.

Fonofos is a light-yellow liquid (ACGIH 1986, p. 275). This substance is used as a soil insecticide (Hawley's 1987, p. 449). When used in pesticidal applications and in accordance with directions on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In male rats, the average acute oral LD_{50} of technical fonofos has been reported to be 13.2 mg/kg (Stauffer Chemical Co. 1974, as cited in ACGIH 1986/Ex. 1–3, p. 275). For female rats, an average oral LD_{50} of 3 mg/kg has been reported (NIOSH 1974d). The acute dermal LD_{50}s reported for rats and guinea pigs are 147 and 278 mg/kg, respectively (Weir and Hazleton 1981/Ex. 1–1135). Weir and Hazleton reported that no localized eye irritation occurred when 0.1 ml of technical fonofos was instilled into rabbit eyes; however, death resulted in these animals within 24 hours after the instillation (1981/Ex.1–1135). Dietary studies in rats lasting 105 weeks have shown 10 ppm (about 0.2 mg/kg) to be a no-effect level. Dogs fed fonofos for 14 weeks showed no-effect dietary levels of 8 ppm; no carcinogenic effects were observed. Rats showed reproductive effects at dietary levels of 10 ppm and 31.6 ppm (about 0.7 mg/kg) (Stauffer Chemical Co. 1974, as cited in ACGIH 1986/Ex. 1–3, p. 275).

There are no reports of human poisonings caused by fonofos, although it is known to be a cholinesterase inhibitor (ACGIH 1986/Ex. 1–3, p. 275).

OSHA is proposing an 8-hour PEL of 0.1 mg/m³ TWA for fonofos to protect exposed workers in construction, maritime, and agriculture from the significant risk of cholinesterase inhibition that is characteristic of exposure to this and other organophosphate pesticides. OSHA considers cholinesterase inhibition a material impairment of health. A skin notation is also proposed, based on evidence in animals that fonofos can readily penetrate the skin and cause death. The Agency preliminarily concludes that this limit will substantially reduce this significant risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

FORMAMIDE

CAS: 75–12–7; Chemical Formula: CH_3NO

H.S. No. 1182

OSHA's limit for formamide in the construction and maritime industries is 20 ppm as an 8-hour TWA. The ACGIH has a TLV®–TWA of 20 ppm and a TLV®–STEL of 30 ppm for this substance. There is no NIOSH REL. OSHA is proposing PELs of 20 ppm as an 8-hour TWA and 30 ppm as a 15-minute STEL for formamide in construction, maritime, and agriculture. These are the limits recently established for this substance in general industry.

Formamide is a colorless, oily liquid that is odorless (ACGIH 1986, p. 278.5). This substance is used as a solvent, a softener, and a chemical intermediate (Hawley's 1987, p. 537).

Formamide has an LD_{50} of approximately 6 g/kg for rats (Thiersh 1962/Ex. 1–690; Zaeva, Vinogradova, Savina, and Osipenko 1969/Ex. 1–1028). Dietary administration at 1.5 g/kg for 2 weeks resulted in fatalities in rats; pathologic examination revealed cumulative changes characteristic of gastritis and malnutrition (E.I. du Pont de Nemours and Company, Inc., as cited in ACGIH 1986/Ex. 1–3, p. 278). Czajkowska (1981, as cited in ACGIH 1986/Ex. 1–3, p. 278) reports the dermal LD_{50} for skin absorption in rabbits as 6 g/kg. Eye, mucous membrane, and skin irritation occur on exposure to this substance (Sax and Lewis 1989, p. 1766). Eye irritation tests in rabbits showed only slight, temporary irritation (Carpenter and Smyth 1946/Ex. 1–303). No signs of toxicity were detected in rats in single 6-hour exposures at 3900 ppm formamide dispensed as a mist, or in 6-hour daily exposures for 10 days at approximately 1500 ppm formamide vapor (equivalent to air saturated with formamide at room temperature); no indications of organ damage were seen in these animals on pathologic examination (E.I. du Pont de Nemours and Company, Inc., as cited in ACGIH 1986/Ex. 1–3, p. 278).

Gross fetal malformations were not noted following dermal applications of formamide to the skin of pregnant rats; the effects that were observed were weak and were produced at overwhelming concentrations (Stula and Krauss 1977/Ex. 1–1068). The no-observed-effect level in a rabbit developmental toxicity study was 22 mg/kg orally (Merkle and Zeller 1980/Ex. 1–683).

According to the ACGIH, there are no reports of industrial poisoning by formamide (E.I. du Pont de Nemours and Company, Inc., as cited in ACGIH 1986/Ex. 1–3, p. 278).

In the prior rulemaking, NIOSH pointed out (Ex. 8–47, Table N2) that formamide is a testicular toxin and has been identified as a teratogen in mice. OSHA is aware of the developing literature on both formamide and dimethyl formamide; however, the primary objective of the present rulemaking is to make OSHA's limits consistent across all regulated sectors. At the present time, OSHA is proposing a PEL of 20 ppm TWA and a STEL of 30 ppm for formamide in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risks of eye and skin irritation as well as other health effects associated with occupational exposure to formamide at levels above the proposed PELs. OSHA considers sensory irritation, testicular toxicity, and teratogenicity material impairments of health within the meaning of the Act. Promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors. At the time of the first PEL update, OSHA will review the literature on the toxicity of formamide to determine whether a further reduction in the PEL is necessary.

GERMANIUM TETRAHYDRIDE

CAS: 7782–65–2; Chemical Formula: GeH_4

H.S. No. 1186

OSHA has no limit for germanium tetrahydride in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 0.2 ppm for this substance. There is no NIOSH REL. The proposed PEL is an 8-hour TWA of 0.2 ppm, with which NIOSH concurs (Ex. 8–47, Table

N1). OSHA is proposing this limit for germanium tetrahydride in the construction, maritime, and agriculture industries. This is the limit recently established for this substance in general industry.

Germanium tetrahydride is a colorless gas. This substance is used in the electronics industry (ACGIH 1986, p. 284).

An early study indicated that germanium tetrahydride has a toxicity between that of tin hydride and arsine (Flury and Zernik 1931e/Ex. 1–993). In this study, a rabbit survived exposure to 100 ppm for one hour. One-hour exposures at 150 and 185 ppm caused fatalities in mice, and similar exposures involving guinea pigs resulted in sickness at the 150-ppm level and death at 185 ppm (Flury and Zernik 1931e/Ex. 1–993). On the other hand, Webster (1946/Ex. 1–399) reported that germanium tetrahydride is less toxic than both tin hydride and arsine. The effect of exposure to germanium tetrahydride is hemolysis. Data concerning chronic or subacute toxicities are not available. Based on germanium's acute toxicity, which is approximately half that of stibine, the ACGIH recommends an 8-hour TLV® of 0.2 ppm TWA.

In construction, maritime, and agriculture, OSHA is proposing a PEL of 0.2 ppm as an 8-hour TWA for germanium tetrahydride to reduce the significant risk of hemolytic effects, which constitute material impairments of health that are associated with occupational exposure to this substance. The Agency preliminarily concludes that implementation of this limit will substantially reduce this significant risk for workers in these sectors. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

INDENE

CAS: 95–13–6; Chemical Formula: C_9H_8

H.S. No. 1212

OSHA's current limit for indene in the construction and maritime industries is 10 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 10 ppm for this substance. The proposed PEL for indene in construction, maritime, and agriculture is 10 ppm as an 8-hour TWA, a limit with which NIOSH concurs (Ex. 8–47, Table N1). This is the limit recently established for indene in general industry.

Indene is a colorless liquid. This substance is used as a chemical intermediate (Hawley's 1987, p. 634).

Early inhalation studies of indene reported injury to the spleen, liver, and kidneys of rats exposed to indene vapor concentrations of 800 to 900 ppm for six 7-hour periods (Cameron and Doniger 1939/Ex. 1–470). Some animals were found at necropsy to have severe necrosis of the liver with hemorrhage; kidney necrosis was also observed. No other organ damage was found and no deaths occurred as a result of these exposures (Cameron and Doniger 1939/Ex. 1–470). By analogy with the effects of exposure to other monoaromatic hydrocarbons, exposure to indene is likely to irritate the mucous membranes. In laboratory animals, chemical pneumonitis, pulmonary edema, and hemorrhage have resulted from the aspiration of indene liquid into the lung, and repeated skin contact has caused dermatitis as a result of the defatting properties of indene (Gerarde 1960b/Ex. 1–738b). In dermal studies of rats, one to eight applications of 0.1 ml to the shaved skin were reported to have no effect; three applications of 0.5 ml to guinea pig skin also produced no effect (Cameron and Doniger 1939/Ex. 1–470). The oral toxicity of indene appears to be moderate, with adult rabbits tolerating a single dose of 1 gram without signs of systemic toxicity (Gerarde 1960b/Ex. 1–738b). Subcutaneous injection of 1 gram, however, caused liver pathology and fatalities; high oral doses (2.5 ml of a 1:1 v/v mixture in olive oil) were uniformly fatal, with characteristic liver, lung, and gastrointestinal changes. Chronic administration of 3 mg/m³ indene for 105 days caused catalase inhibition and stimulation of blood cholinesterase in rats, but no effects were observed in rats exposed at 0.6 mg/m³ (Dyshinevich 1976/Ex. 1–631).

OSHA is proposing to establish an 8-hour TWA PEL of 10 ppm for indene in construction, maritime, and agriculture. OSHA preliminarily concludes that this limit will reduce the significant risks of irritation, pulmonary effects, and systemic toxicity, which constitute material impairments of health that are associated with occupational exposure to this substance. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

IODOFORM

CAS: 75–47–8; Chemical Formula: CHI_3

H.S. No. 1214

OSHA has no limit for iodoform in construction, maritime, or agriculture. The ACGIH has an 8-hour TLV®–TWA limit of 0.6 ppm for this substance. The proposed PEL for iodoform in construction, maritime, and agriculture is 0.6 ppm as an 8-hour TWA; NIOSH concurs (Ex. 8–47, Table N1) with this limit, which is identical to the PEL recently established for this substance in general industry.

Iodoform is a yellow-green solid with a penetrating odor. It is used in medicine as an external antiseptic (Hawley's 1987, p. 643).

The subcutaneous LD_{50} for rabbits is 50 mg/kg, and the oral LD_{Lo} for iodoform in dogs is 1000 mg/kg (Kutob and Plaa 1962/Ex. 1–61). These authors also report that, on a molar basis, iodoform has an acute toxicity in mice similar to that of methyl iodide; this conclusion is based on parameters of lethality, barbiturate sleeping time, and bromsulphalein (BSP) retention time. An NCI bioassay (1978c/Ex. 1–1117) of iodoform indicates that the substance is not carcinogenic nor of high systemic toxicity, although histopathological examination of laboratory animals in this bioassay was judged by NCI to be inadequate.

No human data are available for this compound.

OSHA is proposing an 8-hour TWA limit of 0.6 ppm for iodoform; OSHA preliminarily concludes that this limit will protect workers in construction, maritime, and agriculture from the significant risks of irritation and hepatotoxicity, both material impairments of health that are associated with exposure to iodoform. The Agency has preliminarily determined that this limit will substantially reduce these significant risks for workers in these sectors. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ISOBUTYL ACETATE

CAS: 110–19–0; Chemical Formula: $CH_3COOCH_2CH(CH_3)_2$

H.S. No. 2097

In general industry, construction, and maritime, OSHA currently has an 8-hour TWA limit of 150 ppm for isobutyl acetate. The Agency has no PEL for this substance in agriculture. The 1987–1988 ACGIH TLV®–TWA for isobutyl acetate was 150 ppm as an 8-hour TWA and 187 ppm as a STEL. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the PEL being proposed for this substance. OSHA is proposing an 8-hour TWA PEL of 150 ppm for isobutyl acetate in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Isobutyl acetate is a colorless liquid with a fruity odor similar to that of the acetate esters. Isobutyl acetate is used in thinners, sealants, and topcoat lacquers. It is also used as a solvent for nitrocellulose, as a flavoring agent in

beverages, and in the manufacture of perfume (ACGIH 1986, p. 330; Genium MSDS 1990, No. 396).

Isobutyl acetate causes skin, eye, and mucous membrane irritation and, at high concentrations, this substance causes central nervous system depression in humans and animals (Proctor, Hughes, and Fischman 1988, p. 289; NIOSH/OSHA Occupational Health Guideline 1981, p. 1). This substance appears to be more acutely toxic than n-butyl acetate but less irritating to the eyes, mucous membranes, and skin than the n-isomer (ACGIH 1986, p. 330). The oral LD_{50}s in rats and rabbits are 13,400 mg/kg and 4763 mg/kg, respectively (RTECS 1990). The lowest lethal concentration in rats is 8000 ppm for 4 hours; no deaths occurred in rats exposed to 4000 ppm (Smyth et al. 1962, in Proctor, Hughes, and Fischman 1988, p. 289; RTECS 1990). Rabbits showed moderate skin irritation when 500 mg of isobutyl acetate was left on their skin for 24 hours; moderate eye irritation was observed when the same dose was instilled into rabbit eyes (RTECS 1990). Exposure to 21,000 ppm of isobutyl acetate for 150 minutes caused narcosis and death in six of six rats exposed; no symptoms were observed at a concentration of 3000 ppm for 8 hours (Clayton and Clayton 1981, p. 2273).

In humans, exposure to isobutyl acetate causes mild irritation of the eyes and mucous membranes; at high but unspecified concentrations, this substance causes central nervous system depression (HSDB 1985). The acute symptoms of central nervous system depression include headache, muscle weakness, confusion, giddiness, and delirium (Genium MSDS 1990, No. 396). The onset of isobutyl acetate-induced narcosis is gradual, and recovery after the cessation of exposure is likely to be slow (Clayton and Clayton 1982, p. 2268). Liquid isobutyl acetate defats the skin, and repeated contact causes irritation and may lead to dermatitis (Genium MSDS 19890, No. 396).

Based on this evidence in humans and animals, OSHA preliminarily concludes that isobutyl acetate is a skin, eye, and mucous membrane irritant and, at high concentrations, a narcotic. The Agency concludes that, in the absence of a limit for this substance, workers in agriculture are at significant risk of experiencing these effects, which constitute material impairments of health. OSHA believes that the proposed 8-hour TWA PEL of 150 ppm will substantially reduce these significant risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ISOBUTYL ALCOHOL

CAS: 78–83–1; Chemical Formula: $(CH_3)_2CHCH_2OH$

H.S. No. 1219

In construction and maritime, OSHA currently has a limit of 100 ppm as an 8-hour TWA for isobutyl alcohol. There is no limit in agriculture. The ACGIH has a limit of 50 ppm TLV®–TWA for this substance. In construction, maritime, and agriculture, OSHA is proposing 50 ppm as an 8-hour TWA; NIOSH concurs (Ex. 8–47, Table N1) with this limit. This is the limit recently established for this substance in general industry.

Isobutyl alcohol is a colorless liquid that is used as a solvent, in organic synthesis, and as an ingredient in a wide variety of consumer products (Hawley's 1987, p. 653).

Limited inhalation studies have reported a somewhat higher acute toxicity for isobutyl alcohol than for n-butyl alcohol (which has a ceiling of 50 ppm) (Smyth, Carpenter, and Weil 1951/Ex. 1–439; Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1–440). A 4-hour LC_{50} of 8000 ppm has been reported for isobutyl alcohol in rats. Ingestion studies in rabbits have reported an acute oral toxicity of 3.75 g/kg for isobutyl alcohol (Smyth, Carpenter, and Weil 1951/Ex. 1–439; Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1–440). The dermal LD_{50} is 4.2 g/kg (Stokinger 1976, as cited in ACGIH 1986/Ex. 1–3, p. 331). Weese (1928/Ex. 1–1073) reported that the narcotic inhalation dose over a total of 136 hours is 6400 ppm in mice. Slight changes in the liver and kidneys were reported at autopsy, but no fatalities occurred after repeated narcotizing doses (Weese 1928/Ex. 1–1073).

The effects of liquid isobutyl alcohol on the human eye appear to be comparable to those of n-butanol; no data are available on ocular exposure to the isobutyl alcohol vapor. Dermal application of isobutyl alcohol has caused slight erythema and hyperemia in humans (Schwartz and Tulipan 1939/Ex. 1–1167; Oettel 1936/Ex. 1–921).

OSHA is proposing to reduce the 8-hour TWA PEL for isobutyl alcohol of 100 ppm to 50 ppm in construction and maritime and also proposes a 50-ppm limit in agriculture. The Agency preliminarily concludes that a 50-ppm limit will reduce the significant risk of skin irritation, which is a material impairment of health that is associated with exposure to concentrations at levels above the revised PEL. Promulgation of this limit will also make OSHA's PEL for this substance consistent across all regulated sectors.

ISOOCTYL ALCOHOL

CAS: 26952–21–6; Chemical Formula: $CH_3(CH_2)_3CH(C_2H_5)CH_2OH$

H.S. No. 1220

OSHA has no PEL for isooctyl alcohol in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 50 ppm, with a skin notation, for this substance. The proposed PEL is 50 ppm as an 8-hour TWA, with a skin notation, for isooctyl alcohol in construction, maritime, and agriculture. NIOSH concurs (Ex. 8–47, Table N1) with this limit, which was recently established for this substance in general industry.

Isooctyl alcohol is a clear, combustible liquid. It finds use as an ingredient in plasticizers, an intermediate for nonionic detergents and surfactants, and as an ingredient in a variety of oils and fluids (Hawley's 1987, p. 658).

The single-dose oral LD_{50}s for isooctyl alcohol reported for rats and mice are between 3.2 and 6.4 g/kg; intraperitoneal injection LD_{50}s for these species range from less than 0.4 g/kg to 1.6 g/kg (Hodge 1943/Ex. 1–700; Fassett 1951, as cited in ACGIH 1986/Ex. 1–3, p. 332). The dermal LD_{50} for the guinea pig is greater than 10 ml/kg (Fassett 1951, as cited in ACGIH 1986/Ex. 1–3, p. 332); in the rabbit, the dermal LD_{50} is 2.38 ml/kg (Smyth, Carpenter, Weil et al. 1969/Ex. 1–442). Moderate skin irritation from exposure to isooctyl alcohol has also been reported. Rats and rabbits have shown skin irritation at exposure levels ranging from 1.7 to 3.34 ml/kg (Smyth, Carpenter, Weil, et al. 1969/Ex. 1–442). Fassett (1951, as cited in ACGIH 1986/Ex. 1–3, p. 332) also reported no fatalities in rats after an 8-hour inhalation test at 235 ppm.

OSHA is proposing to establish an 8-hour TWA PEL of 50 ppm, with a skin notation, for isooctyl alcohol in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will reduce the significant risks of skin irritation, a material impairment of health that is associated with exposure to this substance at levels above the new PEL. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

N–ISOPROPYLANILINE

CAS: 768–52–5; Chemical Formula: $C_6H_5NHCH(CH_3)_2$

H.S. No. 1229

OSHA has no limit for N-isopropylaniline in construction, maritime, or agriculture. The ACGIH recommends a TLV®–TWA of 2 ppm, with a skin notation, for this substance. There is no NIOSH REL for this

substance. OSHA is proposing an 8-hour TWA PEL of 2 ppm, with a skin notation, for this substance in construction, maritime, and agriculture; NIOSH concurs (Ex. 8–47, Table N1) with the proposed limit. This is the limit recently established for this substance in general industry.

N–Isopropylaniline is a yellowish, combustible liquid that is used as an intermediate and fiber dye (Hawley's 1987, p. 661).

The oral LD_{50} for rats exposed to N-isopropylaniline is between 0.25 and 0.5 g/kg. Slight irritation of the skin and eyes has been reported in animals as a result of direct contact with this chemical (Dow Chemical Company 1977k, as cited in ACGIH 1986/Ex. 1–3, p. 338). No other data concerning chronic toxicity or human exposure are available (ACGIH 1986/Ex. 1–3, p. 338).

Chemical analysis shows N-isopropylaniline to have toxicologic properties similar to those of its parent compound, aniline. The oral LD_{50}s for the two chemicals are approximately equal. OSHA has established a 2-ppm 8-hour TWA PEL for N-isopropylaniline on the basis of its structural analogy with aniline (which has the same PEL) and N,N-dimethylaniline (which has a 5-ppm 8-hour TWA PEL and a 10-ppm 15-minute STEL); exposure to these substances has been shown to cause hemolytic and central nervous system effects in animals and humans. These substances are also toxic when absorbed through the skin.

OSHA is proposing an 8-hour PEL of 2 ppm for N-isopropylaniline, with a skin notation, in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors from the significant risk of irritation and systemic and hemolytic effects, all material health impairments that are caused by inhalation, ingestion, or dermal absorption of N-isopropylaniline. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

KETENE

CAS: 463–51–4; Chemical Formula:

$CH_2=C=O$

H.S. No. 1231

OSHA's 8-hour TWA limit for ketene in construction and maritime is 0.5 ppm. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.5 ppm and a TLV®–STEL of 1.5 ppm for this substance. There is no NIOSH REL. In construction and maritime, OSHA is retaining the 8-hour TWA and proposing to add a 15-minute STEL of 1.5 ppm; the Agency is also proposing to extend both limits to agriculture. NIOSH concurs (Ex. 8–47, Table N–1) with the proposed limits, which are those recently established for this substance in general industry.

Ketene is a colorless gas with a sharp, penetrating odor. Ketene is used as an acetylating agent and as a starting point in the production of products such as acetic anhydride and acetate esters (ACGIH 1986, p. 341; Hawley's 1987, p. 672).

Ketene is highly irritating to the respiratory tract (Mendenhall and Stokinger 1959/Ex. 1–428), and the effects of its action are delayed (Treon, Sigmon, Kitzmiller 1949/Ex. 1–769). Mendenhall and Stokinger (1959/Ex. 1–428) have reported a 10-minute LC_{50} for mice of 17 ppm. Chronic exposure to 1 ppm for six months on a schedule of 6 hours/day, 5 days/week, was tolerated by animals of several species (Mendenhall and Stokinger 1980, as cited in ACGIH 1986/Ex. 1–3, p. 341). Similar results have been reported in monkeys exposed repeatedly (55 exposures) for seven hours at each exposure (Treon, Sigmon, and Kitzmiller 1949/Ex. 1–769). Evidence strongly suggests that the development of emphysema and fibrosis may occur in individuals who have developed a tolerance to the acute effects of ketene exposure (Stokinger, Wagner, and Dobrogarski 1957/Ex. 1–139).

In construction and maritime, OSHA is retaining the 8-hour TWA PEL of 0.5 ppm and proposing to add a 15-minute STEL of 1.5 ppm for ketene; OSHA is also proposing to extend both limits to agriculture. The Agency preliminarily concludes that workers exposed to this highly irritating and toxic gas are at significant risk of developing respiratory irritation, pulmonary edema, and other severe pulmonary effects that constitute material health impairments. OSHA preliminarily finds that both a TWA and STEL are required to protect workers in construction, maritime, and agriculture from ketene's acute and chronic health effects. The proposed limits are necessary to substantially reduce these risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

METHACRYLIC ACID

CAS: 79–41–4; Chemical Formula:

$CH_2=C(CH_3)COOH$

H.S. No. 1244

OSHA has no limit for methacrylic acid in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 20 ppm for this substance. There is no NIOSH REL. OSHA is proposing an 8-hour TWA PEL of 20 ppm for methacrylic acid in construction, maritime, and agriculture, and NIOSH concurs (Ex. 8–47, Table N1) that this limit is appropriate. This is the limit recently established for this substance in general industry.

Methacrylic acid is a liquid with an acrid, disagreeable odor. Metacrylic acid is used as a monomer for large volume resins and polymers and in organic synthesis (ACGIH 1986, p. 362; Hawley's 1987, p. 751).

The primary toxic hazard associated with exposure to methacrylic acid is irritation, although the degree of irritation from exposure to this substance is significantly less than that from exposure to acrylic acid (ACGIH 1986/Ex. 1–3, p. 362). Direct contact of methacrylic acid with the skin or eye can cause corrosion of the skin or blindness. In rabbits, the skin absorption LD_{50} for methacrylic acid is 0.5 to 1g/kg (Dow Chemical Company 1977m, as cited in ACGIH 1986/Ex. 1–3, p. 362). Rats exposed by inhalation to approximately 1000 ppm of methacrylic acid exhibited eye irritation (Dow Chemical Company 1977m, as cited in ACGIH 1986/Ex. 1–3, p. 362). Rats exposed to a 300 ppm concentration of this substance for 6 hours daily for 20 days showed slight congestion of the kidneys at autopsy (Gage 1970/ Ex. 1–318).

Medical reports of workers exposed acutely to concentrations of up to 113 ppm methacrylic acid in an industrial setting revealed that these workers experienced no respiratory symptoms; however, skin responses and a severe corneal burn were reported (Dow Chemical Company 1977m, as cited in ACGIH 1986/Ex. 1–3, p. 362).

OSHA is proposing a PEL of 20 ppm as an 8-hour TWA for methacrylic acid, with a skin notation, in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors from the significant risk of severe eye and skin irritation, which are material health impairments that are associated with occupational exposure to methacrylic acid. The skin notation is necessary to prevent dermal absorption and systemic toxicity. Promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

4-METHOXYPHENOL

CAS: 150–76–5; Chemical Formula:

$CH_3OC_6H_4OH$

H.S. No. 1247

OSHA has no limit for 4-methoxyphenol in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 5 mg/m³ for this

substance. There is no NIOSH REL, but NIOSH concurs with the proposed limit (Ex. 8–47, Table N1). In construction, maritime, and agriculture, OSHA is proposing a PEL of 5mg/m³; this is the limit recently established for this substance in general industry.

4-methoxyphenol is a white, waxy solid used in the manufacture of antioxidants, pharmaceuticals, plasticizers, and dyestuffs. 4-methoxyphenol is also used as a stabilizer for chlorinated hydrocarbons, as an inhibitor for acrylic monomers and acrylonitriles, and as a UV inhibitor (ACGIH 1986, p. 367; Hawley's 1987, p. 620).

In rats, the oral LD_{50} for 4-methoxyphenol is between 1 and 2 g/kg; the skin absorption LD_{50} in rabbits is greater than 1 g/kg. Results of a two-month dietary study demonstrated no ill effects at 0.1 ppm (approximately 50 mg/kg/day). Direct contact of 4-methoxyphenol with the skin or eyes causes burns or moderate corneal damage (Hodge, Sterner, Maynard, and Thomas 1949/ Ex. 1–41; Dow Chemical Company 1977n, as cited in ACGIH 1986/ Ex. 1–3, p. 367).

To reduce the risk of dermal and ocular effects resulting from exposure to 4-methoxyphenol, a compound similar in chemical structure and toxicity to hydroquinone, OSHA is proposing to establish a permissible exposure limit in construction, maritime, and agriculture of 5 mg/m³ as an 8-hour TWA. The Agency preliminarily concludes that this limit is necessary to protect workers in these sectors against the significant risk of dermal and skin effects potentially associated with exposure to this substance. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

METHYL ACETYLENE-PROPADIENE MIXTURE (MAPP)

CAS: None; Chemical Formula: C_3H_4 isomers

H.S. No. 1250

OSHA has a PEL of 1000 ppm as an 8-hour TWA for MAPP in construction and maritime. There is no limit in agriculture. The ACGIH also has an 8-hour TLV®–TWA limit of 1000 ppm and a TLV®–STEL of 1250 ppm for this substance. There is no NIOSH REL. OSHA is retaining the 8-hour PEL of 1000 ppm, proposing to add a STEL of 1250 ppm for this substance in construction and maritime, and proposing to extend both limits to agriculture. NIOSH concurs (Ex. 8–47, Table N1) with the limits being proposed, which were recently established for MAPP in general industry.

MAPP contains 58 percent of a mixture of propadiene (a colorless, unstable gas with a strong, unpleasant odor) and methyl acetylene (a colorless gas with a sweet odor); the balance of the mixture consists of paraffinic and olefinic C_3 and C_4 hydrocarbons. Methyl acetylene-propadiene mixture is used as an industrial fuel for cutting, welding, brazing, heat treating, and metallizing (Hawley's 1987, p. 756).

Rabbits, dogs, and guinea pigs exposed to an average MAPP concentration of 5000 ppm for 7 hours/day, 5 days/week for 4 months showed no exposure-related effects except decreased lung weights at autopsy. No changes were observed in animals exposed to 1000 ppm on this regimen for four months (Dow Chemical Company 1964, as cited in ACGIH 1986/Ex. 1–3, p. 368).

On the basis of these data, which show MAPP to be a chemical mixture of low toxicity in experimental animals, the Agency is retaining its 8-hour TWA PEL of 1000 ppm and proposing to add a STEL of 1250 ppm in construction and maritime, and is also proposing to extend both limits to agriculture. The Agency preliminarily concludes that both of these limits are necessary to ensure that workers in these sectors are protected and that good industrial hygiene practice is maintained. In addition, promulgation of these limits will make OSHA's PELs for MAPP consistent across all regulated sectors.

METHYLAL (Dimethoxymethane)

CAS: 109–87–5; Chemical Formula: $CH_3OCH_2PCJ_3$

H.S. No. 2108

The OSHA PEL for methylal in general industry, construction, and maritime is 1000 ppm as an 8-hour TWA. There is no limit for methylal in agriculture. The ACGIH has a TLV®–TWA of 1000 ppm for this substance. NIOSH has no REL for methylal, but concurs (Ex. 8–47, Table N1) with the PEL being proposed. OSHA is proposing a PEL of 1000 ppm as an 8-hour TWA for methylal in agriculture. Promulgation of this limit will make the PEL for methylal consistent across all OSHA-regulated sectors.

Methylal is a colorless, volatile, flammable liquid with a chloroform-like odor (Hawley's 1987, p. 756). Methylal is used as a specialized fuel, as a solvent in perfumes, adhesives, and coatings, and as a reaction medium for the Grignard and Reppe reactions in organic synthesis (Merck 1983, p. 864; ACGIH 1986, p. 371).

Methylal is an irritant of the eyes and mucous membranes and, at high concentrations, a central nervous system depressant in both humans and animals. The oral LD_{50} in rabbits is 5708 mg/kg; the LC_{50} in rats is 15,000 ppm for an unspecified period (RTECS 1987; Weaver et al. 1951, in Clayton and Clayton 1982, p. 2657). Exposure to a concentration of approximately 154,000 ppm methylal caused tearing of the eyes, sneezing, nasal discharge, coughing, and vomiting in guinea pigs; these animals became comatose in 20 minutes and died within 2.5 hours (Weaver et al. 1951, in Proctor, Hughes, and Fischman 1988, p. 319). In mice exposed to 15 daily 7-hour inhalation exposures to 11,000 ppm, minor irritation of the eyes and nose occurred and incoordination developed after 3 or 4 hours of exposure. Mice exposed to a 14,000-ppm concentration of methylal exhibited a greater degree of irritation and anesthesia (Weaver et al. 1951, in Clayton and Clayton 1982, p. 2657). Autopsy of guinea pigs exposed to very high but unspecified concentrations of methylal revealed moderate to severe fatty degeneration of the liver and kidneys, as well as extensive bronchopneumonia; however, no significant histopathological changes were noted in guinea pigs exposed for five daily 7-hour exposures to a 45,000-ppm concentration of methylal. Although methylal vapor is irritating to the eyes of experimental animals, no histological abnormalities of the optic nerve or retina have been reported (Weaver et al. 1951, in Clayton and Clayton 1982, p. 2657; Grant 1986, p. 606). In humans, methylal causes eye, mucous membrane, skin, and upper respiratory tract irritation; at high concentrations, central nervous system depression is observed. Methylal was formerly used as an anesthetic in human medicine; however, this use has been discontinued because the anesthetic effect of methylal is slower and more transitory than that of ether (Weaver et al. 1951, in Proctor, Hughes, and Fischman 1988, p. 319). Methylal has a defatting effect on the skin, and frequent or prolonged contact causes dermatitis (Proctor, Hughes, and Fischman 1988, p. 320).

Based on this evidence, OSHA is proposing a 1000 ppm 8-hour TWA limit to protect workers in agriculture from the significant risk of central nervous system depression caused by exposure to higher concentrations of methylal. The Agency preliminarily concludes that this limit is necessary to substantially reduce a significant risk of material health impairment. In addition, promulgation of this limit will make

OSHA's PEL for this substance consistent across all regulated sectors

METHYLAMINE

CAS: 74–89–5; Chemical Formula: CH_3NH_2

H.S. No. 2110

OSHA's PEL for methylamine in general industry, construction, and maritime is 10 ppm as an 8-hour TWA. There is no PEL for methylamine in agriculture. The ACGIH has a TLV®–TWA of 10 ppm for this substance. NIOSH has no REL for methylamine but concurs with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 10 ppm for methylamine in agriculture. Promulgation of this limit will make the PEL for methylamine consistent across all OSHA-regulated sectors.

Methylamine is a colorless, flammable gas with a fishy odor at low concentrations and an ammonia-like odor at high concentrations (Braker and Mossman 1980, p. 498). Methylamine is used as a chemical intermediate for accelerators, dyes, insecticides, fungicides, pharmaceuticals, and surface-active agents. It is also used as a fuel additive, a polymerization inhibitor, a solvent, a rocket propellant, a photographic developer, and a component of some paint removers (HSDB 1988).

Methylamine is an irritant of the eyes, mucous membranes, and respiratory tract in both humans and animals. Administered orally as the base in a 40 percent aqueous solution, the LD_{50} for rats is 0.1 to 0.2 g/k g; the LC_{50} in mice is 2400 mg/m³ for 2 hours (Clayton and Clayton 1982, p. 3148; RTECS 1987). Ocular damage, including hemorrhages of the conjunctiva, shallow corneal opacities, and edema have resulted from the administration of a single drop of 5 percent methylamine in water into the eyes of experimental animals (Grant 1986, p. 606). Corneal damage occurred in rabbit eyes after contact with a 40-percent methylamine solution, and necrosis of the skin developed in rabbits whose skin had been wetted with 0.1 milliliter of the same solution (Smyth and Carpenter 1944. In contact with liquefied methylamine, guinea pig skin swelled and turned purple after a few minutes. The skin turned gray and necrotic after 48 hours; fresh granulation tissue covered by flat epidermis, with no hair follicles, appeared 12 days after the exposure (Goffman and Maguire 1980, in HSDB 1988).

Human toxicity data for methylamine are limited. A chemical worker exposed to methylamine at concentrations ranging from 2 to 60 ppm for an unspecified period of time developed allergic or chemical bronchitis (ACGIH 1986, p. 373). Transient eye, nose, and throat irritation occurs when humans are exposed for brief periods to concentrations ranging from 20 to 100 ppm, although exposures to methylamine concentrations below 10 ppm do not cause irritation. Severe exposure to methylamine would be expected to cause pulmonary edema. Prolonged or repeated exposure of the skin or eyes causes dermatitis or inflammation of the conjunctiva, respectively (Proctor, Hughes, and Fischman 1988, p. 321).

Based on this evidence, OSHA is proposing a 10 ppm 8-hour TWA PEL to protect workers in agriculture from the significant risk of eye, nose, and respiratory tract irritation caused by exposure to methylamine. The Agency preliminarily concludes that this limit is necessary to substantially reduce a significant risk of material health impairment in exposed workers. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

METHYL DEMETON

CAS: 8022–00–2; Chemical Formula: $(CH_3O)_2PSO(CH_2)_2SC_2H5$

H.S. No. 1256

OSHA has no limit for methyl demeton in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 0.5 mg/m³, with a skin notation. There is no NIOSH REL, but NIOSH concurs (Ex. 8–47, Table N1) with the proposed limit. OSHA is proposing an 8-hour TWA of 0.5 mg/m³, and a skin notation, for methyl demeton in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Methyl demeton is an oily, colorless to pale-yellow liquid with an unpleasant odor. Methyl demeton is used as a systemic insecticide and acaricide (ACGIH 1986, p. 388; Hawley's 1987, p. 786). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Methyl demeton has an oral LD_{50} of 40 to 65 mg/kg for the thiolo-isomer and 150 to 250 mg/kg for the thiono-isomer. Both isomers form sulfoxide or sulfone, with an oral LD_{50} similar to that of the parent compounds (Dubois and Pizak 1962/Ex. 1–629; Heath and Vandekar 1965, Klimmer and Plaff 1955, both as cited in ACGIH 1986/Ex. 1–3, p. 388). In solution or storage, methyl demeton may form alkyl sulfonium compounds that have very high intravenous toxicity and oral LD_{50}s of 10 to 20 mg/kg. Dermal toxicity is reported to be moderate, with an LD_{50} of approximately 400 mg/kg (Heath and Vandekar 1965, as cited in ACGIH 1986/Ex. 1–3, p. 388).

In humans, methyl demeton causes changes in intraocular pressure, and acute poisonings produce nausea, headache, dizziness, vomiting, and hyperemia of the nasal mucosa. Chronic exposure causes hyperemia of the respiratory organs and irritation of the inner ear (Dugel'nyy 1970; Rasuleva 1970, both as cited in ACGIH 1986/Ex. 1–3, p. 388).

OSHA is proposing an 8-hour TWA PEL for methyl demeton of 0.5 mg/m³, with a skin notation, in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in this sectors from the significant risk of ocular and nasal irritation, pulmonary effects, and cholinesterase inhibition, all of which constitute material impairments of health that are associated with occupational exposure to this substance. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

METHYLENE BISPHENYL ISOCYANATE (MDI)

CAS: 101–68–8; Chemical Formula: $CH_2(C_6H_4NCO)_2$

H.S. No. 2109

OSHA's permissible exposure limit (PEL) for methylene bisphenyl isocyanate in general industry, construction, and maritime is 0.02 ppm (0.2 mg/m³) as a ceiling limit. There is no limit for this substance in agriculture. The ACGIH has assigned methylene bisphenyl isocyanate a TLV®–TWA of 0.005 ppm (0.050 mg/m³) (ACGIH 1989, p. 30). NIOSH has recommended exposure limits of 50 μg/m³ (0.005 ppm) as a 10-hour TWA and 200 μg/m³ (0.02 ppm) as a 10-minute ceiling limit for methylene bisphenyl isocyanate (NIOSH Recommendations 1988); however, NIOSH concurs with the limit being proposed by OSHA (Ex. 8–47, Table N1). OSHA is proposing a PEL of 0.02 ppm (0.2 mg/m³) as a ceiling limit for methylene bisphenyl isocyanate in agriculture. Promulgation of the proposed limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Methylene bisphenyl isocyanate is an odorless white to light-yellow solid (ACGIH 1986, p. 389(88)). Methylene bisphenyl isocyanate is used in the production of polyurethane foams and plastics. It is also used in coating systems for aircraft, tank trucks, automobiles, and truck trailers (Proctor,

Hughes, and Fischman 1988, p. 330; IARC 1979, Vol. 19, p. 317).

In both humans and animals, methylene bisphenyl isocyanate is an irritant of the skin, eyes, and mucous membranes; it is also a respiratory and skin sensitizer. The 4-hour LC_{50}s in rats are 36.2 ppm (males) and 37.3 ppm (females) (NIOSH 1978). By oral administration, methylene bisphenyl isocyanate is of low toxicity: the oral LD_{50} in rats is greater than 10 g/kg, and rats given oral doses of 5 g/kg/day methylene bisphenyl isocyanate for 5 days survived (Woolrich 1982). Undiluted methylene bisphenyl isocyanate applied to the skin of rabbits caused mild irritation that cleared within 5 days, and the application of 1 mg of a 10-percent methylene bisphenyl isocyanate solution to rabbit eyes produced mild inflammation and tearing but no permanent injury (Woolrich 1982). Administered to guinea pigs by intradermal injection, to mice by skin painting, or to dogs by intrabronchial instillation, methylene bisphenyl isocyanate elicits an immune sensitization response that is characterized by the development of antibodies specific to methylene bisphenyl isocyanate-serum albumin conjugate (Chang and Karol 1984; Thorne, Yeske, and Karol 1987).

Acute exposure to a concentration of 0.001 to 0.026 ppm methylene bisphenyl isocyanate caused eye, nose, and throat irritation in 34 of 35 exposed individuals and shortness of breath in 17 members of the exposed group (NIOSH 1978). Humans exposed to the vapors of methylene bisphenyl isocyanate may develop pulmonary irritation (characterized by increased lung and respiratory tract secretions, coughing, pain on breathing, and, if overexposure is severe, a reduction in breathing rate) and/or allergic sensitization (characterized by cold- or hayfever-like symptoms and asthma, hives, atopic eczema, and allergic reactions) (Woolrich 1982). Estimates of the percentage of occupationally exposed individuals likely to develop sensitization reactions to this substance range from 2 to 20 percent. Workers sensitized to methylene bisphenyl isocyanate are also likely to develop cross-reactivities to other isocyanates, such as toluene diisocyanate or hexamethylene diisocyanate (Woolrich 1982). For example, 12 of 25 individuals sensitized to toluene diisocyanate showed marked declines in lung function when exposed to a methylene bisphenyl isocyanate concentration of 0.009 to 0.02 ppm for 15 to 60 minutes (Innocenti et al. 1988). Exposure to airborne methylene bisphenyl isocyanate concentrations below 0.1 ppm may cause sensitization in susceptible workers, while exposure to concentrations between 0.1 and 1.0 ppm may cause respiratory tract and mucous membrane irritation in all exposed individuals (Woolrich 1982). Workers exposed to both methylene bisphenyl isocyanate and toluene diisocyanate for at least 1 year at concentrations that occasionally exceeded 0.8 ppm had decrements in pulmonary function and an increased frequency of bronchitis compared with non-exposed workers; however, workers exposed to average methylene bisphenyl isocyanate and toluene diisocyanate concentrations of less than 0.003 ppm for 5 years showed no decrement in pulmonary function (Banks, Butcher, and Salvaggio 1986; Musk et al. 1982). Workers who have become sensitized to methylene bisphenyl isocyanate develop either an acute asthma-like response or sensitization pneumonitis when subsequently exposed to this substance; the asthmatic attacks may occur immediately or have a delayed onset, and the pneumonitis is characterized by fever, an increased white blood cell count, an increase in the number of neutrophils observed in bronchoalveolar lavage fluid, and an increase in breathing resistance (Genium MSDS 1980, No. 1105). Workers whose skin has been exposed repeatedly to methylene bisphenyl isocyanate may develop contact eczema (Rothe 1976, in ACGIH 1986, p. 390(86)).

Based on this evidence in humans and animals, OSHA preliminarily concludes that methylene bisphenyl isocyanate causes irritation and both skin and pulmonary sensitization. OSHA preliminarily finds that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these effects. OSHA believes that establishing a PEL of 0.02 ppm (0.2 mg/m^3) as a ceiling limit is necessary to substantially reduce these risks of material health impairments. Promulgation of this limit will also make the PEL for methylene bisphenyl isocyanate consistent across all regulated sectors.

METHYL ETHYL KETONE PEROXIDE
CAS: 1338–23–4; Chemical Formula: $C_8H_{18}O_6$
H.S. No. 1257

OSHA has no limit in construction, maritime, or agriculture for methyl ethyl ketone peroxide (MEKP). The ACGIH has a TLV®-ceiling of 0.2 ppm for this substance. There is no NIOSH REL. OSHA is proposing a ceiling limit of 0.7 ppm for this substance in construction, maritime, and agriculture. NIOSH concurs (Ex. 8–47, Table N1) with this limit, which was recently established for this substance in general industry.

MEKP is sold commercially as a colorless liquid mixture consisting of approximately 60 percent MEKP and 40 percent diluent; the diluent is added to reduce MEKP's sensitivity to shock. Methyl ethyl ketone peroxide is used in the manufacture of acrylic resins and as a hardening agent for fiber glass reinforced plastics (Hawley's 1987, p. 769). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The health effects data for MEKP in animals rely primarily on a study conducted in 1958 by Floyd and Stokinger (Ex. 1–783). In a series of experiments conducted in rats, mice, and rabbits to determine the toxicity of MEKP by various routes of exposure, these investigators found that inhalation exposure for 4 hours to a concentration of 200 ppm was fatal to 50 percent of rats, and a 4-hour exposure to 170 ppm was fatal to 50 percent of mice. Inhalation of MEKP vapors produced petechial and gross hemorrhages of the lungs in rats after four-hour exposures; liver and kidney damage was also observed (Floyd and Stokinger 1958/Ex. 1–783). Two drops of a 40-percent solution of MEKP in dimethyl phthalate caused severe damage when instilled in rabbits' eyes, but at 3 percent, a moderate, transient reaction was produced. The direct application of MEKP to closely shaved rabbit skin caused no immediate discomfort but did cause a severe delayed reaction, consisting of erythema, edema, and vesiculation within 2 or 3 days; of the four organic peroxides tested (di-t-butyl peroxide, t-butyl hydroperoxide, cumene hydroperoxide, and methyl ethyl ketone peroxide), MEKP exhibited the greatest toxicity. The maximal nonirritating strength of MEKP applied dermally was 0.6 percent. In addition, rats died or showed marked evidence of cumulative systemic effects after either oral or intraperitoneal administration of MEKP at 20 percent of the LD_{50} level for 3 days/week for 7 weeks (Floyd and Stokinger 1958/Ex. 1–783). The study of MEKP's toxicity performed by Floyd and Stokinger (1958/Ex. 1–783) was a thorough and comprehensive bioassay involving three species (mice, rats, and rabbits) and five routes of exposure (inhalation, intraperitoneal, oral, dermal, and eye contact). This study demonstrated that MEKP was 20- to 50-fold more acutely toxic than di-t-butyl peroxide by all routes tested. The

consequences of exposure to this substance ranged from skin and eye irritation to gross hemorrhage of the lung and liver and kidney damage; OSHA notes that these effects were observed even after short-term exposures. The Floyd and Stokinger study (1958/Ex. 1–783) demonstrated that MEKP is significantly more toxic than benzoyl peroxide (PEL of 5 mg/m³) and resembles hydrogen peroxide (PEL of 1.4 mg/m³) in terms of its potential to cause irritation on an acute basis. A study by Moskowitz and Grabois (1950, as cited in ACGIH 1986/Ex. 1–3, p. 54) showed that exposure to 12.2 mg/m³ benzoyl peroxide caused "pronounced irritation of the nose and throat" in workers; because MEKP is significantly more irritant than benzoyl peroxide, MEKP concentrations considerably below the 12 mg/m³ level can be expected to cause irritation as well.

OSHA has preliminarily concluded that the available data support the proposed ceiling PEL for MEKP 0.7 ppm. Because MEKP is more irritating than benzoyl peroxide and irritation can result from even very brief exposures to excessive concentrations of MEKP, OSHA also preliminarily concludes that a ceiling limit for MEKP is necessary and appropriate. Therefore, to reduce the significant risk of irritation to workers in construction, maritime, and agriculture who are exposed to MEKP, OSHA is proposing a 0.7 ppm ceiling PEL for MEKP in these sectors. Promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

METHYL FORMATE

CAS: 107–31–3; Chemical Formula: $HCOOCH_3$

H.S. No. 1258

OSHA has a limit of 100 ppm TWA as an 8-hour for methyl formate in construction and maritime. There is no limit in agriculture. The ACGIH also has an 8-hour TLV®–TWA of 100 ppm, with a TLV®–STEL of 150 ppm. OSHA is retaining the 8-hour TWA of 100 ppm for methyl formate and proposing to add a STEL of 150 ppm for this substance in construction and maritime; OSHA is also proposing both limits in agriculture. NIOSH concurs (Ex. 8–47, Table N1) that these limits, which were recently established for methyl formate in general industry, are appropriate.

Methyl formate is a flammable, colorless liquid with an agreeable odor. Methyl formate is used as a fumigant and larvicide. It is also used in organic synthesis and as a cellulose acetate solvent (ACGIH 1986, p. 397; Hawley's 1987, p. 770). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Methyl formate causes nose and eye irritation, vomiting, incoordination, narcosis, and death in guinea pigs exposed to high concentrations (Schrenk, Yant, Chornyak, and Patty 1936/Ex. 1–756). Exposure to a 5-percent concentration of methyl formate was fatal in 20 to 30 minutes, a 1.5- to 2.5-percent concentration was dangerous in 30 to 60 minutes, and a 0.5 percent concentration (5000 ppm) was considered the maximum concentration tolerable for a 60-minute period without serious consequences. Lehmann and Flury (1943b/Ex. 1–963) observed that inhalation of a 1.02 percent concentration of methyl formate for two to three hours caused pulmonary edema and death in cats; a concentration of 1600 ppm resulted in lung inflammation after one hour. Guinea pigs died when exposed by inhalation to a 2.5 percent concentration of methyl formate (Lehmann and Flury 1943b/Ex. 1–963).

In studies of methyl formate exposure in humans, von Oettingen (1959/Ex. 1–499) reported that workers exposed to unspecified concentrations showed temporary blindness, narcosis, mucous membrane irritation, and dyspnea. Fairhall (1957c/ Ex. 1–1107) has reported that methyl formate is more irritating than either methyl or ethyl acetate.

OSHA is retaining the 8-hour PEL of 100 ppm TWA and proposing to add a STEL of 150 ppm for methyl formate in construction and maritime; the Agency is also proposing to extend both limits to agriculture. OSHA believes that these PELs will prevent the significant risks of irritation, narcotic effects, and pulmonary damage, all of which constitute material health impairments, confronting workers in these sectors who are exposed to methyl formate. The Agency preliminarily concludes that these limits will substantially reduce these significant risks. In addition, promulgation of these limits will make OSHA's PELs for methyl formate consistent across all regulated sectors.

METHYL IODIDE

CAS: 74–88–4; Chemical Formula: CH_3I

H.S. No. 1259

OSHA has an 8-hour TWA limit of 5 ppm, and a skin notation, for methyl iodide in construction and maritime operations. The ACGIH has a TLV®–TWA of 2 ppm, with a skin notation, for methyl iodide and classifies it as a suspected human carcinogen (A2). NIOSH recommends reducing exposure to this substance to the lowest feasible limit and also considers this chemical a potential human carcinogen. OSHA is proposing an 8-hour TWA PEL of 2 ppm, with a skin notation, for methyl iodide in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Methyl iodide is a colorless, sweet-smelling liquid that turns yellow, red, or brown when exposed to light and moisture. Methyl iodide is used in microscopy, organic synthesis, and in testing for pyridine (ACGIH 1986, p. 399(86); Hawley's 1987, p. 772).

Methyl iodide has an LD_{50} in rats of 150 to 200 mg/kg; liver damage was evident after these lethal exposures (Kutob and Plaa 1962/Ex. 1–61). A 15-minute exposure to a concentration of 3800 ppm was fatal to rats (Chambers et al. 1950, as cited in ACGIH 1986/Ex. 1–3, p. 399), and Bachem (1927/Ex. 1–1013) has reported that methyl iodide is six times as toxic in mice as methyl bromide. Inhalation studies have shown eye irritation and depressed body weight in rats as a result of 14-week exposures to 30 and 60 ppm (Blank, Nair, Roloff, and Ribelin 1984/Ex. 1–619). The same authors observed fatalities in rats within four weeks of exposure to 143 ppm; 10 ppm was reported to be a no-effect level.

In industry, fatalities have occurred in chemical workers exposed to methyl iodide poisoning (Garland and Camps 1945/Ex. 1–1190; Appel, Galen, O'Brien, and Schoenfeldt 1975/Ex. 1–1076). However, the exposure levels associated with these fatal overexposures are not known (ACGIH 1986/Ex. 1–3, p. 399).

In tests of carcinogenicity, methyl iodide produced local sarcomas in rats injected subcutaneously and lung tumors in mice given intraperitoneal injections (Druckrey, Kruse, Preussman et al. 1970/Ex. 1–246; Poirier, Stoner, and Shimkin 1975/Ex. 1–686). These carcinogenic effects occurred at a dosage approximately equivalent to a daily 8-hour exposure to 20 or 25 ppm for an adult human (ACGIH 1986/Ex. 1–3, p. 399).

OSHA is proposing an 8-hour TWA limit of 2 ppm, and a skin notation, for methyl iodide in construction, maritime, and agriculture. The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant risk of irritation and liver and kidney damage, which are material impairments of health associated with occupational exposure to methyl iodide. The skin notation is needed to prevent dermal absorption of toxic amounts of methyl iodide. Promulgation of this limit will also make OSHA's PEL for this substance consistent across all regulated sectors.

METHYL ISOAMYL KETONE
CAS: 110–12–3; Chemical Formula: $CH_3COCH(C_4H_9)_2$
H.S. No. 1260

OSHA has a TWA-PEL of 100 ppm for methyl isoamyl ketone (MIAK) in construction and maritime. There is no limit in agriculture. The ACGIH has established an 8-hour TLV®-TWA of 50 ppm for MIAK, and this is also the REL recommended by NIOSH. OSHA is proposing a PEL of 50 ppm as an 8-hour TWA for this substance in construction, maritime, and agriculture, and NIOSH concurs with this limit (Ex. 8–47, Table N1). This is the PEL recently established for this substance in general industry.

Methyl isoamyl ketone is a colorless, clear liquid with a pleasant odor. Methyl isoamyl ketone is used as a solvent for cellulose esters, acrylics, and copolymers (ACGIH 1986, p. 400).

The oral LD_{50} for methyl isoamyl ketone in rats is 3.2 g/kg (RTECS 1991). The lowest lethal concentration in rats is 4000 ppm for 4 hours (RTECS 1991). Instilled into the eyes of rabbits, MIAK caused corneal damage rated 2 on an ascending severity of 1 to 10 (Grant 1974, p. 1166). Experimental animals exposed to high (not further specified) concentrations of ketones including MIAK show emphysematous changes to the lungs and damage to the liver, kidneys, and brain at autopsy (HSDB 1991). The dermal LD_{50} in rabbits is 10 g/kg (RTECS 1991).

No data relating exposure levels to specific effects in humans have been reported. However, the ACGIH (1986/Ex. 1–3, p. 400) believes that MIAK is likely to be more irritating and a more potent narcotic than is the case for methyl isobutyl ketone.

The NIOSH criteria document on the ketones (1978f) states that "because methyl isoamyl ketone contains one more carbon atom than does methyl isobutyl ketone, methyl [isoamyl] ketone might produce irritation and narcosis at concentrations at least as low as those at which methyl isobutyl ketone produces these effects" (NIOSH 1978f).

OSHA is proposing an 8-hour TWA limit of 50 ppm for methyl isoamyl ketone in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risk of narcotic and irritant effects, which constitute material health impairments that are associated with occupational exposure to MIAK. Promulgation of this limit will also make OSHA's PEL for this substance consistent across all regulated sectors.

METHYL ISOPROPYL KETONE
CAS: 563–80–4; Chemical Formula: $(CH_3)_2CHCOCH_3$
H.S. No. 1262

OSHA has no limit for methyl isopropyl ketone (MIPK) in construction, maritime, or agriculture. The ACGIH has a TLV®-TWA of 200 ppm. NIOSH has no REL. In construction, maritime, and agriculture, OSHA is proposing a PEL of 200 ppm as an 8-hour TWA; NIOSH concurs (Ex. 8–47, Table N1) with this limit. This is the PEL recently established for MIPK in general industry.

Methyl isopropyl ketone (also called 3-methyl-2-butanone) is a colorless, flammable liquid. This substance is used as a solvent for nitrocellulose lacquers (ACGIH 1986, p. 405).

Animal studies have shown that MIPK has an acute toxicity somewhat greater than that of diethyl ketone and somewhat less than that of di-n-propyl ketone or methyl-n-propyl ketone (ACGIH 1986/Ex. 1–3, p. 405). The oral LD_{50} in rats is 149 mg/kg, and the dermal LD_{50} in rabbits is 6350 mg/kg (RTECS 1991). Rats exposed for four hours at a concentration of 5700 ppm died (NIOSH 1977i, Ex. 1–1182). In contact with the skin of rabbits, this substance caused a moderate degree of irritation (RTECS 1991). Instilled into rabbit eyes, it caused mild irritation (RTECS 1991). Humans exposed to unspecified concentrations of MIPK develop slight eye irritation and skin irritation that ranges from mild to moderate (Clayton and Clayton 1981, p. 4738).

OSHA is proposing a PEL of 200 ppm (8-hour TWA) for methyl isopropyl ketone in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risk of irritation, a material health impairment that is associated with exposure to this ketone at the uncontrolled levels possible in the absence of a PEL. Promulgation of this limit will also make OSHA's PEL for this substance consistent across all regulated sectors.

METHYL PARATHION
CAS: 298–00–0; Chemical Formula: $C_8H_{10}NO_5PS$
H.S. No. 1265

OSHA has no limit for methyl parathion in construction, maritime, or agriculture. The ACGIH has a TLV®-TWA of 0.2 mg/m³, with a skin notation, for methyl parathion, and NIOSH also recommends a TWA of 0.2 mg/m³. In construction, maritime, and agriculture, the proposed PEL is an 8-hour TWA limit of 0.2 mg/m³, with a skin notation; NIOSH concurs (Ex. 8–47, Table N1) with this limit, which is identical to the PEL recently established for this substance in general industry.

Methyl parathion is a tan to brown liquid with a pungent odor like that of garlic. This substance is used as a nonsystemic insecticide, especially for Cotton (Hawley's 1987, p. 777; Hayes 1982, p. 339). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Methyl parathion is an acetylcholinesterase inhibitor, and excessive exposure can cause sweating, salivation, diarrhea, bradycardia, bronchoconstriction, muscle fasciculations, and coma. Methyl parathion's acute oral LD_{50} in male rats is almost identical to that of parathion (i.e., 10 to 25 mg/kg); in female rats, the LD_{50} is 24 mg/kg, or approximately one-sixth that of parathion. By the dermal route, methyl parathion is much less toxic than parathion, with an LD_{50} of 67 mg/kg in rats of both sexes (Hayes 1963/Ex. 1–882). Erythrocyte cholinesterase activity was inhibited in dogs fed methyl parathion for 12 weeks at a rate corresponding to approximately 24 mg/day; inhibition of both plasma and erythrocyte cholinesterase activity occurred at doses of 70 mg/day, without accompanying illness (Williams, Fuyat, and Fitzhugh 1959/Ex. 1–810). Dogs fed 6 mg/day methyl parathion for 12 weeks showed no effects from such exposures (Williams, Fuyat, and Fitzhugh 1959/Ex. 1–810). Lifetime feeding studies of rats and mice fed diets containing methyl parathion concentrations of up to 40 ppm and up to 125 ppm, respectively, produced no evidence of cancer (NCI 1979a/Ex. 1–1116).

Plasma and erythrocyte cholinesterase levels did not differ by more than 20 percent in subjects exposed at 7, 7.5, 8, or 9 mg/man/day, compared with controls (Moeller and Rider 1963/Ex. 1–565). Tiess, Wegener, and Tamme (1982/Ex. 1–774) have reported a case of protracted methyl parathion poisoning resulting from both percutaneous and inhalation exposures; Dille and Smith (1964/Ex. 1–549) attribute the long-term neuropsychiatric illness of two pilots to exposure to methyl parathion and other cholinesterase-inhibiting agents. Chronic exposure to small doses of methyl parathion have not caused chromosomal effects (de Cassia Stocco, Becak, Gaeta, and Rabello-Gay 1982/Ex. 1–540).

In construction, maritime, and agriculture, OSHA is proposing a limit of 0.2 mg/m³ TWA for methyl parathion, with a skin notation. The Agency

preliminarily concludes that this limit will protect workers in these sectors against the significant risk of acetylcholinesterase inhibition, which constitutes a material impairment of health that is associated with workplace exposures to this substance. The skin notation will protect workers from the significant risk of systemic toxicity associated with percutaneous absorption of this substance. In addition, promulgation of this limit will make OSHA's PEL for methyl parathion consistent across all regulated sectors.

METHYLCYCLOHEXANE

CAS: 108–87–2; Chemical Formula: C_7H_{14}

H.S. 1268

OSHA has an 8-hour TWA limit of 500 ppm for methylcyclohexane in construction and maritime, and the ACGIH has a TLV®–TWA of 400 ppm for this substance. There is no OSHA limit in agriculture. NIOSH has no REL, but concurs (Ex. 8–47, Table N1) with the limit being proposed. The proposed PEL for this substance in construction, maritime, and agriculture is 400 ppm. This is the limit recently established for methylcyclohexane in general industry.

Methylcyclohexane is a colorless liquid that is used as a solvent for cellulose esters and in organic synthesis (ACGIH 1986, p. 384).

Lehmann and Flury (1943e, as cited in ACGIH 1986/Ex. 1–3, p. 384) indicate that the acute toxicity of methylcyclohexane is greater than that of heptane but less than that of octane. Lazarew (1929/Ex. 1–1059) found that a 2-hour exposure to a concentration of 7500 to 10,000 ppm caused prostration in mice, and exposure to 10,000 to 12,500 ppm caused death. Treon, Crutchfield, and Kitzmiller (1943b/Ex. 1–394) reported that exposure to 1200 ppm had no effect in rabbits and that prolonged exposures to 370 ppm had no effect in monkeys. Methylcyclohexane's histologic effects in animals resemble those of cyclohexane; the liver and kidney are the sites affected (ACGIH 1986/Ex. 1–3, p. 384).

OSHA is proposing to establish an 8-hour TWA limit of 400 ppm for methylcyclohexane in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risk of irritation, a material health impairment that is associated with exposure to methylcyclohexane. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

2-METHYLCYCLOPENTADIENYL MANGANESE TRICARBONYL (as Mn)

CAS: 12108–13–3; Chemical Formula: $(CH_3)C_5H_5-Mn(CO)_3$

H.S. No. 1271

OSHA has no limit for 2-methylcyclopentadienyl manganese tricarbonyl (MCT) in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 0.2 mg/m³, measured as manganese, with a skin notation. There is no NIOSH REL. The proposed PEL for this substance in construction, maritime, and agriculture is 0.2 mg/m³ as an 8-hour TWA, with a skin notation. NIOSH concurs (Ex. 8–47, Table N1) with the proposed limit, which is identical to the PEL recently established for this substance in general industry.

MCT, also called Cl-2, is a dark orange liquid with a faintly pleasant odor; it is a complex organic compound containing about 25 percent manganese by weight. MCT is used as a gasoline additive.

MCT is highly toxic in its concentrated form, causing adverse effects primarily on the central nervous system. It is somewhat irritating to the eyes, but skin contact does not produce irritation or sensitization; however, MCT is readily absorbed through the skin (ACGIH 1986/Ex. 1–3, p. 387). Animal studies indicate that MCT has a toxicity similar to that of tetraethyl lead and is highly toxic by all routes of exposure (U.S. Navy Smoke Abatement Additive, as cited in ACGIH 1986/Ex. 1–3, p. 387).

The single-dose oral LD_{50} for rats is 23 or 39 mg/kg, depending on sex. The skin LD_{50} for rabbits is 1692±145 mg/kg, and the 1-hour inhalation LC_{50} for rats is about 350 mg/m³ (the Ethyl Corporation, as cited in ACGIH 1986/Ex. 1–3, p. 387). Toxic exposures by all routes produce rapidly appearing symptoms of mild excitement, hyperactivity, tremors, severe clonic spasms, weakness, respiratory distress, and occasional clonic convulsions, followed by terminal coma (U.S. Navy Smoke Abatement Additive, as cited in ACGIH 1986/Ex. 1–3, p. 387).

Acute exposure causes damage to the liver, kidneys, and cerebral cortex, as well as changes in lung tissue (ACGIH 1986/Ex. 1–3, p. 387). Browning (1966/Ex. 1–1018) observed chronic bronchitis, peribronchitis, interstitial pneumonia, and lung abscesses in animals that subsequently died from long-term inhalation exposure to MCT; exposure to MCT concentrations of approximately 12 mg/m³ for 100 days produced no deviation in weight gain patterns and no gross or microscopic changes in two dogs (Browning 1966/Ex. 1–1018). The liver and kidneys are the principal target organs associated with acute overexposures; at autopsy, the lungs of overexposed animals were hemorrhagic (Browning 1966/Ex. 1–1018).

In humans, skin contact with MCT should be entirely avoided. A 5- to 15-ml spill on one worker's hand and wrist was reported to have caused "thick tongue," nausea, giddiness, and headache within 3 to 5 minutes (U.S. Navy Smoke Abatement Additive, as cited in ACGIH 1986/Ex. 1–3, p. 387).

OSHA is proposing a PEL of 0.2 mg/m³ TWA, measured as manganese, and a skin notation for 2-methylcyclopentadienyl manganese tricarbonyl in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risk of CNS effects and systemic damage, which constitute material health impairments that are associated with occupational exposure to MCT. A skin notation is necessary because of MCT's demonstrated ability to penetrate human skin rapidly and to cause systemic effects. Promulgation of this limit will also make OSHA's PEL for this substance consistent across all regulated sectors.

MONOCROTOPHOS (AZODRIN)

CAS: 6923–22–4; Chemical Formula: $C_7H_{14}NO_5P$

H.S. No. 1279

OSHA has no limit for monocrotophos (also called Azodrin) in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 0.25 mg/m³ for this substance. There is no NIOSH REL. The proposed PEL is 0.25 mg/m³ as an 8-hour TWA for monocrotophos in construction, maritime, and agriculture. NIOSH concurs (Ex. 8–47, Table N1) with this limit, which was also recently established for this substance in general industry.

Monocrotophos is a reddish brown liquid with a mild ester odor. It is used as a systemic insecticide to control certain insects on cotton plants (ACGIH 1986, p. 416; Hawley's 1987, p. 110). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Monocrotophos is a highly toxic, direct acting cholinesterase inhibitor that penetrates the intact skin (ACGIH 1986/Ex. 1–3, p. 416). The acute oral LD_{50} values in rats and mice range from 5.7 to 17 mg/kg in a water formulation (Brown et al. 1970, Shellenberger and Newell, both as cited in ACGIH 1986/Ex. 1–3, p. 416) and from 10 to 23 mg/kg in an oil formulation (Shellenberger and Newell,

as cited in ACGIH 1986/Ex. 1-3, p. 416). These authors also report a percutaneous LD_{50} in the rabbit that ranges from 112 to 709 mg/kg, depending on the vehicle used. A two-year dietary study of rats ingesting 0, 1, 10, or 100 ppm monocrotophos revealed that both sexes in the 100 ppm group failed to gain as much weight as the controls, but autopsy showed no significant findings; plasma, erythrocyte, and brain cholinesterase decreased at the two highest dose levels but were unaffected at 1 ppm (Johnston 1966–67, as cited in ACGIH 1986/Ex. 1–3, p. 416). Another two-year feeding study in dogs administered doses of up to 16 ppm monocrotophos revealed no adverse effects at levels of 0.16 and 1.6 ppm, but serious cholinesterase reduction was observed at the 16-ppm level (Johnston 1966–67, as cited in ACGIH 1986/Ex. 1–3, p. 416). Metabolism studies in rats and goats indicate that monocrotophos is excreted rapidly in the rat and does not accumulate in the body (Bull and Lindquist 1966/Ex 1–719); goats given labeled monocrotophos by mouth showed only traces of the material in their milk (Menzer and Casida 1965/Ex. 1–986; Potter, as cited in ACGIH 1986/Ex. 1–3, p. 416). Inhalation exposure of rats to an unknown concentration of 75 percent monocrotophos in air for one hour was not lethal; a four-hour exposure to an unknown concentration of the aerosol (0.4 and 0.75 percent) was fatal to two out of six (0.4 percent aerosol) and five out of eight rats (0.75 percent aerosol). Head-only exposure to the 0.4-percent aerosol resulted in the death of one of eight animals (Wilson, as cited in ACGIH 1986/Ex. 1–3, p. 416).

Intravenous injection of radiolabeled monocrotophos in human volunteers showed maximum excretion at 4 to 8 hours, with 67±5 percent of the material in the urine; absorption of 14±7 percent occurred when the radiolabeled material was applied to the forearm; 33±9 percent of the applied dose was absorbed when it was covered with a vapor-proof film for 72 hours (Maibach 1970, as cited in ACGIH 1986/Ex. 1–3, p. 416). Although gauze patches attached to the clothing and skin of field workers attested to the presence of monocrotophos, no cholinesterase inhibition was observed in post-exposure examinations at 3 hours and at three and seven days (Maibach, as cited in ACGIH 1986/Ex. 1–3, p. 416).

OSHA is proposing a PEL of 0.25 mg/m³ (8-hour TWA) for monocrotophos in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risk of cholinesterase inhibition, a material impairment of health that is associated with exposure to this substance in the workplace. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

MORPHOLINE

CAS: 110-91-8; Chemical Formula: C_4H_9NO

H.S. No. 1281

In construction and maritime, OSHA has a limit of 20 ppm, with a skin notation, for morpholine. There is no limit in agriculture. The ACGIH has a 20-ppm TLV®–TWA limit and a TLV®–STEL of 30 ppm, as well as a skin notation. There is no NIOSH REL. OSHA is retaining the 8-hour TWA PEL of 20 ppm, and the skin notation, in construction and maritime, is proposing to add a STEL of 30 ppm in these two sectors, and is also proposing both limits and the skin notation in agriculture. NIOSH concurs (Ex. 8–47, Table N1) that these limits are appropriate. These are the limits recently established for morpholine in general industry.

Morpholine is a colorless liquid with an amine-like odor. Morpholine is used as a rubber accelerator, a solvent, an additive to boiler water, a corrosion inhibitor, a preservative in book paper, and an organic intermediate (ACGIH 1986, p. 417(89); Hawley's 1987, p. 799).

Exposure to morpholine produces nasal and bronchial irritation and liver and kidney impairment in animals (Shea 1939/Ex. 1–758); the substance readily penetrates the skin and is highly irritating to the eyes (Jefferson Chemical Company, Inc. 1961, as cited in ACGIH 1986/Ex. 1–3, p. 417). The single oral LD_{50} in rats is 1.05 g/kg (range: 0.95 to 1.16 g/kg), and the single skin LD_{50} for 24-hour contact is 0.5 ml/kg (Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1–440). Neither a one-hour exposure to concentrated vapor nor an 8-hour exposure to 8000 ppm was fatal in rats (Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1–440). Rats were exposed for 8 hours daily to a concentration of 18,000 ppm for a total of 5 days; after the first day, all animals showed severely reddened thoracic walls, and one fatality (from kidney and liver congestion) occurred. A similar fatality occurred on the third day; on day 4, a third rat died, and postmortem examination revealed degeneration of the epithelial lining of the kidney tubules. Three additional deaths occurred after the exposures had ended; autopsy revealed thickened alveoli, emphysema, and liver and kidney effects (Shea 1939/Ex. 1–758).

Reporting on his own reactions to morpholine exposure at a concentration of 12,000 ppm, Shea (1939/Ex. 1–758) complained of nose irritation (after 1 minute) and coughing (after 90 seconds); in addition, when he transferred morpholine by pipette, he experienced sore throat and mucosal irritation. All symptoms disappeared after the experiment stopped (Shea 1939/Ex. 1–758). Skin contact poses a moderately high degree of hazard, which diminishes as the product is diluted with water to less than 25 percent (Jefferson Chemical Company, Inc. 1961, as cited in ACGIH 1986/Ex. 1–3, p. 417). Respiratory irritation but no chronic effects have been reported as a result of industrial exposure (Patty 1963e/Ex. 1–858). In comparison with ammonia, morpholine has a greater potential for systemic toxicity (ACGIH 1986/Ex. 1–3, p. 417).

OSHA is retaining the 8-hour TWA limit of 20 ppm TWA and the skin notation for morpholine and is proposing to add a 15-minute STEL of 30 ppm in construction and maritime; OSHA is also proposing both limits and the skin notation in agriculture. The Agency preliminarily concludes that these limits will work together to protect workers in these sectors against the significant risk of eye and respiratory tract irritation, which are material impairments of health that are associated with occupational exposures to this substance. The skin notation for morpholine is necessary because of this substance's ability to be absorbed through the skin in toxic amounts. Promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

NITRIC ACID

CAS: 7697-37-2; Chemical Formula: HNO_3

H.S. No. 1286

OSHA has an 8-hour TWA limit of 2 ppm for nitric acid in construction and maritime. There is no limit in agriculture. The ACGIH has the same TLV®–TWA and a 15-minute TLV®–STEL of 4 ppm, and NIOSH recommends a TWA limit of 2 ppm. OSHA's proposal retains the 8-hour TWA PEL of 2 ppm and adds a STEL of 4 ppm for nitric acid in construction and maritime; the Agency also proposes both limits in agriculture. NIOSH concurs (Ex. 8–47, Table N1) with these limits, which are identical to those recently established for this substance in general industry.

Nitric acid is a fuming colorless or yellowish liquid. Nitric acid is used to destroy residues of organic matter, to dissolve noble metals, for etching and cleaning metals, and in the manufacture of ammonium nitrate for fertilizer (ACGIH 1986, p. 428; Hawley's 1987, p. 824).

Rats receiving a single exposure to nitric acid mist at a concentration of 63 mg/m³ exhibited no apparent adverse effects (Diggle and Gage 1954/Ex. 1–729). Chronic exposure to airborne nitric acid vapor or mist at unspecified levels was reported to cause chronic bronchitis, pneumonitis (Fairhall 1957i, as cited in ACGIH 1986/Ex. 1–3, p. 428), and tooth erosion (Lynch and Bell 1947/Ex. 1–793). Nitric acid's irritant potential is considered similar to that of other strong acids; it typically exists in conjunction with nitrogen dioxide, which is regarded as being more hazardous (ACGIH 1986/Ex.1–3, p. 428).

OSHA is retaining the 8-hour TWA PEL of 2 ppm and proposing to add a STEL of 4 ppm for nitric acid in construction and maritime; the Agency is also proposing both limits in agriculture. The Agency preliminarily concludes that these limits will protect workers in these sectors against the significant risk of irritation, chronic pulmonary disease, and dental erosion, which constitute material impairments of health. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

p-NITROANILINE

CAS: 100–01–6; Chemical Formula: $NO_2C_6H_4NH_2$

H.S. No. 1287

OSHA has a limit of 1 ppm TWA (6 mg/m³) for p-nitroaniline (PNA), and a skin notation, in construction and maritime. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 3 mg/m³, with a skin notation. There is no NIOSH REL. OSHA is proposing an 8-hour TWA of 3 mg/m³ and a skin notation for p-nitroaniline in construction, maritime, and agriculture. NIOSH concurs (Ex. 8–47, Table N1) that this limit is appropriate for this substance. This is the limit recently established for PNA in general industry.

para-Nitroaniline usually exists in the form of yellow needles. p-Nitroaniline is used in medicinals for poultry, as an intermediate for dyes and antioxidants, and as a gasoline gum inhibitor and corrosion inhibitor (ACGIH 1986, p. 430; Hawley's 1987, p. 826).

Exposure to p-nitroaniline causes convulsions in experimental animals (RTECS 1991). The oral LD_{50} in rats is 750 mg/kg (RTECS 1991). Dogs administered a single intravenous injection of 15 mg/kg p-nitroaniline developed methemoglobinemia (Clayton and Clayton 1981, p. 2416). Administered to mice on days 6 to 13 of pregnancy at a dose of 1200 mg/kg/day, p-nitroaniline caused maternal death or systemic toxicity (reduced weight gain and a reduction in the number of litters and in pup survival (Hardin et al. 1987, in Teratog. Carcinog. Mutag. 7:29–48).

p-Nitroaniline is readily absorbed through the skin and is a strong methemoglobin-forming agent; prolonged exposure can cause liver damage (ACGIH 1986/Ex. 1–3, p. 430). Anderson (1946/Ex. 1–1049) reported several cases of PNA-poisoning among shipboard workers assigned to clean up a p-nitroaniline spill; one man with a history of liver disease became jaundiced and died, and the other exposed workers became cyanotic and complained of headache, sleepiness, weakness, and respiratory distress (Anderson 1946/Ex. 1–1049). It has also been reported that children who ingested p-nitroaniline that was contained in wax crayons subsequently became ill (Rieders and Brieger 1953/Ex. 1–798).

Several investigators (Anderson 1946/Ex. 1–1049; Gupta 1953, Fairhall 1957j, both as cited in ACGIH 1986/Ex. 1–3, p. 430; Linch 1974/Ex. 1–747) have concluded that the nitroanilines are more hazardous than aniline, and, on this basis, OSHA established a PEL for PNA in general industry that is lower than the PEL for aniline.

OSHA is proposing a PEL of 3 mg/m³ as an 8-hour TWA for p-nitroaniline, and a skin notation, in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risk of methemoglobinemia and liver damage, both of which constitute material health impairments that are associated with exposure to PNA. A skin notation is necessary because this substance is readily absorbed through the skin in toxic amounts. Promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

NITROMETHANE

CAS: 75–52–5; Chemical Formula: CH_3NO_2

H.S. No. 2120

OSHA's permissible exposure limit for nitromethane in general industry, construction, and maritime is an 8-hour TWA of 100 ppm. There is no limit in agriculture. The ACGIH has a limit for nitromethane of 100 ppm as an 8-hour TLV®–TWA. The ACGIH TLV® for nitromethane was set on the basis of analogy with nitroethane. NIOSH has no REL. OSHA is proposing to establish an 8-hour TWA of 100 ppm for nitromethane in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Nitromethane is a colorless, oily liquid with a mild fruity odor. This substance is used as a solvent for cellulosic compounds, polymers, waxes, and fats; in chemical synthesis; as a rocket fuel; and as a gasoline additive (ACGIH 1986, p. 439; Merck 1983, p. 949).

Nitromethane causes eye and mucous membrane irritation and central nervous system effects in humans; in animals, it causes irritation, central nervous system depression, and kidney and liver damage. In rats and mice, the oral LD_{50}s are 940 mg/kg and 960 mg/kg, respectively, for an unspecified period of time (RTECS 1990). The lowest lethal inhalation dose in monkeys is 1000 ppm for an unspecified time (RTECS 1990). Severe eye irritation occurs in animals (species unspecified) exposed to a 500-ppm concentration of nitromethane (AIHA 1978). Rabbits exposed to a 10,000-ppm concentration of this substance for 6 hours showed signs of weakness, ataxia, muscular incoordination, and convulsions before death; exposure to the same concentration for a period of 3 hours was not fatal to rabbits (Clayton and Clayton 1982, pp. 4153–4155; Machle et al. 1940, in Proctor, Hughes, and Fischman 1988, p. 377). Both liver and kidney damage were seen in acutely poisoned animals at autopsy (Stokinger 1982; Machle et al. 1940, in Proctor, Hughes, and Fischman 1988, p. 377). Eight 6-hour exposures to a 1000-ppm concentration of nitromethane was fatal to an exposed monkey (Stokinger 1982; Machle et al. 1940, in Proctor, Hughes, and Fischman 1988, p. 377). Rats and rabbits exposed to 98 or 745 ppm of nitromethane 5 days/week for 24 weeks showed reductions in weight gain (rats in the high-dose group) and effects on the thyroid (rabbits in both dose groups) (Lewis et al., 1979, J. Environ. Pathol. Toxicol. 2(5):233). At autopsy, rabbits from both treatment groups had signs of pulmonary edema and other pulmonary effects (Lewis et al. 1979).

In humans, nitromethane causes central nervous system depression, and exposure to this substance also causes irritation of the skin and mucous membranes (Gosselin, Smith, and Hodge 1984, p. II–143). The signs and symptoms of overexposure in humans include anorexia, nausea, vomiting, and diarrhea (AIHA 1978).

Based on this evidence in humans and animals, OSHA preliminarily concludes that, in the absence of a limit for nitromethane, workers in agriculture are at significant risk of experiencing its exposure-related effects. OSHA believes that establishing a limit of 100 ppm as an 8-hour TWA will significantly reduce

these risks of material health impairment. Promulgation of this limit will also make OSHA's PEL for nitromethane consistent across all OSHA-regulated sectors.

NITROTOLUENE

CAS: 88–72–2 (o-isomer); 99–08–1 (m-isomer); 99–99–0 (p-isomer); Chemical Formula: $CH_3C_6H_4NO_2$

H.S. No. 1292

OSHA has an 8-hour TWA limit of 5 ppm, with a skin notation, for nitrotoluene in construction and maritime. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 2 ppm, also with a skin notation. NIOSH has no REL. OSHA is proposing a PEL of 2 ppm as an 8-hour TWA, and a skin notation, in construction, maritime, and agriculture, and NIOSH concurred with this limit (Ex. 8–47, Table N1) when it was recently established for nitrotoluene in general industry.

The ortho- and meta-isomers of nitrotoluene are yellow liquids; the para-isomer is also yellow, but exists in crystalline form. All isomers of nitrotoluene are used in organic synthesis in the production of toluidine, tolidine, fuchsia, and various synthetic dyes (ACGIH 1986, p. 444; Hawley's 1987, pp. 833–834).

Nitrotoluene is one of the aromatic nitrogen compounds that may cause methemoglobin formation. The oral LD_{50} in rats is 1072 mg/kg for the m-isomer (RTECS 1991). For the o-isomer, the oral LD_{50} in rats is 891 mg/kg (RTECS 1991). In rats, the oral LD_{50} is 1960 mg/kg (RTECS 1991). Administered to rats orally for 30 days at doses that were 10 to 20 percent of the LD_{50} doses, m-nitrotoluene caused methemoglobinemia (Vasilenko et al. 1972). A mixture of the three isomers administered to rats in their diet increased the number of leukocytes in their blood, as well as their methemoglobin level (Kasochevskaya 1967).

Linch (1974/Ex. 1–747) has studied the nitrotoluene isomers and reported that they have relatively low hemotoxic potential; he considered nitrotoluene comparable to aniline in its toxic effects (Linch 1974/Ex. 1–747). Chronic exposure is believed to cause anemia (Clayton and Clayton 1981, p. 2473).

In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA limit of 2 ppm, and a skin notation, for nitrotoluene. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risk of methemoglobinemia, a material health impairment that is associated with exposure to this substance. The skin notation is necessary because of nitrotoluene's capacity to penetrate the skin in toxic amounts. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

NONANE

CAS: 111–84–2; Chemical Formula: $CH_3(CH_2)_7CH_3$

H.S. No. 1293

OSHA has no limit for nonane in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 200 ppm for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N1) that the proposed limit is appropriate. OSHA is proposing an 8-hour TWA PEL for nonane of 200 ppm in construction, maritime, and agriculture. This is the limit recently established for nonane in general industry.

Nonane is a colorless liquid used in biodegradable detergents, as a distillation chaser, and in organic synthesis (ACGIH 1986, p. 445; Hawley's 1987, p. 836).

The toxicity of nonane is approximately equal to that of VM&P naphtha. Naphtha has a 4-hour inhalation LC_{50} in rats of 3400 ppm, while nonane has an LC_{50} of 3200 ppm (Carpenter, Kinkead, Geary et al. 1975a/Ex. 1–302; Carpenter, Geary, Myers et al. 1978/Ex. 1–301). These investigators found a no-effect level of 590 ppm for rats exposed to nonane 6 hours/day, 5 days/week for a 65-day period; under the same exposure conditions, a no-effect level of 560 ppm was reported for rats exposed to VM&P naphtha (Carpenter, Kinkead, Geary et al. 1975a/Ex. 1–302; Carpenter, Geary, Myers et al. 1978/Ex. 1–301). Earlier studies of octane and heptane have shown much higher LC_{50} values in mice, i.e., 13,500 ppm and 16,000 ppm, respectively, for 30- to 60-minute exposures (Flury and Zernik 1931j/Ex. 1–994). Swann and associates (1974/Ex.1–124) have reported similarly high LD_{50} values in mice for octane and hexane; mice died from respiratory arrest after 3 to 5 minutes of exposure to 16,000 ppm of octane or to 48,000 ppm of hexane (Swann, Kwon, and Hogan 1974/Ex. 1–124).

Based on this evidence, OSHA is proposing an 8-hour TWA limit of 200 ppm for nonane in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risk of narcosis, a material impairment of health that is associated with occupational exposure to nonane. The Agency believes that the proposed PEL will substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

OXALIC ACID

CAS: 144–62–7; Chemical Formula: $H_2C_2O_4$

H.S. No. 1299

In construction and maritime, OSHA currently has a limit of 1 mg/m³ for oxalic acid. There is no limit in agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limits being proposed. The ACGIH has a TLV®–TWA of 1 mg/m³ and a TLV®–STEL of 2 mg/m³. OSHA is retaining the 8-hour TWA limit of 1 mg/m³ for oxalic acid and proposing to add a STEL of 2 mg/m³ in construction and maritime; the Agency is also proposing these limits in agriculture. These are the limits recently established for oxalic acid in general industry.

Anhydrous oxalic acid usually occurs in the form of a white powder; the dihydrate form is a colorless, odorless, crystalline substance. Oxalic acid is used as an automobile radiator cleanser, a purifying agent and chemical intermediate, in leather tanning, in textile bleaching, and in printing and dyeing (Hawley's 1987, p. 862).

Oxalic acid is known to produce severe burns of the eyes, mucous membranes, and skin (Windholz 1983d/Ex. 1–835, p. 991). There have been human fatalities from ingesting as little as 5 grams of oxalic acid. It appears that these deaths were caused by oxalic acid's ability to disturb the calcium-potassium balance in critical tissues (Klauder, Shelanski, and Gabriel 1955/Ex. 1–1057). Solutions of 5- to 10-percent oxalic acid have also been reported to irritate the skin on prolonged exposure.

Because of oxalic acid's severe acute toxicity, OSHA is retaining the 8-hour TWA limit of 1 mg/m³ PEL and proposing to add a STEL of 2 mg/m³ in construction and maritime; the Agency is also proposing both limits in agriculture. OSHA preliminarily concludes that both of these limits are required to protect exposed workers in these sectors from the significant risk of severe eye and skin burns and respiratory tract irritation, which are material health impairments associated with occupational exposures to this substance. OSHA believes that the proposed PELs are necessary to substantially reduce these risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

PERCHLORYL FLUORIDE

CAS: 7616–94–6; Chemical Formula: ClO_3F

H.S. No. 1309

In construction and maritime, OSHA's 8-hour TWA limit for perchloryl fluoride is 3 ppm. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 3 ppm and a TLV®–STEL of 6 ppm for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the proposed limits. OSHA is retaining the 8-hour TWA PEL of 3 ppm and proposing to add a STEL of 6 ppm for perchloryl fluoride in construction and maritime. The Agency is also proposing these limits, which were recently established for perchloryl fluoride in general industry, in agriculture.

Perchloryl fluoride is a colorless noncorrosive gas or liquid with a sweet odor. It is used as an antioxidant in rocket fuels, and as an oxidizing and fluorinating agent in chemical synthesis (Hawley's 1987, p. 887; ACGIH 1986, p. 466.3).

Perchloryl fluoride is an irritant of the eyes, mucous membranes, and upper respiratory tract. The 4-hour LC_{50}s in rats and mice are 385 and 630 ppm, respectively. Dogs exposed for 4 hours to 220- to 450-ppm concentrations of the vapor, followed by exposure to 620 ppm for 2.5 hours, became hyperpneic and cyanotic and showed increased methemoglobin. Dogs succumbing to these exposures had pigment deposition in the liver, spleen, and bone marrow; alveolar hemorrhage and collapse; and emphysema (Greene, Colbourn, Donati, and Weeks 1960, as cited in ACGIH 1986/Ex. 1–3, p. 466).

Exposure to 185 ppm for 6 hours/day, 5 days/week for 7 weeks killed 18 of 20 rats, 20 of 39 mice, and all exposed guinea pigs (Greene, Colbourn, Donati, and Weeks 1960, as cited in ACGIH 1986/Ex. 1–3, p. 466). These animals had difficulty breathing, became cyanotic, and developed alveolar edema and methemoglobinemia; at autopsy, they showed fluorosis, patchy lungs, enlarged spleens, and hemosiderosis of the kidneys, spleen, and liver. When animals were exposed on a similar regimen but to a concentration of 104 ppm for six weeks, all guinea pigs but only one of 20 rats died (Greene, Colbourn, Donati, and Weeks 1960, as cited in ACGIH 1986/Ex. 1–3, p. 466). After a 6-month exposure to 24 ppm, bone fluoride levels increased fourfold in guinea pigs, threefold in rats, and about 50 percent in dogs. Animals exposed at 24 ppm showed no signs of irritation (Greene, Colbourn, Donati, and Weeks 1960, as cited in ACGIH 1986/Ex. 1–3, p. 466).

Workers exposed to an unspecified concentration of perchloryl fluoride reported experiencing upper respiratory tract irritation even after brief exposures (ACGIH 1986, p. 202). Based on effects seen in animals, severe exposure is expected to cause methemoglobinemia (Proctor, Hughes, and Fischman 1988, p. 402).

In construction and maritime, OSHA is retaining the 8-hour TWA of 3 ppm and proposing to add a STEL of 6 ppm for perchloryl fluoride; OSHA is also proposing both PELs in agriculture. These limits are based on the fluoride content of this compound. The Agency preliminarily concludes that both limits are necessary to protect workers in these sectors from the significant risk of fluorosis and hematologic effects, which constitute material impairments of health that are associated with occupational exposures to perchloryl fluoride. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

PHOSDRIN (MEVINPHOS)

CAS: 7786–34–7; Chemical Formula: $C_7H_{13}O_6P$

H.S. No. 1320

In construction and maritime, OSHA's PEL for phosdrin is an 8-hour TWA limit of 0.1 mg/m³, with a skin notation. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.01 ppm (0.1 mg/m³) and a TLV®–STEL of 0.03 ppm (0.3 mg/m³), also with a skin notation. In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA of 0.01 ppm and a STEL of 0.03 ppm; the skin notation is retained in construction and maritime and proposed in agriculture. NIOSH concurred (Ex. 8–47, Table N1) with these limits when they were recently established in general industry.

Phosdrin is a colorless liquid. The commercial product is a mixture of cis- and trans-isomers that have a yellow color. Phosdrin is used as a contact and systemic insecticide and acaricide (ACGIH 1986, p. 412; Hawley's 1987, p. 784). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The acute oral LD_{50} of phosdrin is 4 to 8 mg/kg for male mice and 6 to 8 mg/kg for female rats (Shell Chemical Corporation 1956, as cited in ACGIH 1986/Ex. 1–3, p. 412). Phosdrin is a cholinesterase inhibitor and has been reported to cause slight plasma cholinesterase depression but no decrease in brain cholinesterase activity in rats fed 2 to 5 ppm. The compound may be absorbed dermally and by inhalation or ingestion; the action of the compound is direct and immediate (Cleveland and Treon 1961/Ex. 1–476). The dermal LD_{50} in rats is 4.5 mg/kg (Gaines 1969/Ex. 1–320). Chronic feeding of rats demonstrated a minimal lethal dose of between 100 and 200 ppm. Cholinesterase activity decreased continually when sublethal doses were administered until a maximum reduction in RBC cholinesterase activity of 25 percent was achieved on the 27th day of the administration of 1.5 to 20 mg doses (Huelse and Federspil 1975/Ex. 1–959).

In industry, the primary hazards associated with exposure to phosdrin are absorption of phosdrin through the skin, lung, and mucous membranes, which causes liver damage (Natoff 1970/Ex. 1–966). Phosdrin intoxication is reported to occur in humans, with accompanying symptoms of headache, visual distortion, weakness, cramps, diarrhea, pain, and respiratory distress. Severe exposure may cause convulsions; in one reported case, some symptoms (anxiety, depression, vertigo, and nystagmus) persisted for as long as four months (Zavon, as cited in ACGIH 1986/Ex. 1–3, p. 412).

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 0.01 ppm, a STEL of 0.03 ppm, and a skin notation for phosdrin in construction, maritime, and agriculture. The Agency preliminarily concludes that these limits are necessary to protect workers in these sectors against the significant risk of cholinesterase inhibition and hepatic injury, which are material health impairments associated with exposure to this substance. OSHA believes that the proposed limits will substantially reduce these significant risks. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

PHOSPHORUS OXYCHLORIDE

CAS: 10025–87–3; Chemical Formula: $POCl_3$

H.S. No. 1323

In construction, maritime, and agriculture, OSHA has no limit for phosphorus oxychloride. The ACGIH has a TLV®–TWA of 0.1 ppm and a TLV®–STEL of 0.5 ppm for this substance. NIOSH has no REL but concurred (Ex. 8–47, Table N1) with the proposed limit when it was recently established in general industry. OSHA is proposing an 8-hour TWA PEL of 0.1 ppm for phosphorus oxychloride in construction, maritime, and agriculture.

Phosphorus oxychloride is a colorless, fuming liquid with a pungent odor. It is used in the manufacture of plasticizers, hydraulic fluids, gasoline additives, and fire retarding agents, and as a

chlorinating agent and catalyst (ACGIH 1986, p. 485).

The primary hazards associated with inhalation of phosphorus oxychloride vapor are irritation of the eyes and respiratory tract, as well as narcotic effects, gastric irritation, pulmonary edema, and nephritis (International Technical Information Institute 1978/Ex. 1–837). Weeks and associates (1964, as cited in ACGIH 1986/Ex. 1–3, p. 485) reported 4-hour LC_{50} values for phosphorus oxychloride of 48 ppm and 52 ppm for rats and guinea pigs, respectively. They also observed that ammonia vapor mediates the irritant effects of exposure to phosphorus oxychloride without significantly altering this LC_{50} value (Weeks, Downing, Musselman et al. 1964, as cited in ACGIH 1986/Ex. 1–3, p. 485).

Both chronic and acute occupational intoxication have been reported to occur among workers exposed to phosphorus oxychloride (Sassi 1954/Ex. 1–931). Acute exposure to the vapor causes burns, redness and tearing of the eyes, cough, and pain in the chest (HSDB 1991). In contact with the eyes, the vapor may cause slow-healing burns (Grant 1974, p. 829).

Based on this evidence, OSHA is proposing a PEL of 0.1 ppm (8-hour TWA) for phosphorus oxychloride in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will reduce the significant risk of narcosis and systemic poisoning among workers in these sectors. OSHA considers these adverse health effects and believes that the proposed PEL is necessary to reduce this significant risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PHOSPHORUS PENTASULFIDE

CAS: 1314–80–3; Chemical Formula: P_2S_5

H.S. No. 1324

In construction and maritime, OSHA has a limit of 1 mg/m³ as an 8-hour TWA for phosphorus pentasulfide. There is no PEL in agriculture. The ACGIH also has a TLV®–TWA of 1 mg/m³ but adds a 15-minute TLV®–STEL of 3 mg/m³. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the proposed limits. OSHA is proposing an 8-hour TWA PEL of 1 mg/m³ and a 15-minute STEL of 3 mg/m³ for phosphorus pentasulfide in construction, maritime, and agriculture. These are the limits recently established for this substance in general industry.

Phosphorus pentasulfide is a greenish-yellow crystalline mass with an odor like that of rotten eggs. Phosphorus pentasulfide is used in the manufacture of insecticides such as parathion and malathion, as an intermediate for lubricant additives, and in flotation agents, safety matches, and ignition compounds (ACGIH 1986, p. 486; Hawley's 1987, p. 912).

In contact with the skin or eyes of rabbits, this substance caused moderate irritation (RTECS 1991). The oral LD_{50} in rats is 389 mg/kg (RTECS 1991).

The primary hazard associated with exposure to phosphorus pentasulfide is respiratory irritation (Smyth 1956/Ex. 1–759). In the presence of moisture, phosphorus pentasulfide is rapidly hydrolyzed to phosphoric acid and hydrogen sulfide. The ACGIH (1986/Ex. 1–3, p. 485) considers phosphorus pentasulfide to be as toxic as phosphoric acid.

Based on this evidence, OSHA is retaining the 8-hour TWA PEL of 1 mg/m³ and proposing to add a 15-minute STEL of 3 mg/m³ for phosphorus pentasulfide in construction and maritime. OSHA is also proposing both limits in agriculture. The Agency preliminarily concludes that these limits are necessary to reduce the significant risk of respiratory irritation, a material health impairment. These are the limits recently established for phosphorus pentasulfide in general industry.

PHTHALIC ANHYDRIDE

CAS: 85–44–9; Chemical Formula: $C_6H_4(CO)_2O$

H.S. No. 1326

In construction and maritime, OSHA has an 8-hour TWA limit of 2 ppm for phthalic anhydride. There is no limit in agriculture. The ACGIH has a limit of 1 ppm TWA for phthalic anhydride. NIOSH has no REL but concurs (Ex. 8–47, Table N1) that the proposed limit is appropriate. OSHA is proposing an 8-hour TWA PEL of 1 ppm for phthalic anhydride in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Phthalic anhydride takes the form of white crystalline needles and has a mild odor. It is used in the manufacture of insecticides, pharmaceuticals, resins, polyesters, dyes, and plasticizers (ACGIH 1986, p. 487; Hawley's 1987, p. 915).

The primary exposure hazards associated with phthalic anhydride are severe skin, eye, and respiratory irritation. The substance can also produce skin and, perhaps, pulmonary sensitization. The oral LD_{50} in rats is 4020 mg/kg (RTECS 1991). In contact with the skin of rabbits, phthalic anhydride caused mild irritation; instillation in the eye led to severe irritation (RTECS 1991). Exposure to the heated vapor of phthalic anhydride at unidentified concentrations caused pulmonary injury; in contact with the skin of guinea pigs, this substance caused sensitization (NRC 1977, p. 756). Baader (1955/Ex. 1–1139) has reported irritant effects in experimental animals exposed to 30 mg/m³ (approximately 5 ppm) phthalic anhydride in air.

Workers exposed to phthalic anhydride concentrations of about 4 ppm reported experiencing mucous membrane irritation, and at 5 ppm, conjunctivitis developed in these workers (AIHA 1967).

In studies of workers exposed to unspecified concentrations of phthalic acid vapors and phthalic anhydride, symptoms of respiratory tract injury as well as bronchitis, eye irritation, cough, bloody sputum, and nasal bleeding have been reported (Proctor, Hughes and Fischman 1988, p. 415). Several of these workers also developed bronchial asthma and skin sensitization (Proctor, Hughes, and Fischman 1988, p. 415). Respiratory sensitization has been confirmed by patch testing in at least one case (Maccia, Bernstein, Emmett, Brooks 1976).

Based on this evidence, OSHA is proposing an 8-hour TWA limit of 1 ppm for phthalic anhydride in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will reduce the significant risk among workers in these sectors of respiratory irritation and skin and pulmonary sensitization, which constitute material impairments of health. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PINDONE

CAS: 83–26–1; Chemical Formula: $C_{14}H_{14}O_3$

H.S. No. 2132

In general industry, construction, and maritime, OSHA's permissible exposure limit for pindone is 0.1 mg/m³ as an 8-hour TWA. There is no limit for this substance in agriculture. The ACGIH has a TLV®–TWA of 0.1 mg/m³ for pindone; NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing to establish a PEL of 0.1 mg/m³ as an 8-hour TWA for pindone in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Pindone (also called Pival) is an odorless substance that takes the form of bright yellow crystals. Pindone is used primarily as a rodenticide and insecticide. It is also used as a pharmaceutical intermediate (ACGIH

1986, p. 491; Hawley's 1987, p. 923). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Pindone causes disturbances in blood coagulation in humans and animals; the mechanism of pindone's action is vitamin K antagonism, which, in turn, inhibits prothrombin formation (Proctor, Hughes, and Fischman 1988, p. 417). The oral LD_{50}s in rats, dogs, and rabbits are 280 mg/kg, 75 mg/kg, and 150 mg/kg, respectively (RTECS 1988). Ingestion of a single large dose of pindone caused death in rats as a result of pulmonary and visceral congestion (U.S. Public Health Service 1956, in Proctor, Hughes, and Fischman 1988, p. 417). Although the single-dose LD_{50} in dogs is 75 mg/kg, daily doses of as little as 2.5 mg/kg are fatal in dogs after a few days (Worthing 1983, in HSDB 1988). Rabbits given oral doses of 1 mg/kg/day survived for an average of 8 to 10 days; acutely poisoned animals had evidence of hemorrhage throughout the muscles and organs at autopsy (HSDB 1988). The offspring of pregnant dogs given pindone were stillborn or died shortly after birth; autopsy of the pups revealed severe hemorrhage in most organs (Fitzek and Gembardt 1977, in HSDB 1988). Chronic exposure to low doses of pindone causes death in experimental animals; the cause of death in these cases is multiple internal hemorrhage (Proctor, Hughes, and Fischman 1988, p. 417).

In humans, pindone reduces blood coagulation and occasionally produces symptoms similar to those caused by warfarin, such as anorexia, nausea, vomiting, and diarrhea (Goodman 1975, in HSDB 1988; Merck 1983, p. 1073). Swallowing repeated small doses of pindone may cause generalized bleeding, such as nose bleeds or excessive bleeding from minor cuts. Individuals may experience stomach and back pain for several days after pindone exposure (NIOSH/OSHA Occupational Health Guideline 1981, p. 1).

Based on this evidence in humans and animals, OSHA preliminarily concludes that pindone is an anticoagulant causing internal hemorrhaging. In the absence of a limit for pindone, OSHA believes that workers in agriculture are at significant risk of experiencing this adverse health effect; the Agency believes that establishing a PEL of 0.1 mg/m^3 for pindone will substantially reduce the risk of this material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PROPARGYL ALCOHOL

CAS: 107–19–7; Chemical Formula: $HC{=}CCH_2OH$

H.S. No. 1335

In construction, maritime, and agriculture, OSHA has no limit for propargyl alcohol. The ACGIH has a TLV®–TWA of 1 ppm, with a skin notation, for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the proposed limit. OSHA is proposing an 8-hour TWA PEL of 1 ppm for propargyl alcohol in construction, maritime, and agriculture. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

Propargyl alcohol is a colorless liquid with a mild, geranium-like odor (HSDB 1991). This substance is used as a solvent stabilizer, corrosion inhibitor, chemical intermediate, and soil fumigant (ACGIH 1986, p. 496).

In rats, guinea pigs, and mice, the oral LD_{50}s are 70, 60, and 50 mg/kg, respectively; the 2-hour inhalation LC_{50} in both the rat and mouse is about 850 ppm (NIOSH 1977i/ Ex. 1–1182). The dermal LD_{50} in rabbits is 88 mg/kg (RTECS 1990).

Propargyl alcohol is a primary skin irritant, but it is not a skin sensitizer (Antara Chemicals 1952, as cited in ACGIH 1986/Ex. 1–3, p. 496). The toxicity of propargyl alcohol is estimated to be equal to that of allyl alcohol (oral LD_{50} in rats of 64 mg/kg) (NIOSH 1977i/Ex. 1182). The ACGIH limit is based on the structural and toxicological similarity of propargyl alcohol to allyl alcohol (ACGIH 1986/ Ex. 1–3, p. 496). Propargyl alcohol also causes degenerative changes in liver and kidneys in an 89-day rat study (Rowe and McCollister (1982) in *Patty's Industrial Hygiene and Toxicology* 1982, Vol. 2C, p. 4673).

Based on this evidence, OSHA is proposing an 8-hour TWA for propargyl alcohol of 1 ppm, with a skin notation, in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that these limits are necessary to protect workers in these sectors against the significant risk of skin and mucous membrane irritation, CNS depression, and liver and kidney damage associated with exposure to this substance. OSHA believes that these health effects constitute material impairments of health. In addition, promulgation of the proposed PEL will make OSHA's limit for propargyl alcohol consistent across all regulated sectors.

PROPIONIC ACID

CAS: 79–09–4; Chemical Formula: CH_3CH_2COOH

H.S. No. 1336

OSHA has no limit for propionic acid in construction, maritime, and agriculture. The ACGIH has a TLV®–TWA of 10 ppm for this substance; the TLV® was set on the basis of analogy with acetic acid (10 ppm 8-hour TLV®). NIOSH has no REL but concurred (Ex. 8–47, Table N1) that this limit when it was recently established is appropriate in general industry. OSHA is proposing an 8-hour TWA PEL of 10 ppm for propionic acid in construction, maritime, and agriculture.

Propionic acid is a colorless, oily liquid with a pungent odor. Propionic acid and propionates are used as herbicides, fungicides, mold inhibitors, grain and wood chip preservatives, emulsifying agents, in pharmaceuticals, and in artificial fruit flavors (ACGIH 1986, p. 498; Hawley's 1987, p. 971).

The primary health effects associated with exposure to propionic acid are skin burns and irritation of the eyes and respiratory system. Smyth, Carpenter, Weil, and co-workers (1962/Ex. 1–441) reported that the oral LD_{50} in rats is 4.3g/kg; NIOSH (1977i/Ex. 1–1182) stated that the intravenous LD_{50} in mice is 625 mg/kg and the skin absorption LD_{50} in rabbits is 500 mg/kg. Inhalation of the saturated vapor for eight hours caused no fatalities in rats (ACGIH 1986/Ex. 1–3, p. 498).

Acute industrial exposures to propionic acid have been reported to cause mild to moderate skin burns, eye irritation, and, in a single incident, asthmatic cough. No irritation was observed when human volunteers were exposed to concentrations of propionic acid averaging below 0.25 ppm, with excursions to 2.1 ppm, over an eight-hour period (Dow Chemical Company 1977o, as cited in ACGIH 1986/Ex. 1–3, p. 498).

Based on this evidence, OSHA is proposing an 8-hour TWA limit of 10 ppm for propionic acid in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit is necessary to protect workers in these sectors against the significant risk of eye and respiratory tract irritation, which are material impairments of health. This is the limit recently established for this substance in general industry.

n-PROPYL ACETATE

CAS: 109–60–4; Chemical Formula: $CH_3COOCH_2CH_2CH_3$

H.S. No. 1338

In construction and maritime, OSHA has an 8-hour TWA limit of 200 ppm for n-propyl acetate. There is no PEL in

agriculture. The ACGIH also has a 200-ppm TLV®–TWA but adds a TLV®–STEL of 250ppm. NIOSH has no REL but concurs (Ex. 8–47, Table N1) that these limits are appropriate; these are the limits established recently in general industry. OSHA is proposing limits for n-propyl acetate of 200 ppm as an 8-hour TWA and 250 ppm as a 15-minute STEL; these limits would apply in construction, maritime, and agriculture.

n-Propyl acetate is a pleasant-smelling liquid. This substance is used in flavoring agents, perfumes, natural and synthetic resins, lacquers, plastics and organic synthesis, and as a solvent for nitrocellulose and other cellulose derivatives (ACGIH 1986, p. 500; Hawley's 1987, p. 972).

The primary health effects associated with exposure to n-Propyl acetate are narcosis and eye and respiratory irritation. The oral LD_{50} in rats is 9370 mg/kg, and the lowest lethal concentration in the same species is 8000 ppm for 4 hours (RTECS 1991). In contact with the skin of rabbits, this substance caused mild irritation (RTECS 1991). n-Propyl acetate's narcotic action is 1.3 times that of ethyl acetate; salivation and irritation of cats' eyes occurred at a concentration of 2600 ppm (Flury and Wirth 1933, as cited in ACGIH 1986/Ex. 1–3, p. 500).

n-Propyl acetate appears to be more toxic than isopropyl acetate or ethyl acetate but less so than n-butyl acetate (ACGIH 1986, p. 500). Exposure to a concentration of 200 ppm causes eye irritation in humans, and exposure to higher concentrations causes nasal and upper respiratory tract irritation (Parmezziani 1983, p. 782). Repeated or prolonged contact of the skin may cause defatting and dermatitis (Parmezziani 1983, p. 782).

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 200 ppm and a 15-minute STEL of 250 ppm for n-propyl acetate in construction, maritime, and agriculture. The Agency preliminarily concludes that these limits are necessary to prevent a significant risk of narcosis and eye and respiratory tract irritation among workers in these sectors. OSHA considers these effects material impairments of health. These are the limits recently established for this substance in general industry.

PROPYL ALCOHOL

CAS: 71–23–8; Chemical Formula: $CH_3CH_2CH_2OH$

H.S. No. 1339

In construction and maritime, OSHA has a limit of 200 ppm (8-hour TWA) for n-propyl alcohol. There is no limit in agriculture. The ACGIH has the same TWA limit but adds a 250-ppm 15-minute STEL and a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N1) that these limits are appropriate; these are the limits recently established in general industry. OSHA is proposing limits for propyl alcohol in construction, maritime, and agriculture of 200 ppm as an 8-hour TWA and 250 ppm as a 15-minute STEL.

Propyl alcohol is a colorless liquid with an alcohol-like odor. Propyl alcohol is used in brake fluids and polishing compounds, as a solvent for waxes, vegetable oils and resins, as a degreasing agent and antiseptic, and as a chemical intermediate (ACGIH 1986, p. 500; Hawley's 1987, p. 972).

The primary health effects associated with exposure to propyl alcohol are irritation and narcosis. The oral LD_{50} for propyl alcohol in rats is 1.9 g/kg (Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1–440). The dermal LD_{50} in rabbits is 5040 mg/kg, and the LC_{50} in mice is 48 g/m³ (RTECS 1991). Starrek reported deep narcosis in mice inhaling the vapor at a concentration of 4100 ppm for 240 minutes and of 24,500 ppm for 60 minutes; ataxia appeared in 90 to 120 minutes on exposure to 3250 ppm (Starrek 1938/Ex. 1–872). These effects are almost twice as intense as those reported for exposure to the vapor of isopropyl alcohol. Rats intubated or given subcutaneous injections of propyl alcohol in carcinogenicity bioassays developed tumors (both benign and malignant) at multiple sites; animals also showed severe liver injury and hematopoietic effects (Gibel, Lohs, and Wildner 1975).

Nelson, Enge, Ross, and associates (1943/Ex. 1–66) reported mild eye, nose, and throat irritation in humans exposed at 400 ppm to the vapor of isopropyl alcohol, but no data exist on human sensory response to propyl alcohol vapor. The ACGIH (1986/Ex. 1–3, p. 500) reports that many industrial hygienists consider the vapor of propyl alcohol to be more irritating to the throat than the vapor of the isomer.

Based on this evidence, OSHA is retaining the 8-hour TWA PEL of 200 ppm in construction and maritime, proposing to add a STEL of 250 ppm, and proposing to extend both limits to agriculture. The Agency preliminarily concludes that these limits are necessary to protect workers in these sectors against the significant risk of narcosis and irritation, both material impairments of health, that are associated with exposure to this substance. These are the limits recently established for this substance in general industry.

PROPYLENEIMINE

CAS: 75–55–8; Chemical Formula: C_3H_7N

H.S. No. 2135

In general industry, construction, and maritime, OSHA's permissible exposure limit for propyleneimine is 2 ppm as an 8-hour TWA; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit for propyleneimine in agriculture. The ACGIH has a TLV®–TWA of 2 ppm, with a skin notation, for propyleneimine. The ACGIH TLV® for propyleneimine was set by analogy with ethyleneimine, a potential human carcinogen (ACGIH 1986, p. 504). NIOSH has no REL for this substance but concurs with the limit being proposed (Ex. 8–47). OSHA is proposing an 8-hour TWA limit of 2 ppm, with a skin notation, for propyleneimine in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Propyleneimine, also called 2-methyl aziridine, is a clear, fuming liquid with a strong ammonia-like odor. This substance is used as a chemical intermediate in the production of paper, textile, and rubber and pharmaceutical chemicals and as an ingredient in latex surface coatings (ACGIH 1986, p. 504; Hawley's 1987, p. 974).

Propyleneimine causes severe eye, mucous membrane, and skin irritation in humans and animals and is carcinogenic in animals. The oral LD_{50} in rats is 19 mg/kg (RTECS 1991). The lowest lethal concentrations in rats and guinea pigs are 500 ppm over 4 hours and 500 ppm for 1 hour, respectively (RTECS 1991). The dermal LD_{50} in guinea pigs is 43 mg/kg (RTECS 1991). Rabbits exhibited signs of severe eye irritation when 0.025 mg of propyleneimine was instilled into their eyes (RTECS 1989); injury was graded 9 on an ascending severity scale of 1 to 10 (Grant 1986, p. 770). Rats given 20 mg/kg propyleneimine in water by gavage twice weekly for 28 weeks developed gliomas, ear-duct squamous cell carcinomas, intestinal adenocarcinomas, leukemias, and breast tumors (Ulland et al. 1971, in IARC 1975, Vol. 9, p. 63). In another study, rats given 10 mg/kg propyleneimine by gavage for 60 weeks developed leukemias, gliomas, ear-duct squamous-cell carcinomas, intestinal adenocarcinomas, and breast tumors (Ulland et al. 1971, in IARC 1975, Vol. 9, p. 63). Based on these findings, the International Agency for Research on Cancer has concluded that the evidence for the carcinogenicity of propyleneimine in animals is sufficient (IARC 1975, Vol. 9, p. 64).

Propyleneimine is mutagenic in bacterial

test systems without metabolic activation (RTECS 1991; HSDB 1985).

The toxicity of propyleneimine is believed to be from ¼ to ⅛ that of ethyleneimine (Carpenter et al. 1948, in ACGIH 1986, p. 504). According to the ACGIH, propyleneimine should be classified as a carcinogen of "intermediate * * * potency" (ACGIH 1986, p. 504). There are no reports of systemic effects in workers exposed to this substance.

Based on this evidence and by analogy with ethyleneimine, OSHA preliminarily concludes that propyleneimine is an irritant and potential occupational carcinogen. OSHA believes that, in the absence of a limit for this substance, workers in agriculture are at significant risk of experiencing these exposure-related effects. The Agency believes that establishing a PEL of 2 ppm as an 8-hour TWA for propyleneimine will substantially reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PROPYLENE OXIDE

CAS: 75–56–9; Chemical Formula: CH_3CHOCH_2

H.S. No. 1344

In construction and maritime, OSHA has an 8-hour TWA limit of 100 ppm for propylene oxide. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 20 ppm for this substance. OSHA is proposing an 8-hour TWA of 20 ppm for propylene oxide in construction, maritime, and agriculture; this is the limit recently established in general industry.

Propylene oxide is a colorless, highly flammable, volatile, and ethereal liquid. Propylene oxide is used as a fumigant and as an intermediate in the production of polyols for detergents, surfactants, propylene glycols, and synthetic lubricants (ACGIH 1986, p. 504; Hawley's 1987, p. 974).

The health hazards associated with exposure to this substance are primary skin, eye, and respiratory irritation, as well as central nervous system depression. The oral LD_{50} values in rats and guinea pigs are 930 mg/kg and 690 mg/kg, respectively. In mice, the LC_{50} is 1740 ppm for 4 hours. Dogs and guinea pigs exposed for 4 hours at 2000 and 4000 ppm, respectively, died (NIOSH 1977i/Ex. 1–1182). Although only some species tolerate daily exposures to 200 ppm, all species tested tolerated 100 ppm without ill effects (Rowe, Hollingsworth, Oyen et al. 1956/Ex. 1–609). Jacobson and associates (1956/Ex. 1–702) considered the toxic effects of propylene oxide to be one-half to one-third as intense as those of ethylene oxide (Jacobson, Hackley, and Feinsilver 1956/Ex. 1–702).

Corneal burns and skin necrosis, as well as respiratory and pulmonary irritation, have been reported in humans as a result of direct contact with the liquid or vapor of propylene oxide (Patty 1963h/Ex. 1–857); Central nervous system effects, including ataxia, incoordination, and general depression, also occur as a result of exposure to unspecified concentrations.

In the prior rulemaking, OSHA received several comments on propylene oxide. These commenters (Exs. 3–746, 8–47, Table N6B) noted that propylene oxide is a potential occupational carcinogen, based on an NTP bioassay in rats and mice that demonstrates "some evidence" of carcinogenicity in rats and "clear evidence" of carcinogenicity in mice (Ex. 8–47). In response to these commenters, OSHA stated that the Agency is aware of propylene oxide's serious health effects; however, because the primary purpose of the present rulemaking is to ensure consistency in PELs across OSHA-regulated sectors, OSHA is not at this time considering a further reduction in the PEL. The first PEL update, however, will review the literature on propylene oxide.

In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA limit of 20 ppm for propylene oxide to protect workers in these sectors against the significant risk of primary irritation and CNS depression, which constitute material health impairments that are associated with exposure to propylene oxide. The Agency believes that this limit is necessary to substantially reduce this significant risk. This is the limit recently established for this substance in general industry.

SELENIUM HEXAFLUORIDE

CAS: 7783–79–1; Chemical Formula: SeF_6

H.S. No. 2141

OSHA's permissible exposure limit for selenium hexafluoride in general industry, construction, and maritime is 0.05 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.05 ppm for selenium hexafluoride; this limit was established on the basis of selenium hexafluoride's similarity in physiological effect to ozone (ACGIH 1986, p. 518). NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing to establish an 8-hour TWA limit of 0.05 ppm for selenium hexafluoride in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Selenium hexafluoride is a colorless gas. It is used as a gaseous electric insulator (ACGIH 1986, p. 518; Merck 1983, p. 1213).

Selenium hexafluoride causes severe pulmonary irritation in animals; its acute toxicity is about one-half that of ozone (ACGIH 1986, p. 518). Acute toxicity studies in rats, mice, rabbits, and guinea pigs showed that exposure to selenium hexafluoride concentrations of 10 ppm for 4 hours is fatal (Kimmerle 1960, in ACGIH 1986, p. 518). Exposure of animals of these species to a concentration of 5 ppm selenium hexafluoride caused pulmonary edema; exposure to a 1-ppm concentration produced no grossly observable effects (Kimmerle 1960, in ACGIH 1986, p. 518). Exposing rats, mice, rabbits, and guinea pigs to a 5-ppm concentration of selenium hexafluoride for 1 hour/day for 5 days caused signs of pulmonary injury; exposure to a 1-ppm concentration on the same regimen caused no effects (Kimmerle 1960, in ACGIH 1986, p. 518).

Based on effects seen in animals, selenium hexafluoride is expected to cause severe respiratory irritation in exposed humans; exposure is also likely to cause selenium poisoning (HSDB 1985; Proctor, Hughes, and Fischman 1988, p. 439). The signs and symptoms of acute selenium poisoning include nausea, vomiting, indigestion, headache, irritability, unstable blood pressure, and yellowish skin (Freiberg 1979, in HSDB 1985). Inhalation exposure can impart a garlic-like odor to the breath (Freiberg 1979, in HSDB 1985).

Based on this evidence, OSHA preliminarily concludes that exposure to selenium hexafluoride may cause severe respiratory irritation and selenium poisoning. OSHA believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these material health impairments and that establishing an 8-hour TWA limit of 0.05 ppm is necessary to reduce this significant risk. Promulgation of this limit also will make the PEL for selenium hexafluoride consistent across all OSHA-regulated sectors.

SILICON TETRAHYDRIDE

CAS: 7803–62–5; Chemical Formula: SiH_4

H.S. No. 1361

In construction, maritime, and agriculture, OSHA has no limit for silicon tetrahydride. The ACGIH TLV®–TWA is 5 ppm. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. In construction, maritime, and agriculture, OSHA is

proposing an 8-hour TWA PEL for silicon tetrahydride of 5 ppm. This is the limit recently established for this substance in general industry.

Silicon tetrahydride, a colorless gas, is used in the manufacture of semiconductors. Silicone in a commonly used synonym for silicon tetrahydride.

Silicon tetrahydride is a mild irritant of the eyes and mucous membranes. The LC_{50} in rats is 9600 ppm for 4 hours (RTECS 1991). Studies of rats exposed to silicon tetrahydride at levels of 126 ppm for one hour (Matheson Gas Products 1971, as cited in ACGIH 1986/Ex. 1–3, p. 528) and at 1400 ppm for six hours (Union Carbide Corporation 1980, as cited in ACGIH 1986/Ex. 1–3, p. 528) have failed to identify any systemic effects associated with exposure to this chemical.

Based on this evidence, OSHA is proposing an 8-hour TWA limit of 5 ppm for silicon tetrahydride in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that this limit will protect workers in these sectors from the significant risk of exposure-related eye, skin, and upper respiratory tract irritation, which are material health impairments. This is the limit recently established for this substance in general industry.

STIBINE

CAS: 7803–52–3; Chemical Formula: SbH_3

H.S. No. 2144

In general industry, construction, and maritime, OSHA's current permissible exposure limit for stibine is 0.1 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA limit of 0.1 ppm for this substance; this limit was established on the basis of stibine's similarity in physiological effect to arsine, a gas generally found with stibine. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing a PEL of 0.1 ppm for stibine in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Stibine is a colorless gas with a disagreeable, hydrogen sulfide-like odor; it is usually produced inadvertently when acid solutions of antimony are treated with zinc or other reducing agents (ACGIH 1986, p. 586.3). It was formerly used as a fumigating agent and in chemical synthesis (ACGIH 1986, p. 536.3; NIOSH/OSHA Occupational Health Guideline 1981, p. 2).

Stibine is an irritant and a hemolytic agent in both humans and animals. This substance also causes liver and kidney injury in animals. The lowest lethal concentration (LC_{LO}) in mice is 10 ppm for an unspecified period; in guinea pigs, the LC_{LO} is 92 ppm for 1 hour (RTECS 1991). Cats and dogs exposed to a stibine concentration of 40 to 45 ppm for 1 hour developed pulmonary congestion and edema; death occurred within 24 hours of exposure (Webster 1946, in ACGIH 1986, p. 536.3). Guinea pigs developed hemoglobinuria followed by anemia after being exposed to a 65-ppm concentration of stibine for 1 hour (Webster 1946, in Proctor, Hughes, and Fischman 1988, p. 445).

One of the early signs of overexposure to stibine in humans is hemoglobinuria (Dernehl, Stead, and Nau 1943, in Clayton and Clayton 1981, p. 1511). Other signs and symptoms of acute overexposure to stibine include eye irritation, headache, nausea, weakness, slow breathing, and a weak, irregular pulse (Dernehl, Stead, and Nau 1943, in Clayton and Clayton 1981, p. 1511). No clear-cut case of fatal stibine poisoning in humans has been reported because stibine usually occurs in combination with arsine, another hemolytic agent (Clayton and Clayton 1981, p. 1511). Workers concurrently exposed to several gases (arsine, stibine, and hydrogen sulfide) at unmeasured concentrations developed headache, weakness, nausea, abdominal and lumbar pain, hemoglobinuria, hematuria, and anemia (Dernehl, Stead, and Nau 1944, in Proctor, Hughes, and Fischman 1988, p. 445).

Based on this evidence, OSHA preliminarily concludes that exposure to stibine is associated with hemolysis, respiratory irritation, and possible liver and kidney injury. OSHA believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse effects and that establishing a limit of 0.1 ppm as an 8-hour TWA is necessary to reduce these risks of material health impairment. Promulgation of this limit will also make the PEL for stibine consistent across all OSHA-regulated sectors.

SULFURYL FLUORIDE

CAS: 2699–79–8; Chemical Formula: SO_2F_2

H.S. No. 1379

In construction and maritime, OSHA's limit for sulfuryl fluoride is 5 ppm as an 8-hour TWA. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 5 ppm and a TLV®–STEL of 10 ppm. NIOSH has no REL but concurs (Ex. 8–47, Table N1) that the proposed limits are appropriate. OSHA is proposing an 8-hour TWA PEL of 5 ppm and a 15-minute STEL of 10 ppm for sulfuryl fluoride in construction, maritime, agriculture. These are the limits recently established for this substance in general industry.

Sulfuryl fluoride is a colorless gas with a sulfide odor. Sulfuryl fluoride is primarily used as an insecticide and fumigant. It is also used in the organic synthesis of drugs and dyes (ACGIH 1986, p. 546; Sittig 1985, p. 820). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In extensive animal studies conducted by the Dow Chemical Company (1962 and 1970, as cited in ACGIH 1986/Ex. 1–3, p. 546), sulfuryl fluoride was determined to exhibit one-half to one-third the acute inhalation toxicity of methyl bromide. Acute exposures of animals resulted in tremors that later developed into severe convulsions. Pulmonary edema was seen in laboratory animals after a single severe exposure. Repeated exposures of rats, guinea pigs, and mice to 20 ppm sulfuryl fluoride for seven hours per day produced both kidney and lung injury after six months. Some evidence of fluorosis was observed in the incisors of mice, but not in the teeth of the rats or guinea pigs (Dow Chemical Company 1962 and 1970, as cited in ACGIH 1986/Ex. 1–3, p. 546).

A report by Taxay (1986/Ex. 1–577) that examined an incident of acute workplace overexposure to sulfuryl fluoride noted that abdominal pain, nausea, vomiting, and itching were the major symptoms. On the day after exposure, the serum of the affected worker tested positive for fluoride.

Based on this evidence, OSHA is retaining the 8-hour TWA limit of 5 ppm and proposing to add a STEL of 10 ppm for sulfuryl fluoride in construction and maritime; the Agency is also proposing both limits in agriculture. The Agency preliminarily concludes that these limits will protect workers in these sectors against the significant risks of kidney and lung injury and of fluorosis, which constitute exposure-related material health impairments. These are the limits recently established for this substance in general industry.

TEDP (SULFOTEP)

CAS: 3689–24–5; Chemical Formula: $(C_2H_5)_4P_2S_2O_5$

H.S. No. 2149

OSHA's current permissible exposur limit (PEL) for TEDP (tetraethyldithiopyrophosphate) in construction, maritime, and general industry is 0.2 mg/m³ as an 8-hour

TWA; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.2 mg/m³, with a skin notation, for TEDP. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA limit of 0.2 mg/m³, with a skin notation, for TEDP in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

TEDP, also called Sulfotep, is a yellow, noncombustible liquid with a garlic-like odor. TEDP is an organophosphate pesticide that is used as an insecticide and miticide (ACGIH 1986, p. 541; Merck 1983, p. 1287). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA)

TEDP causes cholinesterase inhibition in humans and animals; the inactivation of cholinesterase causes acetylcholine to accumulate at synapses in the nervous system, skeletal and smooth muscles, and secretory glands (Proctor, Hughes, and Fischman 1988, p. 465). Oral LD_{50}s in rats, mice, and rabbits are 5 mg/kg, 22 mg/kg, and 25 mg/kg, respectively (RTECS 1991). Rats have an LC_{50} of 38 mg/m³ for 4 hours; in mice, the LC_{50} is 40 mg/m³ for 4 hours (RTECS 1991). In rats and rabbits, the dermal LD_{50}s are 65 mg/kg and 20 mg/kg, respectively (RTECS 1991). TEDP affects the autonomic nervous system in rats, mice, dogs, cats, and rabbits by all routes of exposure (RTECS 1991). The signs and symptoms of TEDP poisoning include hypersalivation, lacrimation, sweating, nasal discharge, vomiting, dyspnea, muscle fasciculation, weakness, and paralysis. Effects on the central nervous system consist of nervousness, ataxia, convulsions, coma, and death due to respiratory failure or cardiac arrest (Clarke 1981, p. 153, in HSDB 1988). Fed at a dietary concentration of 60 ppm for 12 weeks, TEDP produced no symptoms in rats; however, feeding at a dose of 180 ppm caused illness and tissue change (Lehman 1952, in ACGIH 1986, p. 541). TEDP is estimated to be about ½ as toxic as parathion (Lehman 1951, in ACGIH 1986, p. 541).

In humans, severe TEDP poisoning can occur by any route of exposure. After inhalation of toxic amounts, respiratory and ocular effects (including a feeling of tightness in the chest, wheezing, laryngeal spasms, cyanosis, blurred vision, miosis, and tearing) occur within a few minutes of exposure (Proctor, Hughes, and Fischman 1988, p. 465). Gastrointestinal signs and symptoms, such as anorexia, nausea, vomiting, cramps, and diarrhea, appear 15 minutes to 2 hours following the ingestion of TEDP (Proctor, Hughes, and Fischman 1988, p. 465). The accumulation of acetylcholine in the central nervous system, which is caused by TEDP-induced cholinesterase inhibition, can cause tension, anxiety, restlessness, insomnia, neurosis, headache, apathy, and confusion (Klaassen 1986, p. 528). An excess of acetylcholine at the neuromuscular junctions of skeletal muscle causes weakness, involuntary twitching, fasciculations, and paralysis. Recovery usually occurs within one week after the cessation of exposure (Proctor, Hughes, and Fischman 1988, p. 465). Chronic exposure to TEDP concentrations that are too small to cause symptoms immediately can eventually cause poisoning, indicating that TEDP accumulates in the system (Proctor, Hughes, and Fischman 1988, p. 465).

Based on this evidence in humans and animals and by analogy with parathion, OSHA preliminarily concludes that TEDP causes the signs and symptoms associated with cholinesterase inhibition. OSHA believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these effects. OSHA believes that establishing a PEL of 0.2 mg/m³ as an 8-hour TWA, with a skin notation, is necessary to reduce this risk of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

TELLURIUM HEXAFLUORIDE

CAS: 7783–80–4; Chemical Formula: TeF_6

H.S. No. 2151

In general industry, construction, and maritime, OSHA's current permissible exposure limit for tellurium hexafluoride is 0.02 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.02 ppm for this substance; the ACGIH limit was established by analogy with the effects of exposure to ozone. NIOSH has no REL but concurs (Ex. 8–47) with the limit being proposed. OSHA is proposing an 8-hour TWA limit of 0.02 ppm for tellurium hexafluoride in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Tellurium hexafluoride is a colorless, noncombustible gas with an unpleasant odor. This substance is used as an analytical reagent in research laboratories (ACGIH 1986, p. 556; Genium MSDS 1988, No. 250).

Tellurium hexafluoride causes pulmonary irritation in animals; signs and symptoms of exposure in humans include headache and dyspnea. The lowest lethal concentrations in various animal species are 5 ppm for 4 hours in rats, rabbits, and guinea pigs, and 5 ppm for 1 hour in mice (RTECS 1991). Exposed to a concentration of 1 ppm for 4 hours, animals of four species (rats, mice, rabbits, and guinea pigs) showed evidence of pulmonary edema; exposures to concentrations of between 5 and 100 ppm tellurium hexafluoride were fatal to all animals of all experimental species (Kimmerle 1960, in ACGIH 1986, p. 556). A one-hour exposure to a 1-ppm concentration of tellurium hexafluoride for 5 days caused no effects in animals of these four species; the author of this study attributed this finding to the development of tolerance to tellurium hexafluoride's acute effects (Kimmerle 1960, in ACGIH 1986, p. 556).

In humans, exposure to tellurium hexafluoride causes headache and difficult breathing (Cooper 1971; Cerwenka and Cooper 1961, in Proctor, Hughes, and Fischman 1988, p. 458). Two workers exposed to an unknown concentration of tellurium hexafluoride generated by a 50-g leak of this substance in a laboratory developed garlic breath, a bluish-black discoloration of the webs of the fingers, and streaks on the neck and face; these individuals also reported fatigue. Both workers subsequently recovered (Blackadder and Manderson 1975, in Proctor, Hughes, and Fischman 1988, p. 458).

Based on this evidence in humans and animals and by analogy with selenium hexafluoride, OSHA preliminarily concludes that tellurium hexafluoride causes pulmonary irritation, headaches, and dyspnea in exposed individuals. OSHA believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these effects and that establishing a PEL for tellurium hexafluoride of 0.02 ppm as an 8-hour TWA is necessary to substantially reduce this risk of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

TEPP

CAS: 107–49–3; Chemical Formula: $(C_2H_5)_4P_2O_7$

H.S. No. 2152

OSHA's current limit for tetraethyl pyrophosphate (TEPP) in general

industry, construction, and maritime is 0.05 mg/m³ as an 8-hour TWA, with a skin notation. There is no PEL for this substance in agriculture. The ACGIH TLV®–TWA for TEPP, which was set by analogy to parathion, is 0.05 mg/m³ (0.004 ppm), with a skin notation. NIOSH has no REL for this substance but concurs (Ex. 8–47) with the PEL being proposed by OSHA. The Agency is proposing a PEL of 0.05 mg/m³ as an 8-hour TWA, and a skin notation, for TEPP in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

TEPP is a colorless to amber-colored liquid with a mild fruity odor. TEPP was one of the first organophosphate pesticides used in the United States. It is used as an insecticide and acaricide against aphids, spider mites, mealybugs, leaf hoppers, and thrips. TEPP has been replaced by other products in many of its applications. It was formerly used in the treatment of glaucoma, pediculosis of the eyelashes, and myasthenia gravis (HSDB 1989; Grant 1986, p. 893). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

TEPP is a cholinesterase inhibitor that is very toxic to warm-blooded animals by all routes of exposure. The oral LD_{50} in rats is 500 μg/kg, and the dermal LD_{50} in the same species is 2400 μg/kg (RTECS 1989). Acutely poisoned animals exhibit hypersalivation, lacrimation, excessive perspiration, nasal discharge, miosis, breathing difficulties, nausea, diarrhea, and central nervous system effects including nervousness, ataxia, convulsions, and coma. Death is caused by respiratory failure or cardiac arrest (HSDB 1991).

In humans, TEPP is a powerful inhibitor of cholinesterase activity. The lowest concentration reported to be lethal in humans by ingestion is 309 μg/kg (RTECS 1989). TEPP causes its toxic effects by inhibiting the activity of the enzyme cholinesterase, which causes, in turn, a buildup of acetylcholine at the synapses in the nervous and skeletal systems, smooth muscles, and secretory glands (Koelle 1963, Taylor 1985, and Hayes 1963, in Proctor, Hughes, and Fischman 1988, p. 464). TEPP causes ocular effects, including miosis, aching of the eyes, and blurring of distant vision, when it is applied to the eyelashes (as was formerly done to treat pediculosis of the eyelashes) or when it is absorbed in systemically toxic amounts (Grant 1986, p. 893). Volunteers given oral TEPP doses of 4 mg exhibited a decrease in plasma cholinesterase activity to zero percent and in red blood cell cholinesterase activity to 60 percent of their pre-exposure values (Grob and Harvey 1949, in ACGIH 1986, p. 558). The onset of the signs and symptoms of TEPP poisoning may be delayed for as long as 12 hours, and the kinds of symptoms developed depend on the route of exposure. After inhalation, respiratory and ocular effects (wheezing, laryngeal spasms, blurred vision, miosis, and tearing) are first to occur. Cyanosis may also result from inhalation exposure to TEPP. Gastrointestinal effects occur after ingestion; these include anorexia, vomiting, diarrhea, and cramps. Skin absorption leads to localized sweating and muscular twitching (Koella 1963, Taylor 1985, in Proctor, Hughes, and Fischman 1988, p. 464). Severe TEPP poisoning by any route may lead to paralysis of the respiratory muscles and death. If the dose is not lethal, complete recovery may occur, although individuals may remain sensitive to organophosphorus poisoning for some time after recovery (Taylor 1985, in Proctor, Hughes, and Fischman 1988, p. 464). Repeated exposure to small doses of TEPP may have cumulative effects and lead to the signs and symptoms described above.

Based on this evidence in humans and animals, OSHA preliminarily concludes that TEPP causes cholinesterase inhibition, which is a material impairment of health. OSHA believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these exposure-related effects. The Agency believes that establishing a PEL of 0.05 mg/m³ as an 8-hour TWA, with a skin notation, is necessary to reduce the risk that agricultural workers will experience this material health impairment. Promulgation of this limit will make the PEL for TEPP consistent across all OSHA-regulated sectors.

THALLIUM (soluble compounds)
CAS: Varies with compound; Chemical Formula: Varies with compound
H.S. No. 2159

OSHA's permissible exposure limit for soluble thallium compounds (measured as thallium) in general industry, construction, and maritime is 0.1 mg/m³; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for these substances. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 0.1 mg/m³, with a skin notation, for the soluble thallium compounds. The ACGIH TLV® is based on analogy with other highly toxic metals (ACGIH 1986, p. 569). There is no NIOSH REL, but NIOSH concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing a limit of 0.1 mg/m³ as an 8-hour TWA for soluble thallium compounds in agriculture. Promulgation of this limit will make the PEL for these substances consistent across all OSHA-regulated sectors.

Soluble thallium compounds include thallium sulfate, thallium acetate, and thallium nitrate, which are all colorless, odorless solids. Thallium sulfate is used as a poison for rodents, in semiconductor research, as a component of mineralogical solutions, in optical systems and photoelectric cells, and in low-range glass thermometers. Thallium nitrate is used as a reagent in analytical chemistry and to make green fire for signaling at sea. Thallium acetate is used in high specific gravity solutions to separate ore constituents by flotation (ACGIH 1986, p. 569; Merck 1983, p. 1325; Hawley's 1987, p. 1142).

Soluble thallium salts can cause hair loss and effects on the gastrointestinal, nervous, and circulatory systems of humans and animals. For thallium acetate, the oral LD_{50} in rats is 41 mg/kg; in mice, it is 35 mg/kg (RTECS 1991). For thallium nitrate, the oral LD_{50} in mice is 15 mg/kg (RTECS 1991). Thallium sulfate has oral LD_{50}s in rats and mice of 16 and 15 mg/kg, respectively (RTECS 1991). Animals acutely poisoned by thallium sulfate show signs and symptoms that include restlessness, ataxia, and convulsive movements followed by paresis, tremor, dyspnea, loss of weight, hemorrhagic diarrhea, and death due to respiratory failure (Hayes 1982, p. 25). The primary functional disturbances to the nervous system caused by exposure to these compounds are hypotonia and ataxia; there are also pathological changes of the central and peripheral axons of the primary neurons (Kennedy and Cavanagh 1977, in Acta Neuropathol 39(1):81–88, as cited in HSDSB 1988). Rats fed thallium acetate at dietary levels of 30 ppm showed a marked depression in growth after 30 days; at this level, the animals lost all of their hair and displayed such histological changes as atrophy of the hair follicles and sebaceous glands (Clayton and Clayton 1981, p. 1921). Young rats given oral doses of 0.1 mg thallium acetate developed bone lesions, eye lesions, keratitis, cataracts, retrobulbar neuropathy, partial optic atrophy, and hormone dysfunction (Browning 1969, p. 319). Rats died suddenly at the end of 4 months after being administered daily oral doses of 0.45 mg/kg thallium acetate; the rats showed typical hair loss at the end of 6

weeks, in addition to loss of weight and appetite (Browning, Toxic Industrial Metals 2nd Ed. 1969, p. 319). Post-mortem examination of rats receiving subacute doses of 10 to 15 mg/kg thallium acetate revealed significant changes in the kidneys, liver, and intestines (Herman and Bensch 1967, in Toxicology and Applied Pharmacology 10:199–222, as cited in Klaassen, Amdur, and Doull 1986, p. 491). Unspecified doses of orally or intravenously administered thallium sulfate caused depression of the cardiovascular and respiratory systems of dogs, cats, rats, rabbits, goats, and pigeons (Marmo et al. 1963, in Acta. Pharmacol. Sin. 4(2):119–122, in HSDB 1989 "Thallium Sulfate"). Administered to pregnant rats on days 12 to 14 of pregnancy, thallium sulfate caused fetotoxic and teratogenic effects at doses as low as 7.5 mg/kg (Toxicol. Appl. Pharmacol. 16:120, 1979). Rats exposed perinatally and postnatally to thallium sulfate in their drinking water showed modified vascular autonomic nervous system development (Rossi, Marrazzo, Berrino, DeSantis, Lisa, Susanna, Montanaro, Fici, and Marmo 1988, in Teratogenesis Carcin. Mutagen. 8(1):13–23).

In humans, exposure to the soluble thallium salts causes signs and symptoms such as nausea, vomiting, diarrhea, abdominal pain, and gastrointestinal hemorrhage (Paulson, Vergara et al. 1972, in Archives of Internal Medicine 129:100–103, as cited in Proctor, Hughes, and Fischman 1988, p. 473). These symptoms are often accompanied by peripheral neuritis, pain, weakness, paresthesias in the legs, tremor, and chest pain (Proctor, Hughes, and Fischman 1988, p. 471–472). The lowest lethal oral dose of thallium acetate in humans is estimated to be 12 mg/kg (Browning 1969, p. 317–322, as cited in Proctor, Hughes, and Fischman 1988, p. 471). The lowest toxic oral dose of thallium nitrate in humans is estimated to be 73 mg/kg, and the lowest lethal oral dose of thallium sulfate is believed to be 7 mg/kg (RTECS 1991, "Thallium nitrate," "Thallium sulfate"). A recent study (Dumitru and Kalantri 1990, in Muscle-Nerve 13(5):433–437) documents the serial conduction and electromyographic findings in a thallium-poisoned worker over a 24-month period. In this case, the plantar nerves of the foot demonstrated severe axonal loss first, and these nerves had not recovered 2 years later. Axonal loss subsequently occurred in the sural and perineal nerves as well, but they recovered within the 2-year follow-up period. A laboratory worker who had worked with thallium for 2 weeks developed acute thallium poisoning. His initial signs and symptoms included loss of consciousness, paresthesia of the finger tips and lips, malaise, and polyuria; later he developed respiratory paralysis, peripheral neuropathy, cerebellar ataxia, intellectual impairment, and cardiovascular abnormalities (Wainwright, Kox, House, Henry, Heaton, and Seed 1988, in Quarterly J. Med. 69(258):939–944). Chronic exposure to thallium salts can cause stomatitis, tremor, cachexia, polyneuropathy, alopecia, and emotional disturbance (Paulson et al. 1972, in Archives of Internal Medicine 129:100–103, as cited in Proctor, Hughes, and Fischman 1988, p. 472). Twelve of 15 workers using organic thallium salts over a period of 7½ years experienced abdominal pain, fatigue, irritability, weight loss, and leg pain; 4 of these workers experienced loss of hair (Richerson 1958, in Ind. Med. Surg. 27:607). Three of these workers had albuminuria, and one had hematuria (Richerson 1958). In cases of mild chronic thallium intoxication, lymphocytosis and eosinophilia have been observed (J.C. Munch 1934, in American Medical Association J. 102:1929, as cited in Clayton and Clayton 1981, p. 1928).

Based on this evidence in humans and animals, OSHA preliminarily concludes that soluble thallium compounds cause alopecia and effects on the nervous, gastrointestinal, and circulatory systems. OSHA therefore believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these effects. OSHA believes that the proposed limit of 0.1 mg/m^3, with a skin notation, for the soluble thallium compounds is necessary to significantly reduce these risks of material health impairment among agricultural workers. Promulgation of this limit will also make the PEL for these substances consistent across all OSHA-regulated sectors.

THIONYL CHLORIDE

CAS: 7719–09–7; Chemical Formula: Cl_2OS

H.S. No. 1393

OSHA has no limit for thionyl chloride in construction, maritime, or agriculture. The ACGIH has a TLV®-ceiling of 1 ppm for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the PEL being proposed. OSHA is proposing a 1-ppm ceiling PEL for thionyl chloride in construction, maritime, and agriculture. This is the limit recently established in general industry.

Thionyl chloride is a colorless to pale yellow liquid with a suffocating odor. Thionyl chloride is used in the manufacture of pesticides, dyes, pharmaceuticals, and engineering plastics, as a chlorinating agent, and as a solvent in high energy density lithium batteries (ACGIH 1986, p. 572, HSDB 1989).

Thionyl chloride vapors are skin, eye, and mucous membrane irritants, probably because they form sulfur dioxide and hydrogen chloride on contact with moisture (ACGIH 1986/Ex. 1–3, p. 572). An inhalation exposure of 17.5 ppm proved lethal to cats within 20 minutes (Sax 1979/Ex.1–866).

OSHA's exposure limit for thionyl chloride in general industry is based on the exposure limits for the decomposition products (hydrogen chloride and sulfur dioxide) of thionyl chloride when this substance is mixed with water. The reaction of one mole of thionyl chloride with water produces two moles of hydrogen chloride and one of sulfur dioxide, so that 1 ppm of thionyl chloride can be shown to produce a total irritant gas concentration of 3 ppm. The exposure limit for hydrogen chloride is 5 ppm as a ceiling limit; for sulfur dioxide, the limit is an 8-hour TWA of 2 ppm. Thus, OSHA believes that the proposed ceiling limit for thionyl chloride will prevent the irritant effects of its reaction products.

In construction, maritime, and agriculture, the Agency is proposing a ceiling limit of 1 ppm for thionyl chloride on the basis of analogy to the irritation potential of hydrogen chloride and sulfur dioxide and to achieve consistency in OSHA limits in all regulated sectors. OSHA preliminarily concludes that this limit will protect workers in these sectors from the significant risk of eye, skin, and mucous membranes irritation, which constitutes a material health impairment that is associated with exposure to this substance.

TRIBUTYL PHOSPHATE

CAS: 126–73–8; Chemical Formula: $(C_4H_9)_3PO_4$

H.S. No. 1402

In construction and maritime, OSHA s current PEL for tributyl phosphate is 5 mg/m^3 as an 8-hour TWA. There is no PEL in agriculture. The ACGIH has a 2.5-mg/m^3 TLV®-TWA for tributyl phosphate. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 2.5 mg/m^3 for tributyl phosphate in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Tributyl phosphate is a colorless liquid. It is used as an anti-foaming agent, a plasticizer, a hydraulic fluid, a heat-exchange medium, and in the solvent extraction of metals (RTECS 1991).

Tributyl phosphate's toxicity affects the skin, mucous membranes, lungs, and central nervous system, and this substance is also a cholinesterase inhibitor. The oral LD_{50} in rats is 3000 mg/kg, and the LC_{50} in mice is 1300 mg/m^3 for an unspecified time (RTECS 1991). A paper by Smyth and Carpenter (1944/ Ex. 1–374) reported that contact with liquid tributyl phosphate caused severe eye injury and skin irritation when tested in rabbits. Chambers and Casida (1967/Ex. 1–305) found that mice injected with 1 g/kg tributyl phosphate intraperitoneally became paralyzed. A study by Vandekar (1957/Ex. 1–498) in which mice were given tributyl phosphate by gavage revealed that a dose of 80 mg/kg resulted in a one-hour period of anesthesia, and a dose of 100 mg/kg resulted in 8 to 10 minutes of anesthesia, followed by respiratory failure and death. Administered intraperitoneally to rats, tributyl phosphate inhibited cholinesterase activity and stimulated plasma beta-glucuronidase activity (Suzuki, Kikuchi, Kato et al. 1977/Ex. 1–1170). This substance did not exhibit mutagenic activity in bacterial or fruit fly assays (Hanna and Dyer 1975/Ex. 1–485).

Nausea and headache were reported by workers exposed to levels of 15 mg/m^3 of tributyl phosphate (Mastromatteo 1964b, as cited in ACGIH 1986/Ex. 1–3, p. 591). In contact with the skin, tributyl phosphate causes irritation (HSDB 1991).

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 2.5 mg/m^3 for tributyl phosphate in construction, maritime, and agriculture. OSHA preliminarily concludes that this limit will protect workers in these sectors against the significant risk of paralysis, anesthetic effects, and skin or eye irritation, all of which constitute material impairments of health that are associated with occupational exposure to tributyl phosphate. The Agency believes the proposed PEL is necessary to substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

TRICHLOROACETIC ACID
CAS: 76–03–9; Chemical Formula: CCl_3COOH
H.S. No. 1404

In construction, maritime, and agriculture, OSHA has no PEL for trichloroacetic acid. The ACGIH has an 8-hour TLV®–TWA of 1 ppm for this substance. There is no NIOSH REL. In construction, maritime, and agriculture, OSHA is proposing a PEL of 1 ppm as an 8-hour TWA, and NIOSH concurs (Ex. 8–47, Table N1) that this limit is appropriate. This is the limit recently established for trichloroacetic acid in general industry.

Trichloroacetic acid is used in medicine, as a decalcifier and fixative in microscopy, and as a chemical intermediate (HSDB 1991). The oral LD_{50} for trichloroacetic acid in rats is 5 g/kg (RTECS 1991). Studies on mice conducted by NIOSH (1984, as cited in ACGIH 1986/Ex. 1–3, p. 592) established that the oral LD_{50} for this species is 4.97 g/kg, and that a 500-mg/kg dose was fatal when administered intraperitoneally. In contact with the skin of rabbits, trichloroacetic acid caused mild irritation; instilled into the eyes, it caused severe irritation (RTECS 1991).

Although corrosive, trichloroacetic acid is not readily absorbed through the skin (ACGIH 1986/Ex. 1–3, p. 592). Contact of the skin with this substance for 1 hour or more causes burns; splashed into the eye, it causes severe pain and may cause permanent injury (HSDB 1991).

Based on this evidence, OSHA is proposing an 8-hour TWA limit of 1 ppm for trichloroacetic acid in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors from the significant risk of skin and eye irritation, which are material impairments of health that are associated with occupational exposure to this substance. OSHA believes that the proposed limit will substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

1,1,2-TRICHLOROETHANE
CAS: 79–00–5; Chemical Formula: $CHCl_2CH_2Cl$
H.S. No. 2162

In general industry, construction, and maritime, OSHA has an 8-hour TWA of 10 ppm, and a skin notation, for 1,1,2-trichloroethane. The Agency has no PEL for this substance in agriculture. The ACGIH TLV®–TWA for 1,1,2-trichloroethane, which was set by analogy to chloroform, is 10 ppm, with a skin notation. Although NIOSH considers this substance a potential human carcinogen, NIOSH concurs (Ex. 8–47) with the limit being proposed by OSHA for this substance. The Agency is proposing an 8-hour TWA PEL of 10 ppm, and a skin notation, for 1,1,2-trichloroethane in agriculture. Promulgation of this limit will make the PEL for 1,1,2-trichloroethane consistent across all OSHA-regulated sectors.

1,1,2-Trichloroethane is a colorless, nonflammable liquid with a pleasant, sweet odor. It is used as a chemical intermediate in the production of 1,1-dichloroethylene (vinylidene chloride) and as a solvent for fats, resins, adhesives, chlorinated rubbers, polyesters, and waxes (ACGIH 1986, p. 596; HSDB 1989).

In animals, exposure to 1,1,2-trichloroethane causes central nervous system depression and damage to the liver and kidneys. This substance is also a mild irritant of the eyes, mucous membranes, and skin; in mice, it is a liver carcinogen. The oral LD_{50} in rats is 830 mg/kg, and the lowest lethal concentration in the same species is 2000 ppm for 4 hours; the dermal LD_{50} in rabbits is 5377 mg/kg (RTECS 1989). Although 1,1,2-trichloroethane is not a severe skin irritant, it causes defatting of the skin on repeated contact. Absorption through the skin has been shown to cause toxic effects, including liver and kidney injury, in both rabbits and guinea pigs (Jakobson, Holmberg, and Wahlberg 1977, in Clayton and Clayton 1981, p. 3511). Instilled into the eyes of rabbits, this substance caused a mild degree of irritation (RTECS 1989). Half of the female rats exposed to a 250-ppm concentration of 1,1,2-trichloroethane for 7 hours died and showed marked liver and kidney damage at autopsy; when the exposure period was shortened to 4 hours, the animals survived the exposure but still showed liver and kidney necrosis at autopsy. Rats exposed to 250 ppm for 2 hours, however, did not exhibit liver or kidney damage at autopsy (Clayton and Clayton 1981, p. 3511). Animals dosed orally also demonstrated liver and kidney damage at autopsy (Gehring 1968; Watrous and Plaa 1972, in Clayton and Clayton 1981, p. 3511). A significant increase in hepatocellular carcinomas occurred in mice given 195 or 390 mg/kg/day 1,1,2-trichloroethane by gavage for 78 weeks. The incidence of adrenal pheochromocytomas was also increased in the high-dose female mice. No statistically significant increase in the incidence of tumors was seen in rats given up to 92 mg/kg/day 1,1,2-trichloroethane (National Cancer Institute 1978, in Proctor, Hughes, and Fischman 1988, p. 486). The International Agency for Research on Cancer has concluded that the evidence for the carcinogenicity of 1,1,2-trichloroethane in animals is limited (IARC 1987, Suppl. 7, p. 73).

In humans, exposure to low (not further specified) concentrations of 1,1,2-trichloroethane has narcotic effects and causes eye and mucous membrane irritation (IARC 1979, Vol. 20, p. 540). Chronic exposure causes fat deposition in the kidney and lung damage (IARC 1979, Vol. 20, p. 540). Based on animal data, the probable lethal oral dose of 1,1,2-trichloroethane in humans is estimated to be 50 to 500 mg/kg (Parmeggiani 1983, p. 2214). Prolonged or repeated contact of the skin with this substance causes defatting and may cause dermatitis (Clayton and Clayton 1981, pp. 3510–3513).

Based on this evidence, OSHA preliminarily concludes that exposure to 1,1,2-trichloroethane potentially causes central nervous system depression and liver and kidney damage. OSHA believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these effects. OSHA believes that establishing a PEL of 10 ppm as an 8-hour TWA, with a skin notation, is necessary to reduce the risk of these material health impairments. Promulgation of this limit will also make OSHA's PEL for this substance consistent across all OSHA-regulated sectors.

TRIMETHYLAMINE

CAS: 75–50–3; Chemical Formula: $(CH_3)_3N$

H.S. No. 1411

In construction, maritime, and agriculture, OSHA has no PEL for trimethylamine. The ACGIH has a 10-ppm TLV®–TWA and a 15–ppm TLV®–STEL. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limits being proposed. OSHA is proposing PELs of 10 ppm as an 8-hour TWA and 15 ppm as a 15-minute STEL in construction, maritime, and agriculture. These are the limits recently established for trimethylamine in general industry.

Trimethylamine has a pungent, fishy odor and is a gas at room temperature. Trimethylamine is used as an insect attractant, a warning agent for natural gas, a flotation agent, in organic synthesis, and in the manufacture of quaternary ammonium compounds (ACGIH 1986, p. 607; Merck 1983, p. 1388).

Trimethylamines are irritants of the eyes, mucous membranes, and respiratory tract. The lowest lethal concentration in rats is 3500 ppm for 4 hours (RTECS 1991). The damage done to the eyes of animals when trimethylamine is instilled into the conjunctival sac ranges from severe irritation to permanent damage, depending on the strength of the solution (Grant 1986, p. 952). Contact of the skin or eyes with this substance also causes burns and irritation in humans (Sittig 1985, p. 896). A student splashed in the eye with trimethylamine showed loss of the corneal epithelium; however, 4 weeks later, recovery was complete (Grant 1986, p. 952).

In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA limit of 10 ppm and a 15-minute STEL of 15 ppm for trimethylamine. The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant risk of eye, mucous membrane, and upper respiratory tract irritation, which constitute material impairments of health that are caused by occupational exposure to this substance. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

n-VALERALDEHYDE

CAS: 110–62–3; Chemical Formula: $CH_3(CH_2)_3CHO$

H.S. No. 1420

In construction, maritime, and agriculture, OSHA has no limit for n-valeraldehyde. The ACGIH TLV®–TWA is 50 ppm. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the proposed PEL. OSHA is proposing a PEL of 50 ppm as an 8-hour TWA for n-valeraldehyde in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

Valeraldehyde is a colorless liquid that is used as a flavoring agent and rubber accelerator, and in resin chemistry (ACGIH 1986, p. 619).

n-Valeraldehyde's toxic effects include both skin and eye irritation. Animal studies showed n-valeraldehyde to be severely irritating when applied to guinea pig skin and to rabbits' eyes (Fassett, as cited in ACGIH 1986/Ex. 1–3, p. 619). The dermal LD_{50} in guinea pigs exceeds 20ml/kg (Fassett, as cited in ACGIH 1986/Ex. 1–3, p. 619). A series of studies of the relative acute inhalation toxicity of 13 aliphatic saturated and unsaturated aldehydes in mice, guinea pigs, and rabbits showed that valeraldehyde was relatively nontoxic systemically (Salem and Cullumbine 1960/Ex. 1–360).

In construction, maritime, and agriculture, OSHA is proposing a 50-ppm 8-hour TWA limit for n-valeraldehyde. The Agency preliminarily concludes that this limit will protect workers in these sectors from the significant risk of severe eye and skin irritation associated with occupational exposure to this substance. OSHA believes that this limit will substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for valeraldehyde consistent across all regulated sectors.

m-XYLENE ALPHA, ALPHA'-DIAMINE

CAS: 1477–55–0; Chemical Formula: $C_6H_4(CH_2NH_2)_2$

H.S. No. 1432

In construction, maritime, and agriculture, OSHA has no exposure limit for this substance. The ACGIH has a TLV®-ceiling of 0.1 mg/m³ and has added a skin notation to indicate that substantial percutaneous absorption can occur through the eyes, mucous membranes, and skin. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the proposed limit. OSHA is proposing a PEL of 0.1mg/m³ as a ceiling for m-xylene alpha, alpha'-diamine in construction, maritime, and agriculture. This is the limit recently established for this substance in general industry.

m-Xylene alpha, alpha'-diamine (MXDA) is a colorless liquid. This substance is used as a curing agent for epoxy resins, as a source of m-Xylene diisocyanate, and in the manufacture of plastics, textiles, and rubber (ACGIH 1986, p. 638; Hazardous Substance Fact Sheet 1986, p. 1).

Animal studies have demonstrated that MXDA is strongly irritating to the skin. When applied to the skin of rabbits, MXDA caused severe irritation; 50 mg applied to the eyes of rabbits also caused severe injury (RTECS 1991). The oral LD_{50} in rats is 930 mg/kg, and the LC_{50} in the same species is 700 ppm for 1 hour (RTECS 1991). The dermal LD_{50} in rabbits is 2 g/kg (RTECS 1991). Rats exposed to concentrations of MXDA ranging from 1.74 to 6.04 mg/liter even for one hour sustained liver, kidney, and lung damage, as determined at necropsy. One study showed mild sensitization when MXDA was applied to guinea pig skin, but this effect was not observed in a second study (Sherwin-Williams Company 1978, as cited in ACGIH 1986/Ex. 1–3, p. 638).

Based on this evidence, OSHA preliminarily concludes that a ceiling limit of 0.1 mg/m³ and a skin notation are necessary to protect workers in construction, maritime, and agriculture from the significant risk of skin irritation, percutaneous absorption, and the potential systemic effects associated with exposure to MXDA. The Agency considers these effects material impairments of health. OSHA believes that the proposed limit will substantially reduce this significant risk. In addition, promulgation of this limit will make OSHA's PEL for MXDA consistent across all regulated sectors.

XYLIDINE
CAS: 1300–73–8; Chemical Formula: $(CH_3)_2C_6H_3NH_2$
H.S. No. 1433

In construction and maritime, OSHA's limit for xylidine is 5 ppm as an 8-hour TWA, with a skin notation. There is no PEL in agriculture. The ACGIH has a TLV®–TWA of 2 ppm, also with a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. In construction, maritime, and agriculture, OSHA is proposing an 8-hour TWA PEL of 2 ppm, and a skin notation, for xylidine. This is the limit recently established for this substance in general industry.

Xylidine is a pale yellow to brown liquid. Commercial xylidine is a mixture of isomers. Xylidine is primarily used in the manufacture of dyes. It is also used in organic synthesis and pharmaceuticals (Merck 1983, p. 1448; ACGIH 1986, p. 639 (88)).

Several studies indicate that the current PEL for xylidine in construction and maritime is insufficient to protect workers against hepatotoxic and other adverse effects, and agricultural workers are currently without any protection against exposure. A paper by von Oettingen, Neal, Sievers et al. (1947, as cited in ACGIH 1986/Ex. 1–3, p. 639) reported liver damage in dogs, rats, cats, and mice repeatedly exposed to 45 ppm xylidine for seven hours per day for a period of 20 to 40 weeks; these exposures also caused death in dogs, cats, and mice. Treon, Sigmon, Wright et al. (1950/Ex. 1–533) noted cardiac, liver, and kidney damage in autopsies of animals fatally exposed at the following doses: cats, 17 ppm; guinea pigs, 50 ppm; and rabbits, 60 ppm; cyanosis was also observed in these animals.

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 2 ppm, and a skin notation, for xylidene in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors from the significant risk of exposure-related cardiac, kidney, and liver damage, all of which constitute material health impairments. OSHA believes the proposed PEL is necessary to substantially reduce these risks. In addition, promulgation of this limit will make OSHA's PEL for xylidine consistent across all regulated sectors.

Preliminary Conclusions for This Group of Substances

Based on the evidence described above, OSHA preliminarily concludes that exposure to any of the substances included in this category places workers in construction, maritime, and agriculture at significant risk of material health impairment and functional incapacity. The adverse health consequences of exposure to these chemicals include neuropathies, skin and respiratory tract irritation, kidney and liver damage, and gastrointestinal disorders, all of which constitute material health impairments within the meaning of the Act. OSHA preliminarily concludes, based on the toxicological evidence, that the new or revised limits being proposed for these hazardous substances will substantially reduce these significant occupational risks for workers in these sectors. In addition, promulgation of the proposed limits will make OSHA's PELs for these substances consistent across all regulated sectors.

12. Substances for Which Proposed Limits Are Based on Avoidance of Biochemical/Metabolic Effects

Introduction

One basis for establishing exposure limits is to prevent toxic substances from interfering with the normal metabolism or biochemistry of the body. A total of 31 substances in this rulemaking belong in the category of biochemical or metabolic toxins. Table C12–1 shows these substances and their CAS and HS numbers. Table C12–1 also shows the current PELs for these substances in construction and maritime, the ACGIH TLV®s (1987–1988), the NIOSH RELs (where these apply), and the limits OSHA is proposing to establish in construction, maritime, and agriculture. Promulgation of these PELs will make OSHA's limits for these biochemical/metabolic toxins consistent across all OSHA-regulated sectors.

Description of the Health Effects

The compounds shown in Table C12–1 are further divided into the following sub-classes, based on their mechanism of action:

- Substances that are cholinesterase inhibitors;
- Substances that interfere with the oxygen-carrying capacity of the blood;
- Substances with Antabuse-like effects.

TABLE C12–1.—SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED PRIMARILY ON AVOIDANCE OF METABOLIC EFFECTS

H.S. No./chemical name	CAS No.	Current OSHA PEL in construction and maritime*	1978–1988 ACGIH TLV®*	NIOSH REL***	Proposed OSHA PEL in construction, maritime, and agriculture*
1025 Aniline and homologs	62–53–3	5 ppm TWA, Skin	2 ppm TWA, Skin		2 ppm TWA, Skin
1058 Calcium cyanamide	156–62–7		0.5 mg/m³ TWA		0.5 mg/m³ TWA
1066 Carbofuran	1563–66–2		0.1 mg/m³ TWA		0.1 mg/m³ TWA
1069 Carbon dioxide	124–38–9	5,000 ppm TWA	5,000 ppm TWA	10,000 ppm TWA	10,000 ppm TWA
			30,000 ppm STEL	30,000 ppm Ceiling (10-min)	30,000 ppm STEL
1071 Carbon monoxide	630–08–0	50 ppm TWA	50 ppm TWA	35 ppm TWA (8-hour).	35 ppm TWA
			400 ppm STEL	200 ppm Ceiling	200 ppm Ceiling
1091 Chlorpyrifos	2921–88–2		0.2 mg/m³ TWA		0.2 mg/m³ TWA, Skin
			0.6 mg/m³ STEL, Skin.		
1103 Crufomate	299–86–5		5 mg/m³ TWA		5 mg/m³ TWA
			20 mg/m³ STEL		
1104 Cyanamide	420–04–2		2 mg/m³ TWA		2 mg/m³ TWA
2051 Dichlorvos (DDVP)	62–73–7	1 mg/m³ TWA, Skin	0.1 ppm (1 mg/m³) TWA, Skin.		1 mg/m³ TWA, Skin
1131 Dicrotophos	141–66–2		0.25 mg/m³ TWA, Skin.		0.25 mg/m³ TWA, Skin
1143 Dimethylaniline	121–69–7	5 ppm TWA, Skin	5 ppm TWA		5 ppm TWA
			10 ppm STEL, Skin		10 ppm STEL, Skin
1146 Dioxathion	78–34–2		0.2 mg/m³ TWA, Skin.		0.2 mg/m³ TWA, Skin
1151 Disulfiram (Antabuse)	97–77–8		2 mg/m³ TWA		2 mg/m³ TWA

TABLE C12-1.—SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED PRIMARILY ON AVOIDANCE OF METABOLIC EFFECTS—Continued

H.S. No./chemical name	CAS No.	Current OSHA PEL in construction and maritime*	1978–1988 ACGIH TLV®**	NIOSH REL***	Proposed OSHA PEL in construction, maritime, and agriculture*
1160 Ethion	563-12-2		0.4 mg/m³ TWA, Skin.		0.4 mg/m³ TWA, Skin
1173 Fenamiphos	22224-92-6		0.1 mg/m³ TWA, Skin.		0.1 mg/m³ TWA, Skin
1174 Fensulfothion	115-90-2		0.1 mg/m³ TWA		0.1 mg/m³ TWA
1175 Fenthion	55-38-9		0.2 mg/m³ TWA, Skin.		0.2 mg/m³ TWA, Skin
1245 Methomyl	16752-77-5		2.5 mg/m³ TWA		2.5 mg/m³ TWA
1280 Monomethylaniline	100-61-8	2 ppm TWA, Skin	0.5 ppm TWA, Skin		0.5 ppm TWA, Skin
2116 Nitric oxide	10102-43-9	25 ppm TWA	25 ppm TWA	25 ppm TWA	25 ppm TWA
2117 Nitrobenzene	98-95-3	1 ppm TWA, Skin	1 ppm TWA, Skin		1 ppm TWA, Skin
1288 p-Nitrochlorobenzene	100-00-5	1 mg/m³ TWA, Skin	0.5 mg/m³ TWA, Skin.		1 mg/m³ TWA, Skin
2122 Parathion	56-38-2	0.1 mg/m³ TWA, Skin.	0.1 mg/m³ TWA, Skin.	0.05 mg/m³ TWA	0.1 mg/m³ TWA, Skin
1319 Phorate	298-02-2		0.05 mg/m³ TWA 0.2 mg/m³ STEL, Skin.		0.05 mg/m³ TWA 0.2 mg/m³ STEL, Skin
2134 Propane	74-98-6				See text
1337 Propoxur	114-26-1		0.5 mg/m³ TWA		0.5 mg/m³ TWA
1349 Ronnel	299-84-3	15 mg/m³ TWA	10 mg/m³ TWA		10 mg/m³ TWA
1380 Sulprofos	35400-43-2		1 mg/m³ TWA		1 mg/m³ TWA
1384 Terphenyls	26140-60-3	1 ppm Ceiling	0.5 ppm Ceiling		0.5 ppm Ceiling
1401 m-Toluidine	108-44-1		2 ppm TWA, Skin		2 ppm TWA, Skin
1413 2,4,6-Trinitrotoluene	118-96-7	1.5 mg/m³ TWA, Skin.	0.5 mg/m³ TWA, Skin.		0.5 mg/m³ TWA, Skin
2168 Warfarin	81-81-2	0.1 mg/m³ TWA	0.1 mg/m³ TWA		0.1 mg/m³ TWA

* OSHA's PELs do not currently apply in Agriculture; OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TLV®-TWA is for an 8-hour exposures; its STELs are 15-minutes limits not to be exceeded more than 4 times in any working day, with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10 hour/day, 40 hour/week exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

The disruption of metabolic processes by toxic substances, if severe enough, results in potentially dangerous effects on the neurological, cardiovascular, and respiratory systems. The adverse health consequences caused by exposure to chemicals having cholinesterase inhibition effects range from wheezing, nausea, vomiting, and confusion to respiratory failure, coma, and death. Table C12–2 shows the primary biochemical/metabolic effects associated with occupational exposure to the substances in this group of toxins. If exposure has localized rather than systemic effects, the signs and symptoms of cholinesterase inhibition can include sweating, blurred vision, and constriction of the bronchial tubes. Substances that interfere with the ability of the blood to carry oxygen cause a broad range of symptoms, including fainting, loss of consciousness, rapid heartbeat, headache, nausea, coma, and death. Carbon monoxide (CO) is the best-known substance in this category of chemicals, and exposure to CO is common throughout the construction, maritime, and agriculture industries.

The Antabuse-like effects associated with exposure to three of the chemicals in this group—disulfiram, cyanamide, and calcium cyanamide—include facial flushing, nausea, and a racing heartbeat. However, these effects are manifested only if the exposed individual has ingested alcohol. The three chemicals in this subgroup cause this effect by inhibiting aldehyde dehydrogenase activity, which is involved in the biotransformation of alcohol.

TABLE C12—2. BIOCHEMICAL/METABOLIC EFFECTS ASSOCIATED WITH EXPOSURE TO THESE SUBSTANCES

H.S. No./chemical name	CAS No.	Biochemical/metabolic effect
1025 Aniline & homologs	62-53-3	Methemoglobinemia.
1058 Calcium cyanamide	156-62-7	Antabuse-like effect.
1068 Carbofuran	1563-66-2	Cholinesterase inhibition.
1069 Carbon dioxide	124-38-9	Hyperventilation.
1071 Carbon monoxide	630-08-0	Carboxyhemoglobinemia.
1091 Chlorpyrifos	2921-88-2	Cholinesterase inhibition.
1103 Crufomate	299-86-5	Cholinesterase inhibition.
1104 Cyanamide	420-04-2	Antabuse-like effect.
2051 Dichlorvos (DDVP)	62-73-7	Cholinesterase inhibition.
1131 Dicrotophos	141-66-2	Cholinesterase inhibition.
1143 Dimethylaniline	121-69-7	Methemoglobinemia.
1146 Dioxathion	78-34-2	Cholinesterase inhibition.
1151 Disulfiram (Antabuse)	97-77-8	Antabuse effect.
1160 Ethion	563-12-2	Cholinesterase inhibition.
1173 Fenamiphos	22224-92-6	Cholinesterase inhibition.
1174 Fensulfothion	115-90-2	Cholinesterase inhibition.

TABLE C12—2. BIOCHEMICAL/METABOLIC EFFECTS ASSOCIATED WITH EXPOSURE TO THESE SUBSTANCES—Continued

H.S. No./chemical name	CAS No.	Biochemical/metabolic effect
1175 Fenthion	55-38-9	Cholinesterase inhibition.
1245 Methomyl	16752-77-5	Cholinesterase inhibition.
1260 Monomethylaniline	100-61-8	Methemoglobinemia.
2116 Nitric oxide	10102-43-9	Methemoglobinemia.
2117 Nitrobenzene	98-95-3	Methemoglobinemia.
1288 p-Nitrochloro-benzene	100-00-5	Methemoglobinemia.
2122 Parathion	56-38-2	Cholinesterase inhibition.
1319 Phorate	298-02-2	Cholinesterase inhibition.
1337 Propoxur	114-26-1	Cholinesterase inhibition.
1349 Ronnel	299-84-3	Cholinesterase inhibition.
1380 Sulprofos	35400-43-2	Cholinesterase inhibition.
1384 Terphenyls	26140-60-3	Mitochondrial changes.
1401 m-Toluidine	108-44-1	Methemoglobinemia.
1413 2,4,6-Tri-nitrotoluene	118-96-7	Methemoglobinemia.
2168 Warfarin	81-81-2	Capillary fragility.

For chemicals that cause systemic toxicity in animals and/or humans, the grossly observable signs and symptoms of intoxication are usually secondary to the interaction of the chemical with a molecular target. In other words, the chemical interacts with (binds with or modifies) an endogenous molecular constituent (protein, nucleic acid, lipid, etc.) in the target tissue. The result of the interaction is ordinarily a modification or elimination of the normal function of the specific molecular constituent which, if sufficiently severe, may lead to secondary effects within the affected cells and/or tissues. It is possible for a number of molecules to be affected by the toxic chemical without there being any overt manifestation of toxicity. In other words, there is an apparent no-effect level governing the overt manifestation of toxicity, although there are usually metabolic effects at levels below those that cause overt effects.

For chemicals for which the molecular target is known and for which methods are available to detect the altered molecular target, it is possible to use the measure of altered biochemical function as a sensitive indicator of exposure to the chemicals at levels below those that cause grossly observable signs and symptoms of poisoning. For other classes of chemicals, studies in animals and/or humans have shed light on the biochemical basis of their toxicity. For some of these classes of chemicals, it is possible to base limits for human exposure on biochemical, metabolic, or pharmacologic indicators of their interaction with molecular targets rather than on grossly visible signs and symptoms of adverse systemic effects.

Substances that are cholinesterase inhibitors. A number of organophosphate and carbamate insecticides produce acute toxicity in humans through inhibition of acetylcholinesterase at cholinergic synapses in the central and peripheral nervous systems. Many of the substances in this group have this effect. This inhibition causes an accumulation of acetylcholine at the effector sites and elicits signs and symptoms consistent with excessive cholinergic activity. These include bronchoconstriction; increased bronchial secretions, salivation, and lacrimation; nausea; vomiting; cramps; constriction of the pupils; muscular weakness; and cardiac irregularities. If sufficiently severe, acetylcholinesterase inhibition may cause coma, irreversible CNS damage, and death.

The mechanisms by which carbamates and organophosphates inhibit acetyl-cholinesterase differ. In general, carbamates form a noncovalently bound complex with the enzyme, while most organophosphates bind covalently with the enzyme. The net result, inactivation of the enzyme, is similar for both groups. In either case, the inhibition is usually reversible. The carbamate-cholinesterase complex dissociates to regenerate the active enzyme, while cholinesterase inactivated by organophosphates is replaced by the *de novo* synthesis of active enzyme. Therefore, unless the inhibition is sufficiently severe to cause brain damage or death, the manifestations of acute toxicity are reversible, and poisoned individuals recover without sequelae. A significant proportion of endogenous cholinesterase activity may be inhibited before the overt manifestations of intoxication appear. The fraction of total cholinesterase activity that can be inhibited without the development of signs and symptoms of toxicity varies from individual to individual and also appears to depend on the intensity and duration of exposure. The lack of warning signs at low levels of exposure increases the need to set exposure limits at levels that will protect those individuals who do not readily manifest the symptoms and signs of toxicity from experiencing the subclinical effects of exposure.

Substances that interfere with the oxygen-carrying capacity of the blood. Several compounds in this section produce their immediate toxicity in humans by altering the ability of hemoglobin in the red blood cells to bind, transport, and release oxygen. Perhaps the best studied of these is carbon monoxide. Carbon monoxide binds to hemoglobin with a greater affinity than does oxygen. It also alters the dissociation characteristics for the oxygen-hemoglobin complex. The overall effect is to reduce the oxygen-carrying capacity of the blood. Also included in this overall category of compounds is a group of aromatic amines and nitro compounds that react with hemoglobin in the blood to reduce it to methemoglobin. Methemoglobin will not bind with oxygen and therefore is not an effective carrier of oxygen.

Because these compounds reduce the ability of the blood to transport oxygen, the overt signs and symptoms of acute toxicity are those of tissue anoxia, i.e., neurobehavioral disturbances, dizziness, cardiac irregularities, cyanosis, unconsciousness, and death. The severity of the symptoms is a function of the degree to which the oxygen-carrying capacity of the blood has been depleted and of the state of the exposed individual's health. In the case of carbon monoxide, individuals with pre-existing cardiovascular disease or healthy individuals engaged in physical labor may be placed at increased risk when more than 5 percent of their hemoglobin is bound to carbon monoxide.

In the cases of both carbon monoxide and the methemoglobin-forming compounds, the primary effect (i.e., formation of carboxyhemoglobin or methemoglobin) is reversible. In the absence of additional carbon monoxide

exposure, carboxyhemoglobin dissociates to carbon monoxide and fully functional hemoglobin. Methemoglobin can be reoxidized to hemoglobin by endogenous mechanisms, but the major recovery mechanism is via the synthesis of new hemoglobin.

Substances with Antabuse-like effects. The ingestion of alcoholic beverages following exposure to disulfiram, cyanamide, or calcium cyanamide results in a characteristic syndrome consisting of flushing of the face, nausea, vomiting, hypotension, and increased heart rate. If exposure is particularly severe, the reaction may trigger convulsions, cardiac arrhythmias, or heart attacks; in some cases, such exposures have caused death. In the vast majority of less severe cases, the reaction is fully reversible, although the symptoms are temporarily completely disabling. Disulfiram (Antabuse) is used therapeutically in the treatment of chronic alcoholism; employees who are currently being treated with disulfiram for alcoholism are therefore at particularly high risk if they are also occupationally exposed to these substances that cause Antabuse-like effects. These compounds do not cause any signs or symptoms of toxicity in the absence of alcohol ingestion unless exposure levels are far above those that trigger the alcohol-induced response.

Dose-Response Relationships and Biochemical/Metabolic Effects

Substances that are cholinesterase inhibitors. Typically, the cholinesterase inhibition potential of a compound is assessed by measuring plasma cholinesterase activity in the treated organism. Data from experiments in animals and limited data from human clinical trials indicate that the percentage of basal plasma cholinesterase activity decreases with increasing dose and that the dose-response curve is S-shaped. Because there is interindividual variation in this relationship, the dose-response curve for a population exposed to a cholinesterase inhibitor would be expected to be much shallower in slope and to have longer tails than the dose-response curve for any single individual.

The relationship between the dose-response curve for plasma cholinesterase inhibition and the dose-response curves for more direct indicators of clinical intoxication, such as acetyl cholinesterase activity in the CNS or the actual appearance of signs of intoxication, is not known. Evidence suggests that there is considerable interindividual variability in these relationships. Some individuals may be free of the symptoms and signs of intoxication when their plasma cholinesterase levels have been inhibited by as much as 90 percent, while others may experience symptoms after only a small decrease in plasma cholinesterase activity. Because of this wide variation in response, any exposure limit should be set with individual variability in mind.

Substances that interfere with oxygen transport. Both carboxyhemoglobin and methemoglobin formation exhibit a classical sigmoidal dose-response relationship in relation to exposure to carbon monoxide or methemoglobin-forming compounds. The loss in the oxygen-carrying capacity of the blood is a function of the intensity and duration of exposure. As stated above, the majority of healthy individuals can tolerate some reduction in the oxygen-carrying capacity of their blood without experiencing symptoms of overt toxicity. However, there is great interindividual variability in the degree of decreased oxygen-carrying capacity that can be tolerated without apparent ill effect. Individuals with pre-existing anemia or with high carboxyhemoglobin levels as a result of other environmental exposures (e.g., smoking) may already be at or above the level at which they will display the signs or experience the symptoms of tissue anoxia. For these individuals, even a small incremental decrease in the oxygen-carrying capacity of the blood can have serious consequences.

Substances causing Antabuse-like effects. The dose-response characteristics of disulfiram, cyanamide, and calcium cyanamide follow the usual S-shaped curve. The proposed rule's limits for the substances in this group have been set at levels below those associated with the Antabuse effect in workers ingesting alcohol either during or after work.

The following paragraphs describe the Agency's preliminary findings with respect to these substances that cause biochemical/ metabolic disturbances. The discussions below also illustrate the risk of material health impairment associated with exposure to these substances.

ANILINE (AND HOMOLOGS)
CAS: 62–53–3; Chemical Formula: $C_6H_5NH_2$
H.S. No. 1025

OSHA's 8-hour TWA permissible exposure limit for aniline in construction and maritime is 5 ppm, with a skin notation. There is no limit in agriculture. The ACGIH-recommended 8-hour TLV®–TWA is 2 ppm, with a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N6A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 2 ppm, and a skin notation, for aniline in construction, maritime, and agriculture; this is the limit recently established in general industry.

When first distilled, aniline is an oily, colorless liquid that darkens on exposure to air; it has a characteristic odor. Aniline and its homologs are used in the manufacture of resins, varnishes, perfumes, shoe blacks, solvents, vulcanizing agents, inks, paint removers, explosives, herbicides, dyes, lacquers, wood stains, corrosion inhibitors, and photographic chemicals. Aniline also finds use as a chemical intermediate and laboratory reagent (HSDB 1990; ACGIH 1936, p. 30). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Aniline causes methemoglobinemia on acute exposure and anemia, weight loss, and cutaneous lesions on chronic exposure. Aniline also causes cancer in animals. The LC_{50} in mice is 175 ppm for 7 hours, and the lowest toxic concentration in rats is 250 ppm for 4 hours (1990). The dermal LD_{50} in rabbits is 820 mg/kg (RTECS 1990). Rats were given single oral doses of aniline (500, 622.9, 775.9, 866.0, 966.6, 1204.1, or 1500.0 mg/kg) in a recent acute toxicity study (EPA 1986, Doc. #86–870001696, OTS). Within 14 days of dosing, 1 rat in the 622.9 mg/kg group, 6 rats in the 775.9 mg/kg group, 7 rats in the 866.0 mg/kg group, 9 rats in the 1204.1 mg/kg group, and all rats in the 1500 mg/kg group had died. Oral LD_{50}s of 930 mg/kg for males and 780 mg/kg for females were established in this study. Acutely poisoned animals showed cyanosis, tremors, tachypnea, lacrimation, and coma before death. Rats exposed to 5 ppm of aniline for 6 months showed a slight increase in methemoglobinemia (Oberst, Hackley, and Comstock 1956/ Ex. 1–685). Guinea pigs, dogs, and mice exposed to aniline on the same regimen showed no effects (Oberst, Hackley, and Comstock 1956/Ex. 685). Rats exposed chronically to aniline hydrochloride developed spleen hemangiosarcomas; dietary levels were 10, 30, or 100 mg/kg. Treated rats showed splenic tumors and fibrosarcomas and sarcomas of multiple body organs (NCI1 1987a/Ex. 1–1118).

Occupational aniline poisoning was a relatively common occurrence in earlier years (ACGIH 1986/Ex. 1–3, p. 30). The early limits for aniline were set to guard against acute toxicity manifested as cyanosis (Henderson and Haggard 1943i, as cited in ACGIH 1986/Ex. 1–3, p. 30).

Cirrhosis and chronic CNS effects were also reported in early studies (Holstein 1955/Ex. 1–913; von Oettingen 1941/Ex. 1–874). Skin absorption occurs when aniline vapor contacts the skin (Dutkiewicz 1962, as cited in ACGIH 1986/Ex. 1–3, p. 30). Early studies suggested that less than full-shift exposure to concentrations of aniline vapor ranging from 7 to 53 ppm caused mild symptoms, while one-hour inhalation exposures to concentrations in the range of 100 to 160 ppm caused severe effects (Henderson and Haggard 1943i, as cited in ACGIH 1986/Ex. 1–3, p. 30). A NIOSH-sponsored study conducted in 1986 reports that altering the workshift from five 8-hour days to four 12-hour days increased the toxicity associated with exposure to aniline at 50 or 150 ppm (Kim, YC and Carlson GP 1986, in Fund. Appl. Toxicol. 7(1):144–152). Aniline-induced methemoglobinemia was cumulative in both 8-hour and 12-hour workers exposed to 50 ppm, but this effect was more severe in those working the longer shift. Methemoglobin levels in the blood returned to normal after overnight recovery in the 8-hour group but did not recover in the workers working the 12-hour shift. Two poisoning incidents attributable to aniline absorption through the skin have been reported; in both cases, the onset of symptoms was delayed for 2 to 4 hours. Both victims became weak, developed headaches, became deeply cyanotic, and collapsed; after treatment, recovery was complete (Gosselin, Smith, and Hodge 1984, p. III–32).

Based on this evidence, OSHA preliminarily concludes that aniline causes methemoglobinemia in humans by inhalation and skin absorption. Accordingly, OSHA is proposing an 8-hour TWA of 2 ppm for aniline, and a skin notation, in construction, maritime, and agriculture. The Agency believes that this limit will protect workers in these sectors from the significant occupational risk of methemoglobinemia, which constitutes a material impairment of health. Promulgation of this limit will also make OSHA's PEL for aniline consistent across all regulated sectors.

CALCIUM CYANAMIDE

CAS: 156–62–7; Chemical Formula: CaNC=N

H.S. No. 1058

OSHA has no limit for calcium cyanamide in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 0.5 mg/m³ for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed, which is 0.5 mg/m³ as an 8-hour TWA. This is the limit recently established for calcium cyanamide in general industry.

Calcium cyanamide is a white-to-gray, crystalline solid. Calcium cyanamide is used as a fertilizer, an herbicide, and a defoliant for cotton plants; it also finds use as a chemical intermediate (Clayton and Clayton 1981, p. 4856). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Calcium cyanamide causes parasympathetic overactivity in overexposed humans and animals; in humans, exposure may cause intense cutaneous and mucosal flushing, headache, vertigo, tachypnea, hypotension, and profound shock (Gosselin, Smith, and Hodge 1984, p. II–351). In rats, the oral LD_{50} is 158 mg/kg (RTECS 1990). Skin and eye irritation have been reported in rats and rabbits, with significant irritation occurring when 100 mg of calcium cyanamide is placed directly into the eyes of rabbits (Martin 1975, as cited in ACGIH 1986/Ex. 1–3, p. 91). Severe skin irritation developed in rabbits when a paste of this substance was applied to the shaved abdominal skin for 24 hours (Martin 1975, as cited in ACGIH 1986/Ex. 1–3, p. 91). Two of five animals died when the dose was 10 g/kg, but all survived a dose of 5 g/kg.

Most cases of industrial calcium cyanamide poisoning involve primary skin irritation or sensitizing dermatitis. Skin irritation develops in the form of an erythematous rash over the surfaces of the body that are exposed to the substance or those body surfaces irritated by clothing or perspiration. Some individuals develop a macular rash on exposure, and this may progress to the weeping stage. In addition, exposed workers may develop temporary vasomotor disturbances of the upper body, with susceptibility increasing with alcohol intake (Fassett 1963d, as cited in ACGIH 1986/Ex. 1–3, p. 91).

The fatal dose in humans is estimated to be 40 to 50 grams. Calcium cyanamide is used medically for its Antabuse-like effect, and the maintenance dose in adults is between 50 and 100 mg/day (Hald, Jacobsen, and Larson 1952/Ex. 1–905). One death has been caused by the ingestion of alcohol when the patient was on calcium cyanamide alcohol aversion therapy (Gosselin, Smith, and Hodge 1984, p. II–351).

Based on this evidence, OSHA is proposing an 8-hour TWA PEL of 0.5 mg/m³ for calcium cyanamide. The Agency believes that this limit is necessary to substantially reduce the significant risks of material health impairment in the form of eye and skin irritation, sensitizing dermatitis, and the occurrence of Antabuse-like effects in workers in construction, maritime, and agriculture. Promulgation of this limit will also make the PEL for calcium cyanamide consistent across all OSHA-regulated sectors.

CARBOFURAN

CAS: 1563–66–2; Chemical Formula: $C_{12}H_{15}NO_3$

H.S. No. 1068

OSHA has no limit for carbofuran in the construction, maritime, or agriculture industries. The ACGIH has a TLV®–TWA of 0.1 mg/m³ for this substance. The PEL OSHA is proposing for carbofuran is 0.1 mg/m³ as an 8-hour TWA, and NIOSH concurs (Ex. 8–47, Table N1) that this limit is appropriate. This is the limit recently established for carbofuran in general industry.

Carbofuran, also called furadan, is a white, crystalline, odorless solid (HSDB 1990). Carbofuran is used as a miticide, a nematocide, and an insecticide on corn, alfalfa, tobacco, and other field groups. Carbofuran belongs to the methyl carbamate group of pesticides (HSDB 1990). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Carbofuran is a cholinesterase inhibitor in animals and humans. The oral LD_{50} in rats is 5.3 mg/kg, and the LC_{50} in the same species is 85 mg/m³ for an unspecified time (RTECS 1990). The dermal LD_{50} in rabbits is 885 mg/kg (RTECS 1990). The N-methyl carbamate insecticides produce their effects by carbamylation of acetylcholinesterase enzyme, which allows the accumulation of acetylcholine at synapses in the nervous system. Rats fed carbofuran at a dietary level of 10 or 25 ppm for 180 days showed no significant histopathological effects at autopsy (Rotaur et al. 1981, Med. del Lavoro 72(5):399–403). Repeated intraperitoneal doses of 0.25 mg/kg carbofuran in mice caused a significant decrease in hemoglobin, number of erythrocytes and platelets, and hematocrits. Bone marrow depression and splenic hyperplasia were also seen (Gupta et al. 1981, in Toxicology 25(2–3):255–260). Rhesus monkeys did not display cholinesterase depression after exposure to concentrations equivalent to 0.56 mg/m³ of a 75-percent wettable carbofuran powder (Tobin 1970/Ex. 1–935). Inhibition of plasma, erythrocyte, and brain cholinesterase levels was evident

in rats at carbofuran levels of 50 ppm in the diet (Tobin 1970/Ex. 1–935). Six-hour exposures to concentrations of 0.86 mg/m³ caused significant cholinesterase inhibition in experimental animals (Tobin 1970/Ex. 1–935).

In humans, overexposure to carbofuran causes the onset of the signs and symptoms of cholinesterase inhibition soon after exposure; however, recovery generally occurs rapidly after the cessation of exposure. Formulators and applicators of carbofuran who have been poisoned displayed the following signs and symptoms: blurred vision, nausea, weakness, and excessive perspiration (Hayes 1982, p. 454). If the exposure is severe, death caused by pulmonary edema and paralysis of the respiratory center may occur (Morgan 1989, in Recognition and Mgmt. of Pesticide Poisonings, p. 13).

Based on this evidence, OSHA is proposing a permissible exposure limit of 0.1 mg/m³ as an 8-hour TWA for this substance to protect employees in construction, maritime, and agriculture from the significant risk of cholinesterase inhibition potentially associated with exposure to this substance. The Agency preliminarily concludes that this limit is necessary to substantially reduce the significant occupational risk of material impairment of health posed by exposure to this substance. In addition, promulgation of this limit will make OSHA's PEL for carbofuran consistent across all regulated sectors.

CARBON DIOXIDE

CAS: 124–38–9; Chemical Formula: CO_2
H.S. No. 1069

OSHA's current limit for carbon dioxide in construction and maritime is 5000 ppm as an 8-hour TWA. There is no PEL in agriculture. The ACGIH has a 5000-ppm TLV®–TWA for this substance with a 30,000-ppm TLV®–STEL, and NIOSH has a TWA REL of 10,000 ppm with a 10-minute 30,000-ppm ceiling limit; OSHA is proposing PELs of 10,000 ppm (8-hour TWA) and 30,000 ppm (15-minute STEL) for carbon dioxide in construction, maritime, and agriculture. These are the limits recently established in general industry.

Carbon dioxide is a colorless, odorless, noncombustible gas. This substance finds use as a refrigerant; in the production of urea, carbonates, methanol, hydrocarbons, and sodium salicylate; in the carbonation of beverages; as a propellant in aerosols; in livestock slaughtering; in food processing and preserving; in oil well stimulation; to inert flammable materials; in welding; in municipal water treatment; in cloud seeding; as a pesticide and fertilizer; and in many other applications (HSDB 1990). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Exposure to high concentrations of carbon dioxide (CO_2) causes metabolic effects (increases in respiratory rate; decreases in serum calcium and urinary phosphorus output (both indicators of mild metabolic stress) and central nervous system depression and excitation. In experimental animals, exposure to a 30,000-ppm concentration of CO_2 caused elevated blood pressure and pulse, exposure to 50,000 ppm for 30 minutes caused signs of intoxication, and exposure to concentrations between 70,000 and 100,000 ppm causes unconsciousness within a few minutes (Hamilton 1974, Indus. Tox. 3rd ed., p. 237). The offspring of rats exposed to a mixture of CO_2 (6%), oxygen (20%), and nitrogen (74%) on a single gestation day showed a significantly higher percentage of cardiac malformations than controls; administration of CO_2 to pregnant rabbits on gestation days 7 to 12 caused 16 of 67 fetuses to have defects of the vertebral column (Shepard 1980, Cat. Teratogenic Agents 3rd ed., p. 52).

Both the ACGIH (1986/Ex. 1–3) and NIOSH (1976 Criteria Document) cite studies indicating that continuous exposure to between 1.5 and 3 percent carbon dioxide (15,000 to 30,000 ppm) results in few, if any, adverse effects in exposed individuals. However, electrolyte imbalances and other metabolic changes have been associated with prolonged exposure to 10,000 to 20,000 ppm CO_2 (Schulte 1964/Ex. 1–366; Gray 1950, as cited in ACGIH 1986/Ex. 1–3, p. 102). Increases in the rate of respiration have been observed among resting subjects exposed to carbon dioxide at a concentration of 39,500 ppm for periods shorter than a day and among exercising subjects exposed to airborne CO_2 concentrations below 30,000 for the same period (Sinclair et al. 1969, as cited in ACGIH 1986/Ex. 1–3, p. 102). Many occupational fatalities have occurred as a result of workers entering CO_2-contaminated wells and silos (Groves and Ellwood 1988, in Ann. Occup. Hyg. 33(4):519–535; Proctor, Hughes, and Fischman 1988, p. 119). The signs and symptoms of acute CO2 poisoning include psychomotor agitation, myoclonic twitching, eye flickering, dyspnea, dizziness, sweating, paresthesias, and restlessness (NIOSH 1976 Criteria Document; Williams 1958, Br. Med. J. 2:1012–1014; Smith, J.C. et al. 1985, in Gilman et al. Goodman and Gilman's Pharm. Basis of Therapeutics 7th ed., p. 333–335).

Based on this evidence, OSHA is proposing a 10,000-ppm PEL as an 8-hour TWA and a 30,000-ppm STEL over 15 minutes to protect employees in the construction, maritime, and agriculture sectors from experiencing the metabolic and central nervous system changes that are associated with exposure to elevated short-term concentrations of CO_2. The Agency believes that these limits are necessary to substantially reduce these risks of material health impairment. Promulgation of these PELs also will make OSHA's limits for carbon dioxide consistent across all regulated sectors.

CARBON MONOXIDE

CAS: 630–08–0; Chemical Formula: CO
H.S. No. 1071

OSHA's limit for carbon monoxide in construction and maritime is 50 ppm as an 8-hour TWA. The limits in marine terminals and longshoring are a 50 ppm and, in confined spaces, a 100 ppm ceiling. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 50 ppm and a TLV®–STEL of 400 ppm for carbon monoxide. NIOSH (1973d/Ex. 1–237) recommends an 8-hour TWA limit of 35 ppm and a 200-ppm ceiling. For all sectors, OSHA is proposing PELs of 35 ppm (8-hour TWA) and 200 ppm (ceiling, measured over 5 minutes), respectively. OSHA is retaining the 100 ppm ceiling for CO in confined spaces in marine terminals and longshoring. NIOSH concurs (Ex. 8–47, Table N1) that the proposed limits are appropriate. These are the limits recently established for carbon monoxide in general industry.

Carbon monoxide is a flammable, colorless, practically odorless gas. This substance is used as a reducing agent in metallurgical operations, in the manufacture of metal carbonyls and zinc-based white pigments, and as a chemical intermediate. Most occupational exposures to this ubiquitous substance are the result of the incomplete combustion of organic material (HSDB 1990; Gosselin, Smith, and Hodge 1984, p. III–94).

Carbon monoxide has caused a large number of industrial fatalities as a result of its tendency to combine readily with hemoglobin to form carboxyhemoglobin (COHb). The Immediately Dangerous to Life and Health (IDLH) level for carbon monoxide is 1500 ppm (NIOSH 1987). At levels above this, workers quickly lose consciousness; if exposure is not terminated immediately, death by asphyxiation follows quickly.

In experimental animals, asphyxiation occurs when the airborne concentration

of CO exceeds 3 percent (30,000 ppm) (HSDB 1990). CO also causes reproductive and developmental effects in animals. The LC_{50} in rats is 1807 ppm for 4 hours (RTECS 1990). Rats were exposed (nose only) to 527, 1091, or 1800 ppm CO for 1 hour/day for 14 days; no treatment-related clinical effects were seen in animals of any dose group, although blood carboxy-hemoglobin levels increased in a dose-dependent manner. In controls, blood COHb levels averaged 2 percent and rose to 60 to 62 percent in the 1800-ppm group. Respiration rate and hemoglobin decreased in all groups in a dose-dependent manner (Ayres, Mosberg, Burger, Hayes, Sagartz, and Coggins 1989, in Inhal. Toxicol. 1(4):349–363). Exposure of pregnant animals to concentrations of CO that are not maternally toxic causes destructive changes to the nervous systems of the fetuses; pregnant rats exposed to 150 ppm CO had fetuses that showed evidence of persistent central nervous system damage (Mactutus and Fletcher 1984, 1985, in Science 223:409–411 and Teratology 31:1–12). The offspring of mice and rabbits exposed during pregnancy also showed embryotoxic or developmental effects (RTECS 1990). Neonatal rats were exposed to 500 ppm CO for up to 32 days after birth; the CO exposure caused cardiomegaly, evidenced by a 47 percent increase in ventricular weight to body weight ratio in rats exposed for 28 days (Clubb, Penney, and Bishop 1989, in J. Mol. Cell. Cardiol. 21(9):945–955).

Excessive accumulations of COHb cause hypoxic stress in healthy individuals as a result of the reduced oxygen-carrying capacity of the blood. In patients with cardiovascular disease, such stress can further impair cardiovascular function. A number of studies show that 8-hour TWA exposures to 50 ppm carbon monoxide generally results in COHb levels of 8 to 10 percent. Such levels are not generally associated with overt signs or symptoms of health impairment in healthy individuals with strong cardiovascular systems who are working under nonstressful conditions. However, the ACGIH believes that a TLV®–TWA of 25 ppm, which results in COHb levels of 4 percent or less, may be necessary to protect workers with cardiovascular disease, because this condition places workers at higher risk of serious cardiovascular injury (ACGIH 1986/Ex. 1–3, p. 106). The NIOSH REL of 35 ppm TWA is also aimed at protecting workers with chronic heart disease (CHD); NIOSH believes that such workers should not be allowed to have carboxyhemoglobin levels that approach 5 percent. In the prior rulemaking, several commenters questioned the need to lower the 8-hour TWA and to add a STEL. In response to these commenters, OSHA quoted the ACGIH (1986/Ex. 1–3):

> Each molecule of CO combining with hemoglobin reduces the oxygen carrying capacity of the blood and exerts a finite stress on man. Thus, it may be reasoned that there is *no dose of CO* that is not without an effect on the body. Whether that effect is physiologic or harmful *depends upon the dose of CO and the state of health of the exposed individual.* The body compensates for this hypoxic stress by increasing cardiac output and blood flow to specific organs, such as the brain or the heart. When this ability to compensate is overpowered or is limited by disease, tissue injury results [emphasis added].
>
> Exposure to CO sufficient to produce COHb saturations in the 3–5% range impairs cardiovascular function in patients with cardiovascular disease and in normal subjects * * *. The primary effect of exposure to low concentrations of CO on workmen results from the hypoxic stress secondary to the reduction in the oxygen-carrying capacity of blood * * *. Workmen with significant disease, both detected and undetected, may not be able to compensate adequately and are at risk of serious injury. For such workers, a TLV of 25 ppm * * * might be necessary. Even such a concentration might be detrimental to the health of some workers who might have far advanced cardiovascular disease * * *. It would appear to the Committee that the time-weighted TLV of 50 ppm for carbon monoxide might also be too high under conditions of heavy labor, high temperatures, or at high elevations (ACGIH 1986/Ex. 1–3, p. 106).

Thus, the ACGIH also regards a lower limit for CO as necessary to protect workers with cardiovascular or pulmonary disease or those working under stressful conditions.

OSHA believes that it is clearly necessary to set a CO level that protects workers who have CHD because (1) a large percentage of employees have it, (2) it is often not diagnosed or diagnosable, and (3) it is frequently fatal. The 35 ppm 8-hour TWA is designed to protect employees with asymptomatic CHD. The term CHD is generally used to refer to the process of atherosclerosis of the coronary arteries, which leads to disturbances in the myocardial blood supply.

Each year, more persons in the United States die from CHD than from any other disease. Coronary atherosclerotic heart disease is the most common form of cardiac disease in adults in the United States (Eros, WF, RH Holmes, and J Beyer. Coronary Disease Among United States Soldiers Killed in Action in Korea, J. Am. Med. Assoc. 152:1090–1093, 1953). During the Korean War, autopsies performed on young soldiers, with an average age of 22 years, revealed that 77.3 percent had a gross pathologic evidence of CHD. A study (Spiekerman, RE, JT Brandenberg, RWP Anchor, and JE Edwards 1982. The Spectrum of Coronary Heart Disease in a Community of 30,000. A Clinicopathologic Study, Circulation 25:57–65) of autopsies in a stabilized population of 30,000 revealed that CHD was the cause of death in 40 percent of the males. In most cases, the first clinical manifestation of CHD is expressed either as the angina pectoris syndrome or as frank myocardial infarction. According to the Framingham, Massachusetts, study conducted by the U.S. Public Health Service, CHD is first manifested as myocardial infarction in one-sixth of all cases of CHD (NIOSH, Criteria for a Recommended Standard: Occupational Exposure to Carbon Monoxide, 1972).

It is clearly evident from these statements that the general worker population in the United States is composed of a very significant number of persons with CHD. Since the identification of such persons in the absence of overt clinical symptoms is virtually impossible, it is necessary to assume that the average worker has asymptomatic CHD, especially when his/her first clinical symptom may be sudden death (NIOSH, Criteria for a Recommended Standard: Occupational Exposure to Carbon Monoxide, 1972). Several studies demonstrate the significant risk associated with CO exposure, particularly with respect to coronary heart disease. A study of firefighters in Los Angeles (Barnard and Weber 1979) suggests that CO exposure during firefighting may be responsible for the high incidence of heart disease in firefighters. In some fires, peak exposures were occasionally as high as 3000 ppm CO, with 40 percent of peak values in the 100- to 500-ppm CO range. However, in some fires, the peak CO exposure was below 100 ppm. Although these peak exposures in firefighters were high, firefighters are likely to be exposed overall for fewer hours than the workers of concern in this rulemaking.

A prevalence study was recently performed on angina pectoris, ECG changes, and blood pressure that involved 1,000 workers from 20 foundries (Hernberg, Karava, Koskela, and Luoma. 1976. Angina pectoris, ECG findings and blood pressure of foundry workers in relation to carbon monoxide exposure. Scand. J. Work Environ. & Health 2). On the basis of actual measurements of air concentrations of

CO, the workers were divided into three exposure groups. The workers' smoking habits, both present and past, were factored into the analysis. No association between CO exposure and blood pressure or ECG changes was found. However, a clear dose-response was found with regard to the prevalence of angina (as obtained by history on a World Health Organization-recommended questionnaire) and CO exposure in workers (both smokers and nonsmokers).

In order to obtain some quantitative estimate of the process of selection for healthy workers in the foundries studied, the authors contacted workers who had left this type of work. Fourteen percent of those who had left foundry work after less than 1year of employment and 7 percent of those who had left it after more than 5years of foundry employment reported health impairment or disability as the reason for quitting foundry work. The authors conclude that this evidence indicates that less healthy workers left employment early on. This selection process, which is apparent in this study and also in the American foundry study by Redmond, results in an over-representation of fit and healthy workers in physically heavy work, such as foundry work.

NIOSH conducted a prospective cohort mortality study among 1,558 white male motor vehicle examiners who were employed in New Jersey for a minimum of 6 months between 1944 and 1973 (Stern, Lemen, and Curtis 1981). Industrial hygiene surveys indicated that the examiners were exposed to carbon monoxide at a time-weighted-average (TWA) of 10 to 24 ppm. Using a modified life table technique, the expected deaths were compared to the expected deaths through August 1973. The overall deficit of mortality observed (SMR 80) in this occupational cohort during the first 10-year period was to be expected as a result of the widely accepted "healthy worker effect." However, the component SMR for cardiovascular disease deaths (134) was unexpected, since the "healthy worker effect" had been most significantly associated with decreased cardiovascular disease mortality (McMichael 1976, in Standardized Mortality Ratios and the "Healthy Worker Effect": Scratching Beneath the Surface, J. Occup. Med. 18:165–168). The authors refer to the excess of cardiovascular disease mortality found within the first 10 years following employment as "non-statistically significant, but highly unusual." OSHA believes that this evidence suggests that slightly elevated COHb may contribute to excess cardiovascular disease rates in a healthy population that is of average fitness (since the work is not physically hard).

A recent study (Kristensen 1989, in Scand. J. Work Environ. Hlth. 15(4): 245–264) reviewed the epidemiological evidence for an association between carbon monoxide and heart disease and concluded that carbon monoxide exerts acute and possibly reversible short-term effects that can increase the risk of cardiovascular disease. Twenty-four male volunteers with stable angina pectoris exercised after breathing air or low levels (not further specified) of CO; subjects' blood COHb levels increased to 3 percent after CO exposure, and oxygen uptake was also significantly reduced (Kleinman, Davidson, Vandagroff, Caiozzo, and Whittenberg 1989, in Arch. Environ. Hlth. 44(6): 361–369).

In another recent study, male volunteers aged 35 to 75 with stable exertional angina pectoris and positive exercise treadmill tests were exposed to CO concentrations designed to cause 2.2 to 4.4 percent COHb levels after exercise. The subjects performed a symptom-limited exercise test on a treadmill, followed by exposure for 1 hour to CO, and then performed a second treadmill test. All subjects who completed the study (N=63) showed significant decreases in time to onset of ischemic ST-segment changes; in the 2 percent COHb group, this change equalled 5.1 percent, and in the 4 percent COHb group it averaged a 12.1 percent decrease (Allred, Bleecher, Chaitman, Dahms, Gottlieb, Hackney et al. 1989, in Res. Rep. Hlth. Effect. Inst. 25:79).

In addition, a number of recent studies point to CO's reproductive and neurotoxic/behavioral effects. For example, episodes of acute CO poisoning have been known to lead to late-onset fatal demyelination in the exposed worker (Winter and Miller 1976, in JAMA 236: 1502–1504), mental damage manifested as gross neuropsychiatric change, or personality deterioration (Smith and Brandon 1973, in Br. Med. J. 1: 318–321). Carbon monoxide is also known to cross the placental barrier in humans (Gosselin, Smith, and Hodge 1984, p. III–97). The infants of mothers who were acutely poisoned by CO during pregnancy often show evidence of neurological damage and may have gross brain damage (Goodman 1980, Pharm. Basis Therap. 6th ed., p. 1643).

Another study, Mortality of Steelworkers Employed in Hot Jobs (Redmond 1977) is a well-designed investigation of the possible relationships between heat stress (both environmental and metabolic heat load) and cause-specific mortality patterns in a cohort of 69,414 steelworkers. Two main sets of data were analyzed: (1) Field data, and (2) mortality data. The field data were divided into four computer files: (1) indoor environmental measurements, (2) outside plant environmental measurements, (3) National Weather Service Data, and (4) activity data. From these data, estimations of heat exposure and work loads were made. Workers were then divided into four categories based on heat and energy expenditures, and the mortality experience of workers in each category was compared to mortality in the control group. The investigators made no attempt to characterize exposures to carbon monoxide.

For mortality from cardiovascular-renal disease (CVR), relative risks were significantly low for those workers in the two higher environmental heat categories, indicating a possible relationship between decreased mortality and environmental heat stress. When the mortality experience was examined by length of exposure, a higher than expected risk was noted among workers with less than 6 months of exposure in all exposure groups. This was less than the expected risk of mortality due to cardiovascular-renal disease once the initial 6 month employment period has been exceeded. The authors state that "(t)hese higher risks may be indicative of a relationship between inability to work in jobs involving heat stress and health. This selection for health may at least partially explain the results for mortality from cardiovascular disease. Workers who are less fit leave the job after varying lengths of exposure, thus resulting in a downward trend in mortality for workers who remain on the job."

When mortality from arteriosclerotic heart disease was separated out from the CVR heading, the risks of dying were significantly low for workers in the two highest exposure categories. When the mortality patterns were examined by length of exposure, higher risks were noted among those with short exposure times. These trends are consistent with those seen for cardiovascular mortality.

The American Iron and Steel Institute (AISI) pointed out in the general industry hearing (Ex. 129) that the high heat stress workers would also have had higher exposure to CO. AISI argued that it was therefore not necessary to lower the exposure limit for CO. OSHA

believes that the more likely explanation of this result, as seen in several of the other studies discussed, is the healthy worker effect. Only persons with substantially above-average cardiovascular fitness can perform these physically demanding jobs. Persons with average or below-average cardiovascular fitness either do not take such jobs in the first place or leave them after a few months. Furthermore, the level of exertion the jobs require keeps cardiovascular fitness high. Consequently, the low CHD rates seen are caused by the high proportion of cardiovascularly fit workers in these jobs. The study cannot therefore be said to contradict the need to reduce COHb levels to prevent excess CHD risk in workers with average or below-average levels of cardiovascular fitness. In addition, the study did not attempt to correlate CO levels and cardiovascular disease, and no CO measurements were taken.

As pointed out above, cardiovascular disease (detected or undetected) and pulmonary impairment are widespread in the general population in this country, in workers as well as other sub-populations. In addition, OSHA is particularly concerned about the adverse effects of CO because workers regularly encounter complex and stressful situations at work, including heat stress, jobs demanding heavy exertion, and tasks requiring both judgment and motor coordination. OSHA standards are intended to protect workers of average and below-average fitness and those who engage only intermittently in heavy physical labor and who do not therefore receive the benefit of physical conditioning.

OSHA thus preliminarily finds that, in construction, maritime, and agriculture, the proposed 8-hour TWA of 35 ppm for carbon monoxide is needed to reduce the significantly increased risk of cardiovascular disease that is associated with overexposure to CO. The Agency believes that a ceiling of 200 ppm is necessary to ensure that peak CO exposures are kept below the 1500 ppm IDLH level by a reasonable safety factor. The ceiling limit will also assist in keeping COHb levels below 5 percent; the ceiling will be measured over 5 minutes to permit the use of simpler monitoring techniques.

Based on this evidence, OSHA is proposing an 8-hour TWA of 35 ppm and a ceiling of 200 ppm as the PELs for carbon monoxide in the construction, maritime, and agriculture industries. In the construction and marine terminal industries, employees regularly enter and work in confined spaces. In these sectors, CO therefore presents an especially great danger of death from IDLH levels of CO. Consequently, OSHA is retaining the 100 ppm ceiling for CO in confined spaces in these sectors, as well as the provision requiring monitoring of these spaces.

The Agency preliminarily concludes that these limits will ensure that the COHb levels of exposed workers (especially of non-smokers) in these sectors are maintained at or below 5 percent, which will protect those workers at greater risk because of cardiovascular or pulmonary impairment. In addition, these revised limits will protect healthy workers in the affected sectors who must work in environments involving intermittent exertion, heat stress, or other strenuous conditions. OSHA believes that these limits are necessary to substantially reduce the significant occupational risk associated with both chronic and peak exposures to carbon monoxide in the workplace. OSHA preliminarily finds that the hypoxic stress associated with overexposures to carbon monoxide clearly constitutes a material impairment of health and functional capacity. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

CHLORPYRIFOS

CAS: 2921–88–2; Chemical Formula: $C_9H_{11}Cl_3NO_3PS$

H.S. 1091

OSHA has no limit for chlorpyrifos in the construction, maritime, or agriculture sectors. The ACGIH has a TLV®–TWA of 0.2 mg/m³ and a TLV®–STEL of 0.6 mg/m³, with a skin notation, for chlorpyrifos. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the PEL being proposed. OSHA is proposing a 0.2-mg/m³ 8-hour TWA and a skin notation for chlorpyrifos; this is the limit recently established in general industry.

Chlorpyrifos is a white, crystalline solid with a mild, mercaptan-like odor (EPA Fact Sheet 1985). Chlorpyrifos is an organophosphate pesticide that is used on grain crops (EPA Fact Sheet 1985). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Exposure to chlorpyrifos causes cholinesterase inhibition in humans and animals. The oral LD_{50} in rats is 82 mg/kg, and the dermal LD_{50} in rabbits is 2000mg/kg (RTECS 1990). Chlorpyrifos is an active inhibitor of plasma cholinesterase but has only moderate capacity to reduce red blood cell cholinesterase or to cause cholinergic symptoms and systemic injury (ACGIH 1986/Ex. 1–3, p. 138). Particle inhalation has been shown to cause mild plasma cholinesterase depression in dogs exposed for 4 hours at the upper end of a 140- to 280-mg/m³ range (Spencer 1968, as cited in ACGIH 1986/Ex. 1–3, p. 138). A recent study (Corley, Calhoun, Dittenber, Lomax, and Landry 1989, in Fund. Appl. Toxicol. 13(3):616–618) examined the subchronic inhalation toxicity of chlorpyrifos in rats. Rats were exposed (nose-only) to 5.2, 10.3, or 20.6 ppb chlorpyrifos vapor for 6 hours/day, 5 days/weeks, for 13 weeks. No significant treatment-related histopathological, hematological, serum chemistry, or cholinesterase activity changes were seen in these animals, leading the authors to conclude that chlorpyrifos has low subchronic inhalation toxicity in rats. Cats were given a single sublethal oral dose of chlorpyrifos; doses ranged from 10 mg/kg to 40 mg/kg. No signs of poisoning were seen at the lowest dose, but the high-dose group developed clinical signs of toxicity (Hooser, Beasley, Sundberg, Harlin 1988, in Am. J. Vet. Res. 49(8):1371–1375). Dogs and rats fed 3.0 mg/kg chlorpyrifos daily for 2 years showed no adverse effects (FAO/WHO (Food and Agriculture Organization/World Health Organization) 1972, as cited in ACGIH 1986/Ex. 1–3, p. 138). Male and female rats showed no teratogenic or reproductive effects when fed 1.0 mg/kg per day (Dow Chemical Company 1972a, as cited in ACGIH 1986/Ex. 1–3, p. 138).

Workers applying chlorpyrifos as a spray were exposed to 0.5 percent chlorpyrifos emulsion and exhibited a marked decrease in plasma and red cell cholinesterase levels (Eliason, Cranmer, von Windeguth et al. 1989/Ex. 1–633). In five of seven exposed sprayers, this reduction was greater than 50 percent. However, another study showed no ill effects on cholinesterase metabolism when human volunteers were exposed to an ultra-low-volume spray (0.8 um/m3 for 3 to 8 minutes) (Ludwig, Kilian, Dishburger, and Edwards 1970/Ex. 1–563). Human cholinesterase levels appear to be less affected by dermal exposure than do those of rabbits (ACGIH 1986/Ex. 1–3, p. 138). However, when chlorpyrifos was administered in four repeated dermal doses of 25 mg/kg, each applied for 12 hours, human volunteers did exhibit depressed plasma cholinesterase levels. Human volunteers ingesting 0.03mg/kg chlorpyrifos for 3 weeks showed no cholinesterase effects, but subjects ingesting 0.1 mg/kg demonstrated plasma cholinesterase depression (Dow Chemical Company

1973f, as cited in ACGIH 1986/Ex. 1–3, p. 138). A recent Dow study (Brenner, Bond, McLaren, Greene, and Cook 1989, Br. J. Ind. Med. 46(2):133–137) showed that the prevalence of illness and of symptoms was not elevated over that of controls in a group of 175 workers exposed between 1977 and 1985 to chlorpyrifos during its manufacture or formulation.

Based on this evidence in humans and animals, OSHA is proposing a PEL of 0.2 mg/m³ as an 8-hour TWA, and a skin notation, for chlorpyrifos in the construction, maritime, and agriculture sectors. The Agency believes that these limits for chlorpyrifos will protect workers in these industries from the significant risk of cholinesterase inhibition caused by exposure to this substance. OSHA believes that the skin notation is necessary to prevent the systemic effects that have been demonstrated to occur in humans dermally exposed to chlorpyrifos. OSHA preliminary finds that the cholinesterase inhibition and systemic effects associated with exposure to chlorpyrifos constitute material impairments of health and that the proposed PEL is necessary to substantially reduce these risks. In addition, promulgation of the proposed limit will make OSHA's PEL for chlorpyrifos consistent across all regulated sectors.

CRUFOMATE

CAS: 299–86–5; Chemical Formula: $C_{12}H_{19}ClNO_3P$

H.S. No. 1103

OSHA has no limit for crufomate in the construction, maritime, or agriculture industries. The ACGIH has a TLV®–TWA of 5 mg/m³ and a TLV®–STEL of 20 mg/m³ for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 5 mg/m³ for crufomate in the construction, maritime, and agriculture industries. This is the limit recently established in general industry.

Pure crufomate exists as a colorless crystalline solid, and commercial crufomate, also called Ruelene, is a yellow oil. Crufomate is an antihelmintic that is used on cattle, sheep, and goats to control grubs, lice, and horn flies (Clayton and Clayton 1981, p. 4833). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Crufomate actively inhibits both plasma and erythrocyte cholinesterase. The oral LD_{50} in rats is 460 mg/kg, the lowest lethal concentration in the same species is 12 mg/m³ for 4 hours, and the dermal LD_{50} in rabbits is 2000 mg/kg (RTECS 1990). Applied to the eyes of rabbits, crufomate (undiluted or as a 10 percent solution) caused corneal cloudiness, conjunctival irritation, and signs that the animals were experiencing pain (Clayton and Clayton 1981, p. 4834). In contact with the intact or abraded skin of rabbits, this substance caused slight redness (Clayton and Clayton 1981, p. 4834).

A study showed that ingestion of 200 mg of crufomate daily for seven days caused no apparent cholinesterase inhibition in the human subjects of this controlled study; however, rats and dogs receiving higher doses (5 mg/kg/day) for 2 years did show this effect (McCollister, Olson, Rowe et al. 1968/Ex. 1–350). Rats fed 1000 ppm crufomate for 2 years showed marked cholinesterase inhibition, growth retardation, testicular degeneration, atrophy of the hind quarters, and slight degeneration of the sciatic nerve (McCollister, Olson, Rowe et al. 1968/Ex. 1–350).

Because cholinesterase inhibition is a very sensitive indicator of exposure, OSHA preliminarily concludes that the proposed 8-hour TWA limit of 5 mg/m³ is needed to provide an appropriate margin of safety below the ingestion NOEL of 200 mg/day for humans, which corresponds approximately to an 8-hour inhalation exposure of 20 mg/m³. The Agency believes that this PEL is necessary to protect workers in construction, maritime, and agriculture from a significant risk of material health impairment in the form of cholinesterase inhibition. In addition, promulgation of the proposed PEL will make OSHA's limit for crufomate consistent across all regulated sectors.

CYANAMIDE

CAS: 420–04–2; Chemical Formula: $H_2NC{=}N$

H.S. No. 1104

OSHA has no limit for cyanamide in the construction, maritime, and agriculture industries. The ACGIH has a TLV®–TWA of 2 mg/m³ for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N1) that the proposed limit is appropriate. OSHA is proposing an 8-hour PEL of 2 mg/m³ for cyanamide in construction, maritime, and agriculture; this is the limit recently established in general industry.

Cyanamide, which is also called hydrogen cyanamide, is a deliquescent, crystalline solid; no information is available on its odor. Cyanamide is used as a chemical intermediate and as a crease- and fire-proofing agent in textiles.

Cyanamide is a severe irritant and caustic of the eyes, mucous membranes, and respiratory tract. In addition, this substance produces Antabuse-like effects when exposed individuals also ingest alcohol. The oral LD_{50} in rats is 125 mg/kg, and the dermal LD_{50} in rabbits is 590 mg/kg (RTECS 1990). In contact with the eye, cyanamide causes severe irritation; this substance is a caustic that destroys any tissue it comes into contact with, especially when moisture is present (Grant 1986, p. 286). Cyanamide dust is irritating to the respiratory tract (Grant 1986, p. 286). Acutely poisoned rats exhibited signs of parasympathetic overactivity, including miosis, excessive salivation, lacrimation, and twitching (Grant 1986, p. 286).

When cyanamide is ingested or inhaled by a person who has also consumed an alcoholic beverage, the person experiences vasodilation of the face and neck, tachycardia, tachypnea, nausea, vomiting, and hypotension. This syndrome is referred to as the Antabuse syndrome. Studies of cyanamide's Antabuse-like effects indicate that the effect is about one-half that of an equivalent dose of tetraethylthiuram disulfide (Antabuse) and one-sixth that of tetramethyl thiuram disulfide (Hald, Jacobsen, and Larsen 1952/Ex. 1–905). Workers whose skin is in contact with cyanamide can develop a severe and persistent dermatitis (Gerrin 1989).

Based on this evidence in humans and animals, OSHA is proposing an 8-hour TWA limit of 2 mg/m³ TWA for cyanamide. The Agency preliminarily concludes that this limit will protect workers in construction, maritime, and agriculture from the significant risks of sensory irritation and of the Antabuse syndrome in workers who have ingested alcohol. The Agency believes that this limit will substantially reduce these significant risks, which constitute material health impairments. In addition, promulgation of the proposed PEL will make OSHA's limit for cyanamide consistent across all regulated sectors.

DICHLORVOS (DDVP)

CAS: 62–73–7; Chemical Formula: $(CH_3O)_2POOCH{=}CCl_2$

H.S. No. 2051

OSHA's permissible exposure limit for dichlorvos in general industry, construction, and maritime is an 8-hour TWA of 1 mg/m³; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit in agriculture. The ACGIH has a limit of 0.1 ppm (≈1 mg/

m^3) as an 8 hour TLV®–TWA, with a skin notation, for dichlorvos. NIOSH has no REL but concurs (Ex. 48–7, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 1 mg/m^3, and a skin notation, for dichlorvos in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Dichlorvos is a colorless to amber liquid that has a mild chemical odor. It is used as an insecticide and fumigant and is a primary ingredient in pest strips (Proctor, Hughes, and Fischman 1988, p. 194; Hayes 1982, p. 343). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Dichlorvos causes cholinesterase inhibition in humans and animals, as well as reproductive effects in animals. In rats, the oral LD_{50} is 25 mg/m^3 and the LC_{50} is 15 mg/m^3 for 4 hours (RTECS 1990). The dermal LD_{50} in rabbits is 107 mg/kg (RTECS 1987). No illness occurred in monkeys exposed to dichlorvos concentrations greater than 7 mg/m^3 for 2 hours/day for 4 days; however, miosis and a rapid drop in blood cholinesterase were observed in these animals (Witter et al. 1961, in Hayes 1982, p. 344). Monkeys exposed continuously for 1 week to dichlorvos concentrations between 1.4 to 2.0 mg/m^3 developed severe cholinesterase inhibition (Durham et al. 1959). Continuous exposure of monkeys and rats to a dichlorvos concentration of 0.27 mg/m^3 for 4 days had no effect on cholinesterase levels (Durham et al. 1959, in Hayes 1982, p. 344). Dichlorvos given to pregnant rats by intraperitoneal injection at a dose of 15 mg/m^3 on the 11th day of pregnancy caused malformations in three of 41 fetuses (Kimbrough and Gaines 1968, in IARC 1979, Vol. 20, p. 108). Mice exposed to a single oral dose of 40 mg/kg showed extensive injury to the seminal epithelium; similar but less extensive changes were seen in the reproductive tissue of animals given doses of 10 mg/kg/day for 18 days (Krause and Homola 1972, in Hayes 1982, p. 347). Several carcinogenicity bioassays have failed to show a statistically significant increase in the incidence of tumors, and the International Agency for Research on Cancer has concluded that the data in animals are inadequate to evaluate the carcinogenicity of this substance (IARC 1979, Vol. 20, pp. 97–123).

Human exposure to dichlorvos can occur by inhalation, ingestion, or skin absorption, and the signs and symptoms associated with exposure include chest tightness, bronchial secretion, laryngeal spasms, cyanosis, blurred vision, rhinorrhea, headache, anorexia, nausea, abdominal cramps, and diarrhea (Proctor, Hughes, and Fischman 1988, p. 194). Signs and symptoms usually onset soon after exposure but may be delayed for as long as 12 hours (Proctor, Hughes, and Fischman 1988, p. 194). Effects on the central nervous system include giddiness, confusion, ataxia, slurred speech, Cheyne-Stokes respiration, convulsions, coma, and loss of reflexes (Proctor, Hughes, and Fischman 1988, p. 194). Complete recovery from exposure-related symptoms usually occurs within 1 week (Proctor, Hughes, and Fischman 1988, p. 194). Dichlorvos also causes a persistent contact dermatitis of allergic origin (Mathias 1983; Matsushita et al. 1985, in Proctor, Hughes, and Fischman 1988, p. 195). Of 28 volunteers exposed to a concentration of 1 mg/m^3 dichlorvos for 7.5 to 8.5 hours (single exposures), 20 to 25 percent experienced plasma cholinesterase depression (ACGIH 1986, p. 192). Studies on pesticide workers handling dichlorvos have shown significant reductions in blood cholinesterase and in the number of lymphocytes and monocytes, as well as leukocytosis and neutrophilia; levels returned to normal within 2 weeks after the cessation of exposure (Pukach et al. 1974, in ACGIH 1986, p. 192). Symptoms of dichlorvos intoxication seen in these workers included weakness, headache, nausea, abdominal cramps, blurred vision, non-reactive pupils, chest tightness, salivation, sweating, convulsions, and coma (Pukach et al. 1974). In a study in which 13 workers were exposed to an average concentration of 0.7 mg/m^3 dichlorvos for 12 months, erythrocyte cholinesterase activity was reduced by 35 percent and serum cholinesterase activity showed a 60 percent reduction (Menz, Luetkemeir, and Sachsse 1971, in Proctor, Hughes, and Fischman 1988, p. 194).

Based on this evidence in humans and animals, OSHA preliminarily concludes that dichlorvos causes cholinesterase inhibition. OSHA believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. OSHA believes that the proposed PEL of 1 mg/m^3 as an 8-hour TWA, and a skin notation, are necessary to substantially reduce the risk of this material health impairment. In addition, establishing this limit for dichlorvos in agriculture will make OSHA's PEL for this substance consistent across all OSHA-regulated sectors.

DICROTOPHOS

CAS: 141–66–2; Chemical Formula: $C_8H_{16}NO_5P$

H.S. No. 1131

OSHA has no limit for dicrotophos in the construction, maritime, or agriculture industries; the ACGIH has a TLV®–TWA of 0.25 mg/m^3, with a skin notation, for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N1) that the proposed limit is appropriate. OSHA is proposing an 8-hour TWA of 0.25 mg/m^3, and a skin notation, for dicrotophos in construction, maritime, and agriculture. This is the limit recently established for dicrotophos in general industry.

Dicrotophos is a yellow-brown liquid with a mild, ester-like odor (Sittig 1985, p. 335). This substance is an organophosphate insecticide that is used on apples, cotton, and other crops (HSDB 1990). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Dicrotophos is a cholinesterase inhibitor in humans and animals. The oral LD_{50} in rats is 13 mg/kg, and the LC_{50} in the same species is 90 mg/m^3 for 4 hours (RTECS 1990). The dermal LD_{50} in rabbits is 168 mg/kg (RTECS 1990). Male rats exposed by inhalation to dicrotophos at a concentration of 910 mg/m^3 for 1 hour rapidly became ill but recovered quickly after exposure was discontinued (ACGIH 1986, p. 193). Two-year feeding studies in rats given 0, 1, 10, or 100 ppm dicrotophos showed no detectable effects at the 1-ppm concentration. At the higher concentrations, decreased body weights (as compared with those of controls) and cholinesterase inhibition were observed (Woodard Research Corporation 1967, as cited in ACGIH 1986/Ex. 1–3, p. 193). Dietary studies in dogs showed both plasma and erythrocyte cholinesterase inhibition after exposure to a 16-ppm concentration of dicrotophos, but no significant ill effects were seen at concentrations of 0, 0.16, or 1.6 ppm (Woodard Research Corporation 196[illegible], as cited in ACGIH 1986/Ex. 1–3, p. 193).

At least two cases of accidental poisoning by dicrotophos have been reported. The first case involved a tractor driver who had been applying dicrotophos to crops and who had additionally sprayed the inside of his house with this substance. He developed abdominal cramps, nausea, vomiting, diarrhea, increased sweating, salivation,

dyspnea, tremor, generalized weakness, and reduced plasma and blood cholinesterase activities (Hayes 1982, p. 362). After appearing to recover over a 5-day period, this patient suffered respiratory arrest and required respiratory ventilation for several days. He subsequently recovered completely (Hayes 1982, p. 362). In the other case, a man accidentally ingested dicrotophos and lost consciousness. On admission to the hospital, he was without respiration or pulse. Aggressive treatment with atropine and pralidoxime, and assisted ventilation, led to his subsequent recovery (Hayes 1982, p. 362).

Based on this evidence, OSHA is proposing an 8-hour TWA permissible exposure limit of 0.25 mg/m³, with a skin notation, for dicrotophos in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit is necessary to protect workers in these sectors from the material impairments of health, such as cholinesterase inhibition, potentially associated with inhalation, ingestion, or dermal exposure to this substance in the workplace. OSHA believes that this limit will substantially reduce this significant risk; in addition, promulgation of this limit will make the PEL for dicrotophos consistent across all regulated sectors.

DIMETHYLANILINE (N-Dimethylaniline)

CAS: 121–69–7; Chemical Formula: $C_6H_5N(CH_3)_2$

H.S. No. 1143

OSHA's permissible exposure limit for dimethylaniline in construction and maritime is 5 ppm as an 8-hour TWA, with a skin notation. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 5 ppm and a 15-minute TLV®–STEL of 10 ppm, with a skin notation, for dimethylaniline. OSHA is proposing to retain its 8-hour TWA PEL of 5 ppm, and the skin notation, and to add a STEL of 10 ppm for this substance in construction and maritime. OSHA is also proposing to apply these limits in agriculture. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limits being proposed, which are identical to those recently established for dimethylaniline in general industry.

Dimethylaniline is a yellow to brown, oily liquid. This substance finds use as an analytical reagent and a dye precursor (HSDB 1990).

Dimethylaniline induces tissue hypoxia by causing the formation of methemoglobin in the blood. This substance is also a mild irritant of the eyes and skin (RTECS 1990). The oral LD_{50} in rats is 1410 mg/kg, and the lowest lethal concentration in the same species is 250 mg/m³ for 4 hours (RTECS 1990). Instilled into the eyes of rabbits, dimethylaniline caused a moderate degree of injury (graded 5 on an ascending severity scale of 1 to 10) (Grant 1974, p. 401). Hamblin (1963a/Ex. 1–1084) reported that dimethylaniline is quantitatively less toxic than aniline. Dogs administered a single oral dose of 50 mg/kg exhibited methemoglobinemia, and absorption of dimethylaniline through the skin can increase the overall exposure (Hamblin 1963b/Ex. 1–1085). Rats survived an 8-hour exposure to the concentrated vapor (Smyth, HF et al. 1941, in Proctor, Hughes, and Fischman 1988, p. 210). Rats continuously exposed to dimethylaniline for 100 days at a concentration of 0.3 mg/m³ showed significant changes in the central nervous system, liver, and blood (Markosyan 1969, in Gigiena Sanit 34(3):7).

The literature on industrial experience with dimethylaniline is limited. Hamilton (1919/Ex. 1–741) reported collapse, prolonged unconsciousness, visual disturbances, and intense abdominal pain following the severe overexposure of two workers. The signs and symptoms of acute overexposure include headache, cyanosis of the lips, nose, and earlobes, and anemia (Proctor, Hughes, and Fischman 1988, p. 209). Exposure to dimethylaniline depresses the central nervous system and both cardiac and smooth muscle function; increased pulse and respiration may be followed by convulsions and death due to respiratory arrest (HSDB 1990).

Based on this evidence, the Agency is proposing to retain the 8-hour TWA PEL of 5 ppm and a skin notation for dimethylaniline in construction and maritime and to add a STEL of 10 ppm to this limit; OSHA is also proposing to apply the TWA and STEL and the skin notation in agriculture. OSHA believes that the STEL is necessary to afford protection from the central nervous system depression that follows acute exposures. OSHA preliminarily concludes that these limits, taken together, will provide workers in construction, maritime, and agriculture with protection from the significant risks of skin absorption, methemoglobinemia, and neuropathic effects associated with exposure to this substance; the Agency believes that these effects clearly constitute material health impairments. In addition, promulgation of the proposed limits will make OSHA's PELs for dimethylaniline consistent across all regulated sectors.

DIOXATHION (DELNAV)

CAS: 78–34–2; Chemical Formula: $C_{12}H_{26}O_6P_2S_4$

H.S. No. 1146

OSHA has no permissible exposure limit for dioxathion in the construction, maritime, or agriculture industries. The ACGIH has a TLV®–TWA of 0.2 mg/m³, with a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed in construction, maritime, and agriculture, which is 0.2 mg/m³ as an 8-hour TWA, with a skin notation. This is the limit recently established for dioxathion in general industry.

Dioxathion, also called Delnav, is an organophosphate pesticide that is used against external livestock pests, including ticks and citrus mites (Hayes 1982, p. 401). This substance is a nonvolatile, very stable, dark amber liquid. When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Dioxathion is a cholinesterase inhibitor in humans and animals. The commercial product contains both the cis- and trans-isomers of 2,3-p-dioxanedithiol; the cis-isomer is approximately four times as acutely toxic as the trans-isomer (ACGIH 1986/Ex. 1–3, p. 219). The oral LD_{50} in rats is 20 mg/kg, and the LC_{50} in the same species is 1396 mg/m³ for 1 hour (RTECS 1990). The dermal LD_{50} in rabbits is 85 mg/kg (RTECS 1990). Acutely poisoned animals show decreases in cholinesterase activity as well as dyspnea, nausea, vomiting, diarrhea, tremor, and ataxia (Hayes 1982, p. 401). Instillation of 0.1 ml dioxathion into the rabbit eye produces mild, transient conjunctivitis but no corneal damage (ACGIH 1986/Ex. 1–3, p. 219). Rats fed 10 mg/kg dioxathion for 14 days and dogs given 10 doses of 0.8 mg/kg dioxathion over a 14-day period showed cholinesterase inhibition (HSDB 1990). Rats fed dioxathion (100 ppm of the diet) for 90 days displayed tremor and excitability (Gosselin, Smith, and Hodge 1984, p. II–299). When dioxathion was administered to three successive generations of albino rats at a dietary level of from 3 to 10 ppm, no abnormalities were observed in rats of any generation (Kennedy, Frawley, and Calandra 1973/Ex. 1–340). A 2-year carcinogenicity bioassay involving rats and mice fed dioxathion had negative results (National Cancer Institute 1978, Technical Report Series 125:43).

Human volunteers who ingested 0.075 mg/kg/day of dioxathion for as long as 60 days had no symptoms related to plasma or blood cholinesterase activity, while those ingesting 0.15 mg/kg/day exhibited a slight decrease in plasma

cholinesterase activity (Frawley, Weir, Tusing et al. 1963/Ex. 1–317). The World Health Organization has estimated an acceptable daily intake for humans of 0.0015 mg dioxathion/kg (WHO 1967, as cited in ACGIH 1986/Ex. 1–3, p. 219).

Based on this evidence in humans and animals, OSHA is proposing an 8-hour TWA PEL of 0.2 mg/m³ for dioxathion in construction, maritime, and agriculture. The Agency is also proposing a skin notation for this substance. OSHA preliminarily concludes that these limits are necessary to protect workers in these sectors from the significant risk of metabolic effects associated with inhalation and oral exposure and with dermal penetration of this substance. The Agency believes that these limits will substantially reduce these significant risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for dioxathion consistent across all regulated sectors.

DISULFIRAM

CAS: 97–77–8; Chemical Formula: $C_{10}H_{20}N_2S_4$

H.S. No. 1151

OSHA has no limit for disulfiram in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 2 mg/m³ for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 2 mg/m³ for disulfiram in the construction, maritime, and agriculture sectors. This is the limit recently established for this substance in general industry.

Disulfiram is a white, crystalline powder that has a slight odor (HSDB 1990). This substance is used as a rubber accelerator and vulcanizer, a seed disinfectant, a fungicide, and as an alcohol deterrent in alcohol aversion therapy (HSDB 1990). When used to treat alcoholics, disulfiram is marketed under many trade names, including Antabuse and Antalcol (RTECS 1990). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Disulfiram causes vasodilation of the vessels of the face and neck, increased heart and respiratory rates, decreased blood pressure, and nausea and vomiting. The LD_{50} in rats is 500 mg/kg (RTECS 1990). This compound is highly toxic when injected intraperitoneally, with an LD_{50} of 75 mg/kg for mice (RTECS 1990). Acutely poisoned animals show ataxia, hypothermia, and flaccid paralysis (IARC 1976, Vol. 12, p. 89). The effects of high-dose ingestion include degenerative changes in the liver and kidneys; very high doses of disulfiram can cause leukopenia and marked hypoplasia or aplasia of the bone marrow (Brieger 1947/Ex. 1–717). In the most seriously afflicted animals, the blood urea nitrogen sometimes increased and the thymol turbidity test was positive (Brieger 1947/Ex. 1–717). Rats given chronic doses of disulfiram (1000 to 2000 mg/kg) in the diet had reduced growth rates, decreased longevity, and reduced reproductive capabilities (Holck et al. 1970, in IARC 1976, Vol. 12, p. 89). Oral, parenteral, or subcutaneous administration of disulfiram to rats or mice during pregnancy leads to embryotoxic and/or developmental effects in the offspring (RTECS 1990; Shepard 1980, Catalog of Teratogenic Agents, 3rd ed., p. 395).

Adverse health effects occur in humans who consume alcohol and are simultaneously exposed to disulfiram. For individuals who drink alcohol and are exposed to disulfiram, the symptoms of exposure are facial vasodilation, tachycardia, tachypnea, nausea, vomiting, pallor, and hypotension. High doses of disulfiram can induce convulsions, cardiac arrhythmias, and myocardial infarction, and the compound has also been associated with polyneuropathy, peripheral neuritis, and skin eruptions (Grant 1986, p. 367; Bouldin et al. 1980, in Neuropathol. Appl. Neurobiol. 6(2):155–160). Disulfiram also causes acneform eruptions, urticaria, lassitude, fatigue, tremor, reduced sexual potency, a garlic-like odor of the breath, and fatal hepatotoxicity (Goodman 1980, Pharm. Basic Therapeutics, 6th ed. p. 388; Berlin 1989, in Alcohol 24(3):241–246). A recent study (Rudzki, Rebarrdel, and Grzywa 1989, in Contact Dermatitis 21(2):121–122) described four cases of skin sensitization caused by disulfiram among pharmaceutical workers in Poland. Disulfiram has induced severe and acute hepatotoxicity in many alcoholic patients on alcohol aversion therapy. If the drug is not withdrawn at the earliest signs of liver toxicity, death caused by massive hepatic necrosis occurs (Berlin 1989, in Alcohol 24(3):241–246). A recent review (Dalvi 1988, in Vet. Human Toxicol. 30(5):480–482) of disulfiram's toxicity reports that workers exposed to this substance in the agriculture, rubber, plastics, and lubricating oils industries develop dermatitis, irritation of the mucous membranes, and gastrointestinal and central nervous system disturbances. In animals, liver and renal tubule necrosis and central nervous system demyelination were also reported. This review also reported disulfiram-induced diseases of the thyroid and gastrointestinal, ocular, and cardiovascular system in workers exposed to disulfiram.

Based on this evidence, OSHA is proposing a PEL of 2 mg/m³ as an 8-hour TWA for disulfiram. The Agency preliminarily concludes that this limit will protect workers in the construction, maritime, and agriculture industries from the significant risk of adverse effects, including dermatitis, liver toxicity, and nervous system effects, that are associated with exposure to disulfiram. OSHA believes that this limit will substantially reduce this significant risk for workers in these sectors and that these effects clearly constitute material impairments of health. In addition, promulgation of this limit will make the PEL for disulfiram consistent across all OSHA-regulated sectors.

ETHION

CAS: 563–12–2; Chemical Formula: $C_9H_{22}O_4P_2S_4$

H.S. No. 1160

OSHA has no permissible exposure limit for ethion in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 0.4 mg/m³, with a skin notation, for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 0.4 mg/m³, and a skin notation, for ethion in the construction, maritime, and agriculture industries. This is the limit recently established for ethion in general industry.

Pure ethion is an odorless and colorless liquid; however, technical-grade ethion has a very disagreeable odor. Ethion is an insecticide and miticide that is sold in a variety of forms, including 25-percent wettable powder, 2-, 3-, and 4-percent dusts, 5-percent granules, in several oil solutions, and in combination with other chemicals (ACGIH 1986, p. 236; Gosselin, Smith, and Hodge 1984, p. II–297). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Like all organophosphorus insecticides, ethion causes cholinesterase inhibition in humans and animals. The oral LD_{50} in rats is 13 mg/kg, and the dermal LD_{50} in rabbits is 890 mg/kg (RTECS 1990). Inhalation studies report LC_{50} values of 710 mg/m³ for female rats exposed to 25-percent wettable powder dust for 1 hour and 7200 mg/m³ for male rats similarly exposed (Niagara Chemical Division, FMC Corp., as cited in ACGIH 1986/Ex. 1–3, p. 236). Instillation of 0.05 ml ethion

in the rabbit eye is immediately irritating but does not cause corneal scarring (ACGIH 1986, p. 236). Dietary studies in rats fed 600, 1000, or 1500 ppm reported complete cholinesterase inhibition; 300 ppm in the diet produced marked cholinesterase inhibition (Association of American Pesticide Control Officials, Inc. 1969, as cited in ACGIH 1986/Ex. 1–3, p. 236). Goats were injected intravenously with 2, 5, or 10 mg/kg doses of ethion. At the 2-mg/kg dose, no signs of toxicity were observed, although blood cholinesterase activity fell to 65 percent of baseline. At higher doses, however, signs of severe toxicity were evident and cholinesterase activity decreased to 10 percent of baseline (in the 5-mg/kg group) or 5 percent (in the 10-mg/kg group) (Mosha and Gyrd-Hansen 1990, in Vet. Hum. Toxicol. 32(1):6–8).

The lowest toxic oral dose of ethion in humans is 100 μg/kg; this exposure caused cholinesterase inhibition (RTECS 1990). Ethion poisonings have been reported in farm workers harvesting grapes and peaches (State of California Department of Industrial Relations, as cited in ACGIH 1986/Ex. 1–3, p. 236).

Based on this evidence, OSHA is proposing a PEL of 0.4 mg/m³ as an 8-hour TWA, and a skin notation, for ethion in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that these limits are necessary to protect exposed workers from the significant risks of organophosphate poisoning and cholinesterase inhibition associated with exposure to this substance. The Agency notes this substance's potential for dermal absorption and is proposing a skin notation to protect against the risk of systemic toxicity by this route of exposure. OSHA believes that the systemic poisoning and cholinesterase inhibition caused by overexposure to ethion constitute material health impairments. In addition, promulgation of this limit will make the PEL for ethion consistent across all OSHA-regulated sectors.

FENAMIPHOS

CAS: 22224–92–6; Chemical Formula: $C_{13}H_{22}NO_3PS$

H.S. No. 1173

OSHA has no limit for fenamiphos in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 0.1 mg/m³ for this substance, with a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA limit of 0.1 mg/m³, with a skin notation, for fenamiphos in the construction, maritime, and agriculture industries. This is the limit recently established in general industry.

Fenamiphos, also called Nemacur®, is a tan-colored, waxy solid. It is used as a nematocide and insecticide (ACGIH 1986, p. 265; Farm Chemicals Handbook 1990, p. C207). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Fenamiphos is a cholinesterase inhibitor that produces both central and peripheral cholinergic reactions. The oral LD_{50} in rats is 8 mg/kg, and the LC_{50} in the same species is 91 mg/m³ for 4 hours (RTECS 1990). The dermal LD_{50} in rabbits is 178 mg/kg (RTECS 1990). Rats exposed to fenamiphos aerosol at concentrations of 0.03, 0.25, or 3.5 mg/m³ of air for 3 weeks exhibited no signs of toxicity. At 3.5 mg/m³, however, rats showed significant depression of plasma cholinesterase; 0.25 mg/m³ was the highest no-effect concentration observed (Kimmerle 1982c, as cited in ACGIH 1986/Ex. 1–3, p. 265). Rats fed fenamiphos in the diet at dose levels of 4, 8, 16, or 32 ppm for 90 days showed no effects other than cholinesterase inhibition. Decreases in cholinesterase activity decreased in a dose-dependent manner (Loser and Kimmerle 1988, as cited in EPA 1987 Health Advisory, ODW). Rats were fed fenamiphos at 3, 10, or 30 ppm for 2 years; in the high-dose group, statistically significant increases in the weights of the thyroid gland, lungs, and hearts were seen. Cholinesterase activity in the high-dose group was decreased by 28 to 60 percent (Loser and Kimmerle 1988, in EPA 1987). Pregnant rabbits were gavaged with 0.1, 0.3, or 1.0 mg/kg/day fenamiphos on days 6 to 18 of gestation. In the high-dose group, fetal death and resorptions were reported, and there was a significant increase in the incidence of skeletal abnormalities in the offspring (MacKenzie et al. 1982, in EPA 1987). Carcinogenicity bioassays in mice and rats were negative.

There are no reports of human poisonings caused by exposure to fenamiphos, and no quantitative data are available relating adverse health effects to measurable airborne concentrations of this substance.

OSHA is proposing a PEL for fenamiphos of 0.1 mg/m³ as an 8-hour TWA in construction, maritime, and agriculture; the Agency believes that this limit is necessary to protect against the significant risk of anticholinesterase effects presented by exposure to this substance. A skin notation is also proposed based on the evidence of fenamiphos's systemic toxicity via percutaneous absorption. The Agency believes that these limits are necessary to substantially reduce the risk of cholinesterase inhibition, which constitutes a material impairment of health. In addition, promulgation of this limit will make the PEL for fenamiphos consistent across all OSHA-regulated sectors

FENSULFOTHION (DASANIT)

CAS: 115–90–2; Chemical Formula: $C_{11}H_{17}O_4PS_2$

H.S. No. 1174

OSHA has no limit for fensulfothion in construction, maritime, or agriculture. The ACGIH has a TLV®–TWA of 0.1mg/m³ for this substance. There is no NIOSH REL, but NIOSH concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA limit of 0.1 mg/m³ for fensulfothion in the construction, maritime, and agriculture sectors. This is the limit recently established for this substance in general industry.

Fensulfothion, also called Dasanit, is a brown liquid at room temperature. It is used as an insecticide and nematocide (ACGIH 1986, p. 286; Hawley's 1987, p. 510). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Fensulfothion is an organophosphorus insecticide that causes cholinesterase inhibition in humans and animals. The oral LD_{50} in rats is 2 mg/kg, and the dermal LD_{50} in the same species is 3 mg/kg (RTECS 1990). Rats fed 5 or 20 ppm fensulfothion for 17 months showed increased mortality, and animals in the high-dose group showed reduced growth rates; even at a dietary level of 1 ppm, blood, plasma, and brain cholinesterase activity was inhibited (Hayes 1982, p. 402). Tests in mice and rabbits have shown no embryotoxic, reproductive, or mutagenic effects.

Fensulfothion has been responsible for a number of fatalities in adults and children. A farmer applying fensulfothion developed diarrhea and vomiting and subsequently died; at autopsy, decreased cholinesterase levels and pulmonary edema were seen (Hayes 1982, p. 403). A family of five was poisoned by fensulfothion after it was used as a household pesticide; one of the family members died (Hayes 1982, p. 403). Dermal studies have shown irritation without cholinesterase effects from 2-hour, twice-daily applications of a 5-percent granular formulation to the forearms of three subjects. Systemic absorption through the lungs has been demonstrated after inhalation of

fensulfothion aerosols (ACGIH 1986/Ex. 1–3, p. 266).

Based on this evidence, OSHA is proposing a PEL of 0.1 mg/m³ TWA for fensulfothion in construction, maritime, and agricultural operations to reduce the significant risks of metabolic effects and skin irritation potentially associated with exposure to this substance. The Agency believes that this limit will substantially reduce these risks among workers in these sectors and that skin irritation and cholinesterase inhibition are material impairments of health. In addition, promulgation of this limit will make the PEL for fensulfothion consistent across all OSHA-regulated sectors.

FENTHION

CAS: 55–38–9; Chemical Formula: $C_{10}H_{15}O_3PS_2$

H.S. No. 1175

OSHA has no limit for fenthion in the construction, maritime, or agriculture industries. The ACGIH has a TLV®–TWA of 0.2 mg/m³, with a skin notation, for this substance. NIOSH has no REL. OSHA is proposing a limit of 0.2 mg/m³ as an 8-hour TWA, and a skin notation, for fenthion. This is the limit recently established for this substance in general industry.

Fenthion is an oily, yellow- to tan-colored liquid that smells slightly like garlic. It is used extensively as a structural control insecticide (ACGIH 1986, p. 267; Hayes 1982, p. 367). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

The primary health effect associated with exposure to fenthion is plasma cholinesterase inhibition. The oral LD_{50} in rats is 180 mg/kg, and the lowest lethal concentration in the same species is 1 g/m³ for 2 hours (RTECS 1990). The dermal LD_{50} in rats is 330 mg/kg (Farm Chemicals Handbook 1976/Ex. 1–1147b). Rats given single intramuscular injections of 5, 25, or 50 mg/kg fenthion exhibited both enduring electroretinogram changes (ERG) and changes in cholinesterase activity; pseudocholinesterase activity in the plasma dropped to 50 percent of normal on the fourth day after injection. The retinal effects of fenthion persisted for as long as 50 days (Imai 1975/Ex. 1–910). Groups of Donryn rats fed 300 ppm fenthion daily showed symptoms of organophosphate intoxication, including nervousness, general spasms, diarrhea, salivation, and ophthalmologic effects (Kawai, Tojo, Miyazawa et al. 1976/Ex. 1–1157). The no-effect inhalation level for rats is 1 mg/m³ for exposures to the aerosol of 6 hours/day, 5 days/week for 3 weeks; at a concentration of 3 mg/m³, cholinesterase inhibition was found (Thyssen 1979, as cited in ACGIH 1986/Ex. 1–3, p. 267). Single and repeated applications of the compound produced no delayed neurotoxic effects in chickens (WHO 1972, as cited in ACGIH 1986/Ex. 1–3, p. 268). Two-year feeding studies of rhesus monkeys showed plasma cholinesterase inhibition at the highest oral dose given, i.e., 0.2 mg/kg/daily (Rosenblum 1980, as cited in ACGIH 1986/Ex. 1–3, p. 268). Fenthion has caused skin tumors in mice (RTECS 1990).

The lowest lethal dose in humans is reported to be 50 mg/kg (RTECS 1990). Four out of six Nigerian agricultural workers who sprayed fenthion 6 hours/day for 5 days showed no reduction in cholinesterase activity; however, the other two workers showed cholinesterase activity decreases of 37.5 and 12.5 percent, respectively. Eight of nine U.S. workers spraying fenthion to control mosquitoes showed progressive declines in plasma cholinesterase activity but not in blood cholinesterase activity (Hayes 1982, p. 369).

Based on this evidence, OSHA is proposing an 8-hour TWA limit of 0.2 mg/m³, with a skin notation, for fenthion in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that these limits are necessary to protect workers in these sectors against the significant risk of cholinergic effects associated with exposure to this substance. A skin notation is proposed because of evidence that fenthion is toxic when absorbed through the skin. OSHA believes that cholinesterase inhibition constitutes a material impairment of health. Promulgation of this limit will also make the PEL for fenthion consistent across all OSHA-regulated sectors.

METHOMYL

CAS: 16752–77–5; Chemical Formula: $C_5H_{10}N_2O_2S$

H.S. No. 1245

OSHA has no limit for methomyl in the construction, maritime, or agriculture industries. The ACGIH has a TLV®–TWA of 2.5 mg/m³ for this substance. There is no NIOSH REL, but NIOSH concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing a PEL of 2.5 mg/m³ as an 8-hour TWA for methomyl in the construction, maritime, and agriculture industries. This is the limit recently established for methomyl in general industry.

Methomyl, also called Lannate, is a white, crystalline solid with a slightly sulfurous odor. This substance is used as an insecticide on vegetables, fruits, and ornamentals (ACGIH 1986, p. 363; Farm Chemicals Handbook 1990, p. C190). When used in pesticidal applications and in accordance with directions on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Methomyl is a cholinesterase-inhibiting carbamate insecticide. The acute oral approximate lethal dose (ALD) for methomyl in rats is 26 mg/kg (Kennedy, Ferenzi, and Burgess 1986, in J. Appl Toxicol. 6(3):145–148). The LC_{50} in rats is 77 ppm, and the dermal LD_{50} in rabbits is 5880 mg/kg (RTECS 1990). Applied to the skin of guinea pigs, methomyl caused no appreciable irritation or sensitization. Instillation of a 10-percent solution of methomyl in propylene glycol or of the dry material into rabbit eyes caused mild conjunctivitis without corneal injury. However, marked pupillary constriction, a health effect produced commonly by cholinesterase inhibitors, was observed (E.I. du Pont de Nemours and Co., Inc., as cited in ACGIH 1986/Ex. 1–3, p. 363). Rats fed methomyl at dietary levels of 200 or 400 ppm for 22 months showed decreased hemoglobin levels and, in the high-dose animals, a statistically significant increase in testis to body weight ratios and histopathological changes to the kidneys and spleens (NRC 1983, Vol. 5, p. 58). No depression of cholinesterase activity could be detected in rats fed at levels of 0, 200, 400, or 800 ppm methomyl for 79 days. In dogs, 90-day and two-year feeding studies showed no effects at 0, 50, 100, or 400 ppm; however, animals fed at 1000 ppm did demonstrate toxicity. Similar studies of rats have shown kidney, liver, and spleen damage at higher feeding levels, but the no-effect level for both rats and dogs has been reported to be 100 ppm (Kaplan and Sherman 1977/Ex. 1–337).

In humans, methomyl causes mild conjunctivitis when the dust contacts the eyes (HSDB 1990), and inhalation of powdered methomyl has caused numerous cases of poisoning in workers in several countries (Hayes 1982, p. 456). Eleven of 102 workers in a pesticide plant had been hospitalized for pesticide poisoning, and most of these cases were attributed to methomyl poisoning (Morse et al. 1979, in Hayes 1982, p. 456).

Based on this evidence, OSHA is proposing a PEL of 2.5 mg/m³ (8-hour TWA) for methomyl in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that this limit will protect exposed workers in

these sectors from the risk of cholinesterase inhibition associated with exposure to this substance. The Agency believes that this limit will substantially reduce the significant risk of cholinergic effects, which constitute material impairments of health. In addition, promulgation of this limit will make the PEL for methomyl consistent across all OSHA-regulated sectors.

MONOMETHYL ANILINE

CAS: 100–61–8; Chemical Formula: $C_6H_5NHCH_3$

H.S. No. 1280

OSHA's PEL for monomethyl aniline (N-methyl aniline) in the construction and maritime industries is 2 ppm, measured as an 8-hour TWA; this limit has a skin notation, indicating that it can readily penetrate the skin. There is no limit for monomethyl aniline in agriculture. The ACGIH has a TLV®–TWA of 0.5 ppm TWA for monomethyl aniline, also with a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing to reduce the limit for this substance to 0.5 ppm in the construction and maritime industries, to retain the skin notation, and to extend the limit and notation to agriculture. These are the limits recently established for monomethyl aniline in general industry.

Monomethyl aniline is a colorless liquid that turns reddish-brown after standing. It is used as an acid acceptor, a solvent, and in organic synthesis (ACGIH 1986, p. 375; Hawley's 1987, p. 758).

Monomethyl aniline causes methemoglobinemia in humans and animals. The lowest lethal oral dose in rabbits is 280 mg/kg (RTECS 1990). Treon, Deichmann, Sigmon, and Associates (1949/Ex. 1–676) found that monomethyl aniline applied to the skin of laboratory animals resulted in systemic poisoning. A later study by Treon and Associates (1950/Ex. 1–533) showed that guinea pigs, rabbits, and rats died from 130 or fewer 7-hour exposures to a 7.6-ppm concentration of monomethyl aniline. Before death, these animals showed prostration, labored breathing, and cyanosis, and the rabbits and cats developed methemoglobinemia. In the same study, a monkey survived the same number and length of exposures at 2.4 ppm, and a dog survived 50 exposures to 86 ppm. Autopsy revealed pulmonary effects and liver and kidney damage in these animals (Treon, Sigmon, Wright et al. 1950/Ex. 1–533).

Data on human exposures to monomethyl aniline are scarce. A recent study (Kaminskaia, Soboleva, Kolpakov, Datsishina, and Zhilko 1989, in Vrach-Delo 8:112–113) reports that Russian workers engaged in the production of this substance had a higher than normal incidence of methemoglobinemia, disorders of the pulmonary system, and cardiovascular changes.

Based on this evidence, OSHA is proposing a 0.5-ppm TWA limit, with a skin notation, for this substance in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that these limits will protect workers in these sectors from the significant risk of metabolic and blood effects, such as methemoglobinemia, that are potentially associated with exposure to monomethyl aniline. The proposed skin notation will protect workers from the risk of systemic poisoning posed by the skin absorption of this substance. OSHA believes that methemoglobinemia constitutes a material health impairment and that the proposed limit will reduce this risk substantially. In addition, promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

NITRIC OXIDE

CAS: 10102–43–9; Chemical Formula: NO

H.S. No. 2116

In general industry, construction, and maritime, OSHA's permissible exposure limit for nitric oxide is 25 ppm as an 8-hour TWA. There is no limit in agriculture. The ACGIH's TLV®–TWA for nitric oxide is 25 ppm as an 8-hour TWA; NIOSH has a REL of 25 ppm as a 10-hour TWA, and concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 25 ppm for nitric oxide in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Nitric oxide is a colorless nonflammable gas used to make nitric acid. It is also used in rayon bleaching and as a stabilizer (ACGIH 1986, p. 429; Merck 1983, p. 944).

Exposure to nitric oxide causes central nervous system effects, lung damage, and methemoglobin formation. The LC_{50} in rats is 1068 mg/m³ (870 ppm) for an unspecified time (RTECS 1990). In rabbits exposed for 15 minutes, the LC_{50} is 315 ppm (RTECS 1990). Guinea pigs survived exposure to a 175-ppm concentration of nitric oxide for 120 to 150 minutes; no exposure-related effects were seen (NIOSH Criteria Document 1976). Mice exposed to a 2500-ppm concentration of nitric oxide for 6 or 7 minutes showed signs of narcosis; death occurred within 12 minutes (NIOSH/OSHA Occupational Health Guideline 1981, p. 4). Studies in experimental animals (species unspecified) showed that 6 hours of exposure to a 322-ppm concentration produces a 60 percent methemoglobin level (Paribok and Grokholskaya 1962, in ACGIH 1986, p. 429). Mice exposed to a 10-ppm concentration of nitric oxide for 2 hours/day, 5 days/week for various periods up to 30 weeks showed pronounced emphysematous changes (Holt et al. 1979, in Clayton and Clayton 1981, p. 4101). Nitric oxide is mutagenic both in bacterial and mammalian test systems (RTECS 1987).

In humans, exposure to a 25-ppm concentration of nitrogen oxides can cause pulmonary signs and symptoms after 8 hours; signs and symptoms may not be manifest for as long as 5 to 48 hours after exposure (Braker and Mossman 1980, p. 514). Exposure to a 100- to 150-ppm concentration for 30 to 60 minutes causes delayed-onset pulmonary edema, and at concentrations of 200 to 700 ppm, the pulmonary damage may be severe enough to cause death 5 to 8 hours after exposure (Braker and Mossman 1980, p. 514). Two cases of human poisoning have been reported in which the victims were anesthetized with nitrous oxide contaminated with 1.5 percent nitric oxide. The two individuals developed cyanosis and methemoglobinemia, both of which were attributed to the nitric oxide; one patient died 18 hours after exposure and the other subsequently recovered (NIOSH 1976, pp. 35–36). Methemoglobin levels ranging from 2 to 3 percent have been detected in arc welders exposed to nitric oxide during welding, and levels as high as 44 percent have been seen in silo fillers and other agricultural workers. The normal methemoglobin level is less than 1 percent (Rom 1983, p. 227). Chronic exposure to concentrations of nitrogen oxides (including nitric oxide) too low to cause edema may cause chronic respiratory irritation, corrosion of the teeth, and weakness (Braker and Mossman 1980, p. 515).

Based on this evidence in humans and animals, OSHA preliminarily concludes that nitric oxide causes lung damage and methemoglobinemia in exposed individuals. The Agency believes that, in the absence of a limit for nitric oxide, workers in agriculture are at significant risk of experiencing these effects. OSHA preliminarily finds that the proposed limit of 25 ppm as an 8-hour TWA is necessary to reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

NITROBENZENE
CAS: 98–95–3; Chemical Formula: $C_6H_5NO_2$
H.S. No. 2117

In general industry, construction, and maritime, OSHA's permissible exposure limit for nitrobenzene is 1 ppm (5 mg/m³) as an 8-hour TWA; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit for nitrobenzene in agriculture. The ACGIH has a TLV®–TWA of 1 ppm as an 8-hour TWA, also with a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the PEL being proposed. OSHA is proposing to establish an 8-hour TWA PEL of 1 ppm, with a skin notation, for nitrobenzene in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Nitrobenzene is an oily, colorless to pale yellow liquid with a strong odor similar to that of bitter almonds. Nitrobenzene is used in the manufacture of aniline; as a solvent for cellulose esters; in the esterification of cellulose acetate; as an ingredient of soaps, metal polishes, and shoe polishes; in the manufacture of benzidine, quinoline, and azobenzene; as a substitute for almond essence; as a preservative in spray paints; and as an ingredient in perfume (Grayson 1985, p. 790; Hawley's 1987, p. 826; HSDB 1988).

Nitrobenzene causes eye and skin irritation, cyanosis, and blood changes in humans and animals. This substance can also cause kidney and liver effects and central nervous system depression. In addition, testicular damage has been reported in animals. The oral LD_{50}s in rats and mice are 489 mg/kg and 590 mg/kg, respectively (RTECS 1987). The dermal LD_{50} in rats is 2100 mg/kg, and the lowest lethal dermal dose in rabbits is 600 mg/kg (RTECS 1987). Applied to the eyes or skin of rabbits for 24 hours, nitrobenzene caused only mild irritation (RTECS 1987). The leading toxic effect of nitrobenzene exposure is the formation of methemoglobin, with concomitant cyanosis and the risk of death from respiratory failure (Browning 1965, in HSDB 1988). Rabbits given subcutaneous injections of 0.75 g of nitrobenzene showed marked decreases in hematocrit and hemoglobin levels; a tendency toward spherocytosis was seen, and Heinz bodies were observed in 17 out of 27 of these animals. Other organ changes observed at autopsy included fatty infiltration of the kidneys and liver and nutmeg-sized nodules in the liver (Browning 1965, in HSDB 1988). Rats given intraperitoneal injections of 0.18 g/kg nitrobenzene showed signs of central nervous system depression (Browning 1965, in HSDB 1988). At an oral dose of 200 mg/kg, one rabbit died; 30 hours later, fat deposits were seen in the tissues and gastrointestinal tract (Browning 1965, in HSDB 1988). Degenerative testicular lesions were observed in rats given single oral doses of 50 to 450 mg/kg of nitrobenzene; hepatic centrilobular necrosis was also seen in these animals at autopsy (Beauchamp 1983, in Proctor, Hughes, and Fischman 1988, p. 368; Bond et al. 1981 in HSDB 1988). Chronic exposure to low concentrations of nitrobenzene caused an increase in the number of erythrocytes, methemoglobinemia or hemoglobinemia, albuminuria, and bile in the urine (Browning 1965, p. 300, in HSDB 1988). Rats and mice exposed to nitrobenzene concentrations of 5, 16, or 50 ppm for 6 hours/day, 5 days/week for 90 days showed a dose-related increase in methemoglobinemia and splenic changes; in the high-dose group of rats, degeneration of the seminiferous epithelia was seen (Hamm, Phelps, Raynur, and Irons. Toxicol. 4:181, 1984). Rats given a single oral dose of 300 mg/kg nitrobenzene showed complete cessation of sperm production 32 to 48 days later; 100 days after the administration of nitrobenzene, more than 90 percent of the seminiferous epithelium had regenerated (Levin, Bosakowski, Earle, and Butterworth 1988, in Toxicol. 53:219–230). Administered subcutaneously to pregnant rats during preimplantation and placentation, nitrobenzene caused a delay in embryogenesis, abnormal placentation, and gross abnormalities in the offspring (National Research Council 1981, Vol. 4, p. 227).

In humans, nitrobenzene causes cyanosis, liver damage, and blood changes. The lowest oral dose reported to be toxic in women is 200 mg/kg; this dose produced behavioral symptoms and affected the cardiovascular and pulmonary systems (RTECS 1987). Nitrobenzene can be absorbed through the skin in lethal amounts (Gosselin, Smith, and Hodge 1984, p. II–214). Ocular effects caused by nitrobenzene exposure include methemoglobinemia-induced brown discoloration of the vessels of the fundus and conjunctiva (Grant 1986, p. 663). Hepatotoxicity, as evidenced by changes in liver function (including hyperbilirubinemia and decreased prothrombin activity), has been seen in humans exposed to nitrobenzene (Beauchamp 1983, in Proctor, Hughes, and Fischman 1988, p. 368). The signs and symptoms of inhalation exposure to nitrobenzene include cyanosis, headache, vertigo, nausea, visual disturbance, stupor, coma, and death from respiratory failure (Arena 1974, in HSDB 1988). Workers in a chemical plant that had an average nitrobenzene concentration of 6 ppm did not experience signs or symptoms of nitrobenzene poisoning, although one or two cases of headache or vertigo were reported among the 39 workers (Pascéri et al. 1958, in ACGIH 1986, p. 431). Tests of the blood of these workers showed a low level of methemoglobinemia and sulfhemoglobinemia and the presence of some Heinz bodies (Pascéri et al. 1958, in ACGIH 1986, p. 431). When these workers were exposed to nitrobenzene concentrations of close to 40 ppm, however, they did experience toxic episodes (Pascéri et al. 1958, in ACGIH 1986, p. 431). Two cases of occupational overexposure to nitrobenzene have resulted in the development of mild anemia, believed to be of the hemolytic type (Browning 1965, p. 301, in HSDB 1988).

Based on this evidence in humans and animals, OSHA preliminarily concludes that nitrobenzene causes eye and skin irritation, cyanosis, blood dyscrasias, central nervous system depression, and splenic and hepatic effects. Accordingly, the Agency believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these effects. OSHA preliminarily finds that the proposed 8-hour TWA PEL of 1 ppm, and a skin notation, are necessary to substantially reduce these significant risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for nitrobenzene consistent across all regulated sectors.

p-NITROCHLOROBENZENE
CAS: 100–00–5; Chemical Formula: $NO_2C_6H_4Cl$
H.S. No. 1288

In the construction and maritime industries, OSHA currently has an 8-hour TWA limit of 1 mg/m³, and a skin notation, for p-nitrochlorobenzene (PNCB). There is no limit for this substance in agriculture. The ACGIH has a TLV®–TWA of 0.5 mg/m³ (3 mg/m³), with a skin notation for this substance. NIOSH has no REL for nitrochlorobenzene. The Agency is proposing a 1 mg/m³ 8-hour TWA limit, and a skin notation, for PNCB in agriculture.

Para-Nitrochlorobenzene is a yellow crystalline solid that has a sweet odor. It is used in the manufacture of agricultural chemicals, dyes, and rubber and as a chemical intermediate (ACGIH 1986, p. 321.1(86); Hawley's 1987, p. 269).

The primary hazard associated with exposure to PNCB is

methemoglobinemia. The oral LD_{50} in rats is 420 mg/kg, and the dermal LD_{50} in the same species is 16 g/kg (RTECS 1990). PNCB was absorbed through rabbit skin in sufficient quantity to produce methemoglobinemia (Kubota 1960, as cited in ACGIH 1986/Ex. 1–3, p. 432.2). Rusakov, Korotkova, and Bikbulatov (1973/Ex. 1–660) described the development of sensitization in guinea pigs after dermal application of PNCB. A 4-hour inhalation exposure of rats (heads only) showed that the lethal concentration of PNCB is approximately 16.1 mg/L (E.I. du Pont de Nemours & Co., Inc. 1981, as cited in ACGIH 1986/Ex. 1–3, p. 432.2). Head-only exposures to 0.05, 0.29, or 0.64 mg/L PNCB for 6 hours/day, 5 days/week for 2 weeks resulted in spleen-weight increases and blood effects in all groups. In addition, there were dose-related effects in blood methemoglobin levels (i.e., decreased hemoglobin, hematocrit, and red blood cell count values). Microscopic changes in the spleen, bone marrow, and kidneys were seen in the two higher-dose groups, and both pathological degeneration of the seminiferous tubules and abnormal epididymal sperm contents were also observed in these groups (E.I. du Pont de Nemours & Co., Inc. 1984, as cited in ACGIH 1986/Ex. 1–3, p. 432.2). The Monsanto Company (1981, as cited in ACGIH 1986/Ex. 1–3, p. 432.2) reported that gavage administration of PNCB for 90 days at daily doses of 0.3, 10, or 30 mg/kg to male and female rats produced hemolytic effects and spleen changes at all levels, kidney and liver effects at mid- to high-level doses, and hyperplasia of bone marrow and testicular atrophy at the highest dose (30 mg/kg/day). In 1985, Monsanto reported the results of another gavage study in rats. After 2 years of PNCB feeding at 0.1, 0.7, or 5.0 mg/kg/day, animals in the mid-and high-dose groups exhibited hemolytic effects; in addition, mid- and high-dose groups showed microscopic spleen, kidney, and liver changes and, at the highest dose, bone marrow hyperplasia and testicular atrophy (Monsanto Company 1985, as cited in ACGIH 1986/Ex. 1–3, p. 432.2).

Rats fed PNCB at doses of 0, 0.1, 0.7, or 5 mg/kg/day for up to 2 years showed methemoglobinemia at the two highest levels, and animals in the 5-mg/kg/day group had indications of anemia and pigment accumulation in spleen cells. No treatment-related increase in tumors was observed (Monsanto Company 1985, as cited in ACGIH 1986/Ex. 1–3, p. 432.2). In a dietary cancer bioassay, rats and mice were given PNCB at unspecified levels for 2 years (Weisberger, Russfield, Homburger et al. 1978/Ex. 1–535). Only mice were affected, with mice of both sexes showing an increase in vascular tumors at the highest dose and male mice showing an increase in liver tumors at the lowest dose (Weisberger, Russfield, Homburger et al. 1978/Ex. 1–535).

Maternal toxicity was seen in rats given PNCB by gavage at doses of 15 or 45 mg/kg/day on days 9 through 16 of gestation; at the 45-mg/kg level, fetotoxicity and teratogenicity were also observed (Nair, Johannsen, and Schroeder 1985/Ex. 1–752). At 15 mg/kg, maternal toxicity but no fetotoxicity or teratogenic effects occurred; at the lowest dose, the only effect was a small increase in maternal spleen weight. A two-generation reproductive study resulted in a reduced mating index in rats given 0.7 or 5.0 mg/kg/day (Monsanto Company 1984, as cited in ACGIH 1986/Ex. 1–3, p. 432.2). Positive responses were observed in a mutation assay of L5178Y TK mouse lymphoma cells (both with and without metabolic activation) and in a microbial assay of *Salmonella* strain TA 1535 (in the absence of metabolic activation); however, no evidence of mutagenicity was noted in assays of three other *Salmonella* strains or in assays of Chinese hamster ovary cells, rat hepatocyte primary culture/DNA repair, or rat bone marrow cell clastogenesis (Monsanto Company 1980–1984, as cited in ACGIH 1986/Ex. 1–3, p. 432.2). PNCB produced DNA damage in the liver, kidney, and brain cells of rats after a single intraperitoneal dose of 30 to 1000 mg/kg (Cesarone, Bolognesi, and Santi 1983/Ex. 1–542) and in cultured hepatocytes at 1.5 hours after a 3-hour treatment (Cesarone, Fugassa, Galle et al. 1984/Ex. 1–541).

p-Nitrochlorobenzene may be absorbed through the lungs and skin in humans to produce methemoglobinemia. Reports of industrial exposures indicate that overexposure causes cyanosis, weakness, and headache (Saita and Moreo 1958/Ex. 1–930; Renshaw and Ashcroft 1926/Ex. 1–522). In a study of workers exposed to average concentrations of PNCB of 55, 125, or 143 ppm and to a 23-ppm concentration of a PNCB-nitrophenol mixture, the authors concluded that the mixed exposure did not produce chronic intoxication but did cause increased methemoglobin, the appearance of Heinz bodies, headache, vertigo, and occasional eczema; these effects could not be attributed definitely either to skin absorption or to the level of PNCB in the mixture (Pascéri, Magos, and Batskor 1958/Ex. 1–521). No data were reported for the p-nitrochlorobenzene exposures only (Pascéri, Magos, and Batskor 1958/Ex. 1–521). Severe allergic dermatitis is reported to be frequent in workers handling PNCB (Gosselin, Smith, and Hodge 1984, p. II–214).

Based on this evidence, OSHA is proposing to extend an 8-hour TWA limit of 1 mg/m³ for p-nitrochlorobenzene, and a skin notation, to agricultural workplaces. The Agency preliminarily concludes that these limits are necessary to protect workers in agriculture from the significant risks of methemoglobinemia and changes in the spleen, liver, and kidney possible at higher exposure levels. OSHA is also proposing a skin notation in agriculture because dermal absorption of PNCB has been shown to cause systemic effects in humans and animals. The Agency preliminarily finds that methemoglobinemia and spleen, kidney, and liver damage constitute material impairments of health. In addition, promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

PARATHION

CAS: 56–38–2; Chemical Formula: $C_{10}H_{14}NO_5PS$

H.S. No. 2122

In general industry, construction, and maritime, OSHA's permissible exposure limit for parathion is an 8-hour TWA of 0.1 mg/m³; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.1 mg/m³, with a skin notation. NIOSH has a 10-hour TWA REL of 0.05 mg/m³ for parathion. OSHA is proposing a limit of 0.1 mg/m³ as an 8-hour TWA, and a skin notation, for parathion in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Parathion is a yellow to deep brown liquid with a faint, garlic-like odor. It is an organophosphorus pesticide that is used as an insecticide and acaricide (Hawley's 1987, p. 874; ACGIH 1986, p. 458). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Parathion causes cholinesterase inhibition in humans and animals. The oral LD_{50} in rats is 2 mg/kg, and the dermal LD_{50} in rabbits is 15 mg/kg (RTECS 1990). In rats, the LC_{50} is 84 mg/m³ for 4 hours (RTECS 1990). Signs and symptoms of acute exposure in animals include lacrimation, tachypnea,

dyspnea, asynergy, convulsions, diarrhea, depression, paresis, tremors, and prostration (HSDB 1990). The highest dietary concentration that produced no symptoms in rats was 10 ppm for 2 years; a dietary level of 25 ppm caused illness (Lehman 1952, in ACGIH 1986, p. 458). Edson observed that rats given a dose equivalent to 0.42 mg/person/day for 84 days showed no effects but that a 46 percent drop in red blood cell cholinesterase levels occurred when these animals were administered a dose equivalent to 4.2 mg/person/day (Edson et al. 1964, in ACGIH 1986, p. 458). Dogs were given dietary doses of 1, 2, or 5 ppm parathion for 24 weeks; 1 ppm produced a minimal but significant reduction in plasma cholinesterase, and a dose of 2 or 5 ppm reduced plasma cholinesterase levels by 60 to 70 percent (NRC 1977, (Drinking Water and Health 1977)).

In humans, the lowest lethal oral parathion dose is 171 μg/kg; the lowest lethal dose by dermal absorption is 7143 μg/kg (RTECS 1990). Fatal human poisonings have occurred through ingestion, skin exposure, and inhalation (Proctor, Hughes, and Fischman 1988, p. 391). The signs and symptoms of parathion overexposure include miosis, blurred vision, tearing, chest tightness, wheezing, laryngeal spasms, and excessive salivation (Proctor, Hughes, and Fischman 1988, p. 392). Gastrointestinal effects such as anorexia, vomiting, abdominal cramps, and diarrhea occur within 15 minutes to 2 hours after ingestion of parathion (Koelle 1963, in Proctor, Hughes, and Fischman 1988, p. 392). The most serious effect of exposure is muscle paralysis; central nervous system effects include giddiness, confusion, ataxia, slurred speech, convulsions, and coma (Taylor 1985, in Proctor, Hughes, and Fischman 1988, p. 392). Four volunteers given oral doses of 1 or 2 mg/person/day did not show signs of cholinesterase inhibition but did show distinctly different rates of excretion (Morgan et al. 1977, in Hayes 1982, p. 381). A 12-year old boy exposed to bed sheets contaminated with parathion suffered an elevated blood pressure and respiratory failure and became comatose; the pupils of his eyes were characteristically contracted (Waldbott 1973, in HSDB 1985). In Colombia, 600 people were poisoned by eating bread baked with flour contaminated with parathion; approximately 25 percent of the victims suffered mild poisoning and experienced symptoms such as abdominal pain, headache, blurred vision, tremor, dizziness, and weakness. Others showed profuse sweating, constricted pupils, and irritability; a few experienced acute pulmonary edema, severe diarrhea, cyanosis, and salivation (Waldbott 1973, in HSDB 1985). Absorption of parathion by inhalation caused a greater depression of cholinesterase activity than absorption by the percutaneous route (Hartwell et al. 1964, in ACGIH 1986, p. 458).

Based on this evidence in humans and animals, OSHA preliminarily concludes that parathion inhibits cholinesterase activity and causes nervous system effects. OSHA preliminarily finds that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that establishing a permissible exposure limit of 0.1 mg/m³ as an 8-hour TWA, and a skin notation, is necessary to significantly reduce the risk of these material health impairments. In addition, promulgation of this limit will make the PEL for parathion consistent across all OSHA-regulated sectors.

PHORATE

CAS: 298–02–2; Chemical Formula: $C_7H_{17}O_2PS_3$

H.S. No. 1319

OSHA has no limit for phorate in the construction, maritime, or agriculture industry. The ACGIH has limits of 0.05 mg/m³ as an 8-hour TWA and 0.2 mg/m³ as a STEL for phorate, with a skin notation. NIOSH has no REL for this substance but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour PEL of 0.05 mg/m³ and a 15-minute STEL of 0.2 mg/m³, and a skin notation, for phorate in these industries. These limits are the same as those recently promulgated for phorate in general industry.

Phorate is an organophosphate that takes the form of a clear liquid and is used as an insecticide. When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Phorate is a highly toxic cholinesterase inhibitor in animals. The oral LD_{50} in rats ranges from 1.1 to 2.0 mg/kg (Hayes 1982, p. 391) and is 6.6 mg/kg in mice (RTECS 1990). The dermal LD_{50} in rabbits is 99 mg/kg (RTECS 1990). Rats exposed to daily doses of phorate showed cholinesterase inhibition effects above 0.15 mg/kg/day but no effects below this level. The no-effect level in dogs is between 0.01 and 0.05 mg/ kg/day (Gaines 1969/ Ex. 1–320). In humans, phorate causes classical symptoms of cholinesterase inhibition, including anxiety, restlessness, headache, emotional and behavioral changes, excessive dreaming, and confusion. In severe cases, exposure to phorate causes loss of muscle coordination, difficulty in breathing, pulmonary rales and rhonchi, convulsions, coma, and death. Several cases of human phorate poisoning have been reported (Hayes 1982, p. 391).

The proposed rule's limits of 0.05 mg/m³ as an 8-hour TWA, supplemented by a STEL of 0.2 mg/m³ and a skin notation, are based on calculations that the no-effect level in humans would lie in the range between 0.21 and 0.7 mg/day, and that use of an appropriate safety factor would suggest an 8-hour limit of 0.05 mg/m³, with a STEL of 0.2 mg/m³, to ensure against excursions greatly in excess of the TWA limit.

OSHA preliminarily finds that limits of 0.05 mg/m³ as an 8-hour TWA and 0.2 mg/m³ as a STEL, and a skin notation, will protect workers exposed to phorate in construction, maritime, and agriculture against cholinesterase inhibition and its associated effects, which include respiratory symptoms, nausea, confusion, and vomiting. The Agency preliminarily concludes that, in the absence of an OSHA limit, phorate-exposed employees are at significant risk of experiencing such effects and that establishing a PEL, STEL, and skin notation will substantially reduce these risks. OSHA believes that cholinesterase inhibition and its symptoms clearly constitute material impairments of health. Promulgation of this limit will also make the PEL for this substance consistent across all OSHA-regulated sectors.

PROPANE

CAS: 74–98–6; Chemical Formula: C_3H_8

H.S. No. 2134

OSHA is not proposing to extend the limit that currently exists for propane in general industry—1000 ppm—to the construction and agriculture sectors. The only known health effect associated with exposure to this substance is asphyxiation, which occurs at levels that far exceed 1000 ppm.

In 1968, the ACGIH TLV® (and thus OSHA's PEL for general industry) was 1000 ppm as an 8-hour TWA. By 1970, however, the ACGIH had deleted its TLV®–TWA for propane in favor of a listing in Appendix E, "Some Simple Asphyxiants." Since 29 CFR 1926, the construction standards, reference the 1970 TLVs, no numerical limit for propane is currently in effect in construction.

The stated goal of this rulemaking is to achieve consistency across all OSHA-regulated sectors; however, there

appears to be no health basis for adopting the general industry limit for propane in other sectors. This case is thus the only exception to this stated objective.

The real hazard with propane is its explosivity, and 29 CFR 1910.110 effectively sets a PEL for propane of approximately 5500 ppm, which is 25% of the LEL (lowest explosive limit) of 22,000 ppm. It is the opinion of the Agency that 1910.110 sufficiently regulates propane, and that a PEL established under 29 CFR 1910.1000 would not therefore reduce a significant health risk.

Because propane is only one of the simple asphyxiants in use in general industry (others are acetylene, argon, helium, hydrogen, propylene, ethanol, etc.), OSHA solicits comment on the policy enunciated here, which is to regulate the simple asphyxiants as explosive substances, i.e., in accordance with their physical, rather than chemical, hazards. Specifically, OSHA seeks comments on whether PELs based on health effects are also needed for these substances to reduce a significant risk to exposed workers.

PROPOXUR (Baygon)

CAS: 114–26–1; Chemical Formula: $C_{11}H_{15}NO_2$

H.S. No. 1337

OSHA has no limit for propoxur in the construction, maritime, and agriculture industries. The ACGIH has established an 8-hour TLV®–TWA of 0.5 mg/m³ for this substance. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA limit of 0.5 mg/m³ for propoxur in the construction, maritime, and agriculture industries. This limit is the same as the limit recently promulgated for general industry.

Propoxur is a white, odorless, crystalline carbamate. It is used as an insecticide, particularly for flea and tick control on animals, and as a molluscicide (ACGIH 1986, p. 499; Hawley's 1987, p. 660). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Like all carbamate insecticides, propoxur causes reversible cholinesterase inhibition. The oral LD_{50}s in male and female rats are 83 and 86 mg/kg, respectively; for both sexes, the dermal LD_{50} is greater than 2400 mg/kg (Gaines 1969/Ex. 1–320). Dietary studies in rats at levels of 7.5 mg/kg/day for 28 days or at 800 ppm for 3 months found no adverse effects (Association of American Pesticide Control Officials, Inc. 1986/Ex. 1–1011). Rats were exposed to propoxur concentrations of 5, 7, 16.7, or 31.7 mg/m³ 6 hours/day, 5 days/week for 12 weeks; animals in the high-dose group showed depressed red blood cell and brain cholinesterase levels, and plasma cholinesterase was depressed by as much as 20 to 30 percent (Association of American Pesticide Control Officials, Inc. 1986/Ex. 1–1011).

In humans, a few cases of mild propoxur poisoning have been reported among sprayers of this insecticide and among residents of propoxur-treated homes (Vandekar, Hedayat, Plestina, and Ahmady 1968/Ex. 1–679). Based on a survey of 696 pet care facility workers, Hines et al. (American Industrial Hygiene Association Journal 50:466–472, 1989) reported an association between exposure to propoxur and an increased frequency of convulsions, muscle twitches, fatigue, and headaches. In a study of human volunteers, a single oral dose of 1.5 mg/kg propoxur caused a depression in red blood cell cholinesterase and gastrointestinal symptoms that disappeared 2 hours after ingestion. Oral doses of 0.75 to 1.0 mg/kg produced no symptoms but did depress erythrocyte cholinesterase (Vandekar, Plestina, and Wilhelm 1971/Ex. 1–680).

OSHA is proposing an 8-hour TWA of 0.5 mg/m³ for propoxur in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risk of cholinesterase inhibition associated with exposure to this substance. OSHA believes that cholinesterase inhibition is a material health impairment. Promulgation of this limit will also make the PEL for this substance consistent across all OSHA-regulated sectors.

RONNEL

CAS: 299–84–3; Chemical Formula: $(CH_3O)_2PSOC_6H_3Cl_3$

H.S. No. 1349

OSHA has a limit of 15 mg/m³ TWA for ronnel in construction and maritime operations. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 10 mg/m³ for this substance. NIOSH has no REL for this substance but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA of 10 mg/m³ for ronnel in agriculture, construction, and maritime. This limit is the same as the PEL recently promulgated for ronnel in general industry.

Ronnel is a white powder. It is used as an insecticide (ACGIH 1986, p. 513; Farm Chemicals Handbook 1990, p. C252). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Ronnel is an indirect cholinesterase inhibitor that affects the blood plasma rather than red cell acetylcholinesterase (Plapp and Casida 1958a/Ex. 1–657). The acute oral LD_{50} in rats is reported as 1250 and 2630 mg/kg for males and females, respectively. The oral LD_{50} in dogs is greater than 500 mg/kg (McCollister, Oyen, and Rowe 1959/Ex. 1–594). Two-year dietary studies of rats fed up to 50 mg/kg/day showed no effect on growth rate, food consumption, survival, or hematopoiesis (McCollister, Oyen, and Rowe 1959/Ex. 1–594). In a study by Gladenko and Stuk (1972, as cited in ACGIH 1986/Ex. 1–3, p. 513), albino rats developed clinical symptoms of motor irritation, tremor, increased auditory and tactile sensitivity, lacrimation, and salivation within 2 weeks of exposure to ronnel concentrations of between 164 and 328 mg/kg; some animals died during the latter part of the study. At exposures below 16.4 mg/kg, no ill effects were observed (Gladenko and Stuk 1972, as cited in ACGIH 1986/Ex. 1–3, p. 513). A 2-year feeding study in dogs exposed to daily doses of 10 mg/kg showed no ill effects except cholinesterase depletion (Worden, Noel, and Mawdesley-Thomas 1972/Ex. 1–583). Oral administration of ronnel to pregnant rats and rabbits has been associated with adverse reproductive and developmental effects (RTECS 1990).

Patch tests of 50 human subjects showed that ronnel has no skin-sensitizing potential (McCollister, Oyen, and Rowe 1959/Ex. 1–594).

OSHA is proposing an 8-hour TWA PEL of 10 mg/m³ for ronnel in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risk of cholinergic effects associated with exposure to this substance. OSHA believes that this limit will substantially reduce this significant risk and that cholinesterase inhibition constitutes a material health impairment. Promulgation of this limit will also make the PEL for ronnel consistent across all OSHA-regulated sectors.

SULPROFOS

CAS: 35400–43–2; Chemical Formula: $C_{12}H_{19}O_2PS_3$

H.S. No. 1380

OSHA has no limit for sulprofos in the construction, maritime, or agriculture industry. The ACGIH has an exposure

limit of 1 mg/m³ as an 8-hour TWA. NIOSH has no REL for this substance but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing an 8-hour TWA of 1 mg/m³ for sulprofos in agriculture, construction, and maritime. This limit is the same as that recently promulgated for general industry.

Sulprofos, also known as the insecticide Bolstar, is a tan liquid (ACGIH 1986, p. 547). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Like all organophosphate pesticides, sulprofos inhibits the enzyme cholinesterase. Kimmerle (1982b, as cited in ACGIH 1986/Ex. 1–3, p. 547) conducted an extensive animal study on the effects of sulprofos. He reported that the acute toxicity of sulprofos is species-dependent: rats have an oral LD_{50} of 100 to 300 mg/kg and mice have an oral LD_{50} of 1600 to 1800 mg/kg. The reported dermal LD_{50}s are greater than 1000 mg/kg in rats and 800 to 1000 mg/kg in rabbits. In rabbits, sulprofos did not irritate the skin or eyes, and it had no dermal sensitization effects in guinea pigs. Inhalation studies showed no fatalities in rats exposed to sulprofos aerosol concentrations of up to 4130 mg/m³ over a period of 4 hours. In a 3-week inhalation study in which rats were exposed to aerosol concentrations of 6, 14, or 74 mg/m³, the two highest concentrations produced cholinergic symptoms; no observable effects were seen at the lowest concentration. Two-year feeding studies by Kimmerle (1982b, as cited in ACGIH 1986/Ex. 1–3, p. 547) in dogs, rats, and mice showed that sulprofos concentrations of 150 ppm, 250 ppm, or 400 ppm were tolerated by all species, with no sulprofos-related tissue changes, signs of toxicity, or oncogenic effects. The overall NOELs were 10 ppm in dogs, 6 ppm in rats, and 2.5 ppm in mice. Kimmerle's ingestion studies in rats and rabbits dosed at levels of 3, 10, or 30 mg/kg/day of sulprofos showed no embryotoxic or teratogenic effects among the offspring of these animals, and a three-generation diet study in rats also produced no adverse reproductive effects. Mutagenic studies reported by the same author in mice were negative. Separate subacute inhalation studies also showed no effects on blood cholinesterase levels in rats exposed to 6 mg/m³ (Zielhuis and van der Kreek 1979/Ex. 1–613). There are no reported cases of poisoning in humans (ACGIH 1986/Ex. 1–3, p. 547).

OSHA is proposing an 8-hour TWA limit of 1 mg/m³ for sulprofos in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that this limit will protect workers in these sectors from the significant risk of cholinesterase inhibition, the most sensitive indicator of exposure to this substance. The Agency believes that this limit will substantially reduce this significant risk, and that cholinesterase inhibition constitutes a material impairment of health. Promulgation of this limit will also make the PEL for this substance consistent across all OSHA-regulated sectors.

TERPHENYLS

CAS: 26140–60–3; Chemical Formula: $C_{18}H_{14}$

H.S. No. 1384

OSHA's PEL in construction and maritime for the terphenyls is 1.0 ppm as a ceiling limit. There is no limit in agriculture. The ACGIH has a 0.5-ppm ceiling limit for these substances. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limit being proposed. OSHA is proposing a PEL in construction, maritime, and agriculture of 0.5 ppm as a ceiling limit for terphenyls. This limit is the same as that recently promulgated for general industry.

Terphenyls are colorless or light yellow solids; commercial preparations contain mixtures of ortho-, meta-, and paraterphenyls. Terphenyls are used as heat transfer fluids and reactor coolants (ACGIH 1986, p. 559).

The terphenyls are primary irritants that cause eye, skin, and respiratory tract irritation. Haley, Detrick, Komesu et al. (1959/Ex. 1–326) reported that mixtures of terphenyls caused conjunctival irritation when instilled into the eyes of rabbits and damaged guinea pig skin following intracutaneous injection. Cornish, Bahor, and Ryan (1962/Ex. 1–410) determined LD_{50} values of 1900, 2400, and greater than 10,000 mg/kg for the ortho-, meta-, and para-terphenyls, respectively. These authors also conducted 30-day feeding studies of rats involving doses of 250 or 500 mg/kg/day of the individual terphenyl isomers. Rats fed ortho-terphenyl showed elevated liver and kidney weight ratios; rats fed meta-terphenyl displayed elevated kidney weight ratios only; and rats fed para-terphenyl showed no elevation in liver or kidney weight ratios. Two studies by Petkau and Hoogstraaten (1965/Ex. 1–432) and Young, Petkau, and Hoogstraaten (1969/Ex.1–459) have shown that the terphenyls have nephrotoxic effects and cause hepatic damage in rats fed 33 mg/kg/day.

Inhalation by rats of high concentrations (66 to 339 ppm) of mixed or individual isomers for periods ranging from 1 hour to 14 days resulted in tracheobronchitis, pulmonary edema, and death at higher doses (Proctor, Hughes, and Fischman 1988, p. 459). Adamson, Bowden, and Wyatt (1969/Ex. 1–293) published a study in which rats exposed to terphenyl aerosols for 7 hours per day at a concentration of 50 mg/m³ (approximately 5 ppm), for a period of 8 days developed morphological changes in their pulmonary cell mitochondria; the number of vacuolated mitochondria was directly related to duration of exposure.

Weeks (1971/Ex. 1–580) and Weeks and Lentle (1970/Ex. 1–682) conducted a clinical survey of 47 workers with ongoing exposure to terphenyl coolant in a nuclear facility. The study represented 122 person-years of occupational exposure, with durations of exposure ranging from 6 months to 7 years. The airborne concentrations of terphenyl varied, measuring 0.094 mg/m³ in general working areas and up to 0.89 mg/m³ in areas with organic piping equipment. The terphenyl coolant was determined to be a primary irritant, even in those workers wearing protective clothing, because skin moistness increased dermal sensitivity to the terphenyls (Weeks 1971/Ex. 1–580; Weeks and Lentle 1970/Ex. 1–682). Testa and Masi (1964/Ex. 1–578) reported that, at concentrations above 10 mg/m³ (approximately 1 ppm, the current OSHA ceiling limit in construction and maritime), workers reported both eye and respiratory irritation.

OSHA is proposing a ceiling limit of 0.5 ppm for the terphenyls in construction, maritime, and agriculture. The Agency preliminarily concludes that this limit will protect workers in these sectors against the significant risk of primary irritation of the eyes, skin, and upper respiratory tract potentially associated with exposure to very low airborne levels of the terphenyls. The Agency believes that this limit will substantially reduce these significant risks and that primary irritation and metabolic effects constitute material health impairments. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

m-TOLUIDINE

CAS: 108–44–1; Chemical Formula: C_7H_9N

H.S. No. 1401

OSHA has no PEL for m-toluidine in the construction, maritime, and agriculture industries. The ACGIH has a 2-ppm 8-hour TLV®-TWA, with a skin notation. NIOSH has no REL for this substance. OSHA is proposing a PEL of 2 ppm as an 8-hour TWA, and a skin notation, for this substance in the construction, maritime, and agriculture industries. This is the limit recently promulgated for m-toluidine in general industry.

m-Toluidine is a light yellow liquid. It is used as an intermediate in the manufacture of dyes and other chemicals (ACGIH 1986, p. 589).

Like other aniline derivatives, m-toluidine produces methemoglobinemia in humans and experimental animals. The oral LD_{50}s in rats and mice are 450 and 740 mg/kg, respectively (RTECS 1990). When m-toluidine was tested on the eyes and skin of rabbits, moderate to strong irritation resulted (NIOSH 1979b, as cited in ACGIH 1986/Ex. 1–3, p. 589). A mean maximal methemoglobinemia of 60.2 percent was reported to occur following the intravenous administration of 27 mg/kg m-toluidine in cats (McLean, Starmer, and Thomas 1969/Ex. 1–425). Rodent carcinogenicity studies cited by the ACGIH (1986/Ex. 1–3, p. 589) were either inconclusive or negative; however, NIOSH (Ex. 8–47, Table N–2) pointed out in the prior rulemaking that this substance is an aromatic amine like o- and p-toluidine, both of which are carcinogenic.

The effects in humans of exposure to m-toluidine, when it is either absorbed through the skin or delivered via inhalation, are hematuria and methemoglobinemia. Exposure to 40 ppm for 60 minutes causes severe poisoning (Goldblatt 1955/Ex. 1–417). There are no epidemiological studies of workers exposed only to m-toluidine (ACGIH 1986/Ex. 1–3, p. 589).

OSHA is proposing a 2-ppm 8-hour TWA and a skin notation for m-toluidine in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that this limit will protect workers from the significant risk of metabolic effects, such as hematuria and methemoglobinemia, associated with exposure to m-toluidine. OSHA believes that hematuria, methemoglobinemia, and the other metabolic effects associated with exposure to m-toluidine constitute material impairments of health. Promulgation of this limit will also make the PEL for this substance consistent across all OSHA-regulated sectors.

2,4,6–TRINITROTOLUENE (TNT)

CAS: 118–96–7; Chemical Formula: $C_7H_5N_3O_6$

H.S. No. 1413

OSHA's PEL for 2,4,6-trinitrotoluene (TNT) in construction and maritime is 1.5 mg/m³ as an 8-hour TWA, with a skin notation. There is no limit in agriculture. The ACGIH has a TLV®-TWA of 0.5 mg/m³, also with a skin notation, for this chemical. NIOSH has no REL for this substance but concurs (Ex. 8–47, Table N–1) with the limit being proposed. OSHA is proposing an 8-hour TWA of 0.5 mg/m³, and a skin notation, for this substance in construction, maritime, and agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

TNT occurs as yellow, needle-like crystals. It is used as a high explosive (ACGIH 1986, p. 610).

TNT causes destruction of the red blood cells and methemoglobinemia in experimental animals. The oral LD_{50}s in rats and mice are 795 and 660 mg/kg, respectively. Rats given 50 mg/kg per day in the diet showed anemia, lesions of the spleen, liver and kidney damage, and bladder carcinoma (Proctor, Hughes, Fischmann 1988, p. 497). In a study by Dilley et al. (cited in HSDB 1990), rats were given 0, 0.002, 0.01, 0.05, or 0.25 percent TNT in the diet for 13 weeks. Rats receiving the highest dose exhibited anemia with reduced erythrocytes, hemoglobin, and hematocrit, as well as reduced testicular size; all effects except the testicular atrophy were reversible after the exposure period.

The ACGIH's limit was selected on the basis of health surveys conducted among occupationally exposed workers. Fairhall (1957e, as cited in ACGIH 1986/Ex. 1–3, p. 610) describes dermatitis, cyanosis, gastritis, acute yellow atrophy of the liver, and aplastic anemia as possible effects of exposure to TNT. According to Sollman (1957/Ex. 1–991), blood cell destruction, leucocytosis or leucopenia, and varying degrees of central nervous system change (probably resulting from anoxia), peripheral neuritis and muscular pain, cardiac muscular and menstrual irregularities, and urinary and renal irritation can also occur as a consequence of TNT exposure. TNT has irritant properties and may cause sneezing, sore throat, or skin irritation (von Oettingen 1941/Ex. 1–874).

A study by Goodwin (1972/Ex. 1–556) revealed 36 cases of liver damage in a munitions plant where workers were exposed to a mean air level of 2.38 mg/m³ TNT over a period of 20 years.

Another study (Morton, Ranadive, and Hathaway 1976/Ex. 1–566) found elevated levels of liver enzymes in 43 TNT shell-packers and loaders who worked where TNT exposures ranged from 0.3 to 0.8 mg/m³ over a period of 5 months. In 1975, Djerassi and Vitany (Ex. 1–550) published a paper describing hemolytic episodes in three TNT workers with glucose-6-phosphate dehydrogenase deficiency; although these workers were from Iraq, where G–6–PDase deficiency has a high (25 percent) frequency of occurrence, the study is also of concern for other workers having a high frequency of G–6–PDase deficiency. TNT has been reported to cause cataracts among workers exposed above 1 mg/m³ for 5 years or more (Proctor, Hughes and Fischmann 1988, p. 496). The cataracts induced by TNT may not regress after the cessation of exposure.

OSHA is proposing an 8-hour TWA of 0.5 mg/m³ for 2,4,6-trinitrotoluene, and a skin notation, in the construction, maritime, and agriculture industries. The Agency preliminarily concludes that this limit is necessary to protect workers in these sectors against the significant risk of liver damage, hemolytic effects, and cataract formation potentially associated with exposure to TNT. OSHA believes that this limit will substantially reduce these significant risks and that liver damage and hemolysis constitute material health impairments. Promulgation of this limit will also make the PEL for this substance consistent across all OSHA-regulated sectors.

WARFARIN

CAS: 81–81–2; Chemical Formula: $C_{19}H_{16}O_4$

H.S. No. 2166

In general industry, construction, and maritime, OSHA's permissible exposure limit for warfarin is 0.1 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®-TWA of 0.1 mg/m³ for this substance; NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL in agriculture of 0.1 mg/m³ for warfarin. This is the limit recently established for warfarin in general industry.

Warfarin is an odorless, colorless to white, crystalline powder. It is available commercially as a dust or liquid concentrate and in various formulations with other pesticides (HSDB 1987; Hayes 1982, p. 508). Warfarin is primarily used as a rodenticide; it is also used in medicine as an anticoagulant (HSDB 1987). When used in pesticidal applications and in accordance with the label, this substance is regulated by the

EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

In both animals and humans, warfarin acts on the liver to inhibit prothrombin formation, which interferes with blood clotting; warfarin also damages the blood vessels directly. The lowest reported oral LD_{50} in rats is 1500 μg/kg, and the LC_{50} in the same species is 320 mg/m³ (RTECS 1990). The dermal LD_{50} in rats is 1400 mg/kg (RTECS 1990). Although a large single dose of warfarin can cause poisoning, this substance is most toxic when ingested daily in small amounts over a 5- to 7-day period. Rats and mice die after ingesting 1 mg/kg/day for 6 days (Booth 1982, in HSDB 1987). The clinical signs of lethal warfarin poisoning include massive hemorrhage, visible hematomas under the skin and around the joints, and bloody discharges from body orifices. Shock, weakness, and labored breathing may also occur (Booth 1982, p. 955). Intramuscular exposure to 10 mg/kg on days 8 through 28 of pregnancy caused stillbirths and developmental abnormalities in the offspring of dosed rabbits (RTECS 1990).

In humans, the lowest reported lethal dose is 6667 μg/kg (RTECS 1989). Several days of warfarin administration are usually required to deplete the body's stores of clotting factor and to produce an anticoagulant effect. A single large dose can cause the anticoagulant effect after a latency period of several days (Wisconsin Alumni Research Foundation 1951; Back et al. 1978). Toxic doses of warfarin also produce dilation and engorgement of blood vessels and increased capillary fragility, which may further increase the risk of hemorrhage (Hayes and Gaines 1950; Hayes 1963; Heisey et al. 1956). Except for abnormal bleeding, warfarin has few toxic effects, although anorexia, nausea, vomiting, diarrhea, and skin lesions may occur in some individuals (Goodman 1975, p. 1359). As in animals, warfarin is most toxic when ingested in small doses over a period of 5 to 6 days; 1 to 2 mg/kg/day for 6 days is estimated to be lethal (Klaassen, Amdur, and Doull 1986, p. 564). Warfarin can be absorbed through the skin in toxic amounts. A 23-year old farmer was exposed to a 0.5-percent warfarin sodium salt by skin contact for several hours on ten occasions during a 24-day period. On day 26, he developed gross hematuria. On day 27, hematomas appeared on his arms and legs and he complained of dull pain in both sides of his groin area. On day 30, the hematuria resolved and he developed nosebleeds. The patient recovered fully following administration of vitamin-K (Fristedt and Sterner 1965, p. 205). Warfarin causes fetotoxic and teratogenic effects in the offspring of women taking this substance as an anticoagulant during pregnancy; many cases of stillbirths and deformities have been attributed to the therapeutic use of warfarin (Hall et al. 1980).

Based on this evidence, OSHA preliminarily concludes that, in the absence of a permissible exposure limit, workers in agriculture are at significant risk of experiencing capillary fragility and hemorrhage, the principal effects of warfarin exposure. The Agency believes that establishing an 8-hour TWA PEL of 0.1 mg/m³ will substantially reduce this risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

Preliminary Conclusions for the Group of Biochemical/Metabolic Toxins. For the class of toxic substances having biochemical/ metabolic effects, OSHA preliminarily concludes that occupational exposure presents significant risks to health. The effects associated with exposure to these substances (which inhibit cholinesterase activity, interfere with the blood's ability to carry oxygen, or produce Antabuse-like symptoms and signs) range from nausea, bronchoconstriction, cardiac irregularities, neurobehavioral effects, and unconsciousness to coma and death, depending on the severity of the exposure. OSHA believes that all of these symptoms and signs constitute material health impairments. Accordingly, the Agency believes that establishing the proposed limits for this group of toxicants is necessary to substantially reduce these significant occupational risks among workers in construction, maritime, and agriculture.

13. Substances for Which Proposed Limits Are Based on Avoidance of Sensitization Effects

Introduction. OSHA is proposing limits for 12 substances on the basis of their ability to cause pulmonary or skin sensitization. Table C13–1 lists OSHA's current permissible exposure limits (PELs) for these substances in construction and maritime; there are no PELs in agriculture for this group of sensitizers. Table C13–1 also shows the 1987–1988 ACGIH TLV® and the NIOSH recommended exposure limit (REL) for these substances. The last column on this table shows the limits for these substances recently established by OSHA in general industry; these are the limits being proposed for these substances in construction, maritime, and agricultural workplaces. The CAS and HS numbers for these substances are also shown in Table C13–1.

Description of the Health Effects. A sensitization reaction, also known as an allergic reaction, is defined as an adverse response to a chemical following a previous exposure to that substance or to a structurally similar one (Klaassen, Amdur, and Doull 1986/ Ex. 1–99). A person who suffers an allergic reaction to a chemical is said to have become sensitized to that substance.

TABLE C13–1. SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON AVOIDANCE OF SENSITIZATION

H.S. No./chemical name	CAS No.	Current OSHA PEL in construction and maritime*	1987–1988 ACGIH TLV®**	NIOSH REL***	Proposed OSHA PEL in construction, maritime, and agriculture*
1066 Captafol (Difolatan)	2425–06–1		0.1 mg/m³ TWA, Skin.		0.1 mg/m³ TWA.
1100 Cobalt metal, dust and fume (as Co)	7440–48–4	0.1 mg/m³ TWA	0.05 mg/m³ TWA		0.05 mg/m³ TWA.
1222 Isophorone diisocyanate	4098–71–9		0.01 ppm TWA, Skin	0.005 pmm TWA 0.02 pmm Ceiling (10-min).	0.005 pmm TWA. 0.02 pmm STEL, Skin.
1313 Phenothiazine	92–84–2		5 mg/m³ TWA, Skin		5 mg/m³ TWA, Skin.
2128 p-Phenylene diamine	106–50–3	0.1 mg/m³ TWA, Skin.	0.1 mg/m³ TWA, Skin.		0.1 mg/m³ TWA, Skin.
1315 Phenyl glycidyl ether (PGE)	122–60–1	10 pmm TWA	1 pmm TWA	1 pmm Ceiling (15-min).	1 pmm TWA.

TABLE C13-1. SUBSTANCES FOR WHICH PROPOSED LIMITS ARE BASED ON AVOIDANCE OF SENSITIZATION—Continued

H.S. No./chemical name	CAS No.	Current OSHA PEL in construction and maritime*	1987-1988 ACGIH TLV®**	NIOSH REL***	Proposed OSHA PEL in construction, maritime, and agriculture*
1329 Picric acid	88-89-1	0.1 mg/m³ TWA, Skin.	0.1 mg/m³ TWA 0.3 mg/m³ STEL, Skin.		0.1 mg/m³ TWA, Skin.
2133 Platinum soluble salts (as Pt)	7440-06-4	0.002 mg/m³ TWA	0.002 mg/m³ TWA		0.002 mg/m³ TWA.
1347 Rhodium, metal, fume and insoluble compounds (as Rh).	7440-16-6	0.1 mg/m³ TWA	1 mg/m³ TWA		0.1 mg/m³ TWA.
1348 Rhodium, soluble compounds (as Rh)	7440-16-6	0.001 mg/m³ TWA	0.01 mg/m³ TWA		0.001 mg/m³ TWA.
1373 Subtilisins (Proteolytic enzymes)	1395-21-7		0.06 µg/m³ STEL (60-min)†.		0.06 g/m³ STEL (60-min) †*.
1398 Toluene-2,4-diisocyanate (TDI)	584-84-9	0.02 ppm Ceiling	0.005 ppm TWA 0.02 ppm STEL	0.005 pmm TWA 0.02 pmm Ceiling (10-min).	0.005 ppm TWA. 0.02 ppm STEL.

*OSHA's PELs do not currently apply in Agriculture; OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

**The ACGIH TLV®-TWA is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times in any working day, with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

***NIOSH TWA limits are for 10 hour/day, 40 hour/week exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

† Compliance with the subtilisins PEL is assessed by sampling with a high volume sampler (600-800 liters per minute) for at least 60 minutes.

Sensitization is the result of an immune system reaction to a substance; although the initial exposure does not generate an immediate response, the immune system "remembers" the substance and reacts strongly at the next encounter. A related phenomenon is cross-sensitization. Cross-sensitization occurs when exposure to one substance elicits a sensitization reaction not only on subsequent exposure to the same substance but also on exposure to a different substance (usually one with a similar chemical structure).

The toxic manifestations of sensitization reactions vary in both location and severity. In humans, common target organs are the skin and the eyes; typical allergic conditions in these organ systems are allergic contact dermatitis and conjunctivitis, respectively. The respiratory system can also be sensitized; the resulting pathologies include bronchitis and asthma (Dean, Murray, and Ward 1986/Ex. 1-195). These allergic reactions are mediated by the two immunoglobulins IgD or IgE. The involvement of IgD results in delayed contact dermatitis. In contrast, IgE-mediated reactions cause very severe and potentially fatal effects, such as acute asthmatic attacks, urticaria, and anaphylactic shock. The unpredictability and potential seriousness of sensitization reactions demand that exposures to sensitizing substances be carefully controlled.

Sensitivity to a chemical frequently persists throughout the lifetime of an individual; in some cases, however, sensitization disappears over time. Sensitization symptoms are not observed after exposure to the sensitizing agent (or to a structurally similar chemical) has been discontinued. Although it is possible to treat some allergies, avoidance is considered the best way—and is sometimes the only way—to regain good health.

An additional cause for concern about exposure to sensitizing chemicals is recent evidence that residual respiratory symptoms may continue even after exposure is discontinued. For example, in the case of toluene-2,4-diisocyanate (TDI), studies by Weill, Butcher, Dharmarajan et al. (1981/Ex. 1-1188) and Innocenti, Franzinelli, Sartorelli et al. (1981/Ex. 1-180) found that sensitized workers may exhibit decreased pulmonary function or chronic bronchitis for as long as three and one-half years after cessation of exposure.

Dose-Response Relationships and Sensitization Effects. Like other toxic effects, allergic reactions are dose-related; that is, in response to larger doses of the substance, increasing numbers of subjects become sensitized and the subsequent reactions become more severe. The time course of sensitization for any one individual is unpredictable. Some individuals are sensitized after only one exposure; others remain resistant to sensitization after a lifetime of exposure. Different people are generally sensitive to different substances, although some substances are more universally reactive than others, such as the active agent in poison ivy. Various parameters influence the likelihood of sensitization by a particular chemical; these include such factors as "the nature of the chemical, concentration, type of exposure, genetic susceptibility and nongenetic idiosyncrasies" (Emmett 1986/Ex. 1-226). The sensitization reactions observed in occupational settings are often the result of dermal or inhalation exposure.

For most of the substances in this group, OSHA is proposing limits in construction, maritime, and agriculture on the basis of health surveys and reports of occupationally exposed populations; the proposed limits are those recently established by OSHA for workplaces in general industry. The studies available indicate that exposures to airborne concentrations of these substances that are below a certain no-effect level generally do not cause sensitization in individuals. Where human data were absent or sparse, OSHA relied on animal evidence to determine where the proposed limit should be set. Sensitization reactions in animals are generally good predictors of immune system reactions in humans because chemically induced immunological sensitization in laboratory animals involves the same mechanism as in humans (that is, immune reactions in animals can be mediated by either IgD or IgE immunoglobulins).

The discussions below describe the evidence and explain OSHA's rationale for proposing these limits for these substances. These discussions illustrate the nature of the risk confronting exposed employees in construction, maritime, and agriculture and indicate the extent to which the risk of developing immune sensitization will be reduced among workers in these sectors by the promulgation of the proposed limits.

CAPTAFOL (DIFOLATAN)

CAS: 2425-06-1; Chemical Formula: $C_{10}H_9Cl_4NO_2S$

H.S. No. 1066

OSHA has no permissible exposure limit for captafol in the construction, agriculture, and maritime industries. The ACGIH has a TLV®–TWA of 0.1 mg/m³, with a skin notation, for captafol; there is no NIOSH REL. The Agency is proposing a TWA PEL of 0.1 mg/m³ for captafol in the construction, agriculture, and maritime industries. In the prior rulemaking, NIOSH (Ex. 8–47, Table N6A) concurred that this limit was appropriate. The 0.1 mg/m³ 8-hour TWA PEL was recently established in general industry.

Captafol is a colorless to white, crystalline solid with a faint and characteristically pungent odor. This substance was formerly used as a fungicide in agriculture; however, EPA canceled the registration for this substance in 1987 on the basis of captafol's carcinogenicity in laboratory animals (HSDB 1990; ACGIH 1986, p. 97). Although OSHA is not aware of any current use of captafol in those sectors being covered by this rule, OSHA includes this substance for the sake of completeness and to ensure that any occupational exposure to captafol of which OSHA may not be aware is covered by this rule.

Captafol is a skin sensitizer in guinea pigs and a teratogen in several species of animals (Clayton and Clayton 1981, p. 2705). The oral LD_{50} in rats is 2500 mg/kg (RTECS 1990). Rats fed captafol at dietary levels of 1500 or 5000 ppm had enlarged livers and, at the higher dose, an increase in mortality (Hayes 1982, p. 583). At autopsy, both liver and kidney damage were seen (Hayes 1982, p. 583). Dogs fed 100 or 300 mg/kg/day vomited and had diarrhea for the first 4 weeks and subsequently became slightly anemic and developed enlarged kidneys and livers. At 10 mg/kg/day, no effects were seen (Hayes 1982, p. 583). Tested in rats, rabbits, and hamsters by the oral or intraperitoneal routes of administration, captafol caused embryotoxic, developmental, or reproductive effects (RTECS 1990). Captafol is mutagenic in bacterial and mammalian test systems (RTECS 1990).

In humans, captafol is a known skin and respiratory sensitizer (Clayton and Clayton 1981, p. 2705). Both agricultural and non-agricultural workers have developed captafol-induced dermatitis; the rash produced in these cases resembles that of poison oak (Clayton and Clayton 1981, p. 2710). In one case, a welder brushed up against bags of captafol at a pesticide distribution plant for a period of 1.5 years with no reaction but then developed skin and respiratory sensitization, complete with facial edema, swelling of the hands, wheezing, and marked vesiculation (Clayton and Clayton 1981, p. 2710). Several cases of photosensitization dermatitis have been reported among farm workers (ACGIH 1986, p. 97). A recent study in florist shop workers (Thiboutot, Hamory, and Marks 1990, in J. Am. Acad. Dermatol. 22(1):54–58) showed that at least one case of allergic dermatitis in these workers was due to captafol; this finding was confirmed by patch test.

The Agency is proposing a permissible exposure limit for captafol of 0.1 mg/m³ as an 8-hour TWA. This limit will protect workers construction, maritime, and agriculture from the significant risks of contact dermatitis, respiratory and skin irritation, and sensitization that are associated with exposure to captafol at the levels currently permitted by the absence of an OSHA limit. The Agency preliminary concludes that the proposed 8-hour TWA PEL will substantially reduce these significant risks. In addition, promulgation of these limits will make OSHA's PEL for this substance consistent across all regulated sectors.

COBALT METAL, DUST, AND FUME
(measured as Co)
CAS: 7440–48–4; Chemical Formula: Co
H.S. No. 1100

OSHA currently has an 8-hour TWA limit of 0.1 mg/m³ for cobalt metal, dust, and fume (measured as cobalt) in the construction and maritime. There is no PEL in agriculture for these substances. The ACGIH has a TLV®–TWA of 0.05 mg/m³ for cobalt and its dust and fume; there is no NIOSH REL. The Agency is proposing a TWA PEL of 0.05 mg/m³ for cobalt metal, dust, and fume in the construction, maritime, and agriculture industries. The Agency recently established this limit for these substances in general industry, and NIOSH (Ex. 8–47, Table N1) concurred that this limit was appropriate for these sensitizers.

Cobalt is a hard, gray, magnetic, and somewhat malleable metal. It is used in steel alloys, jet engines, and in cemented carbide abrasives and tools (ACGIH 1986, p. 144).

In addition to sensitization, exposure to cobalt fume and dust causes lung disease in animals and humans. The oral LD_{50} in rats is 6170 mg/kg, and the lowest lethal dose in rabbits by oral administration is 750 mg/kg (RTECS 1990). Instilled into the eyes of rabbits, finely divided cobalt metal caused severe irritation and abscesses of the lens, ciliary body, and retina (Grant 1986, p. 247). Animals acutely poisoned by inhalation develop pulmonary effusions, hemorrhage, and edema (HSDB 1990). Chronic inhalation of a cobalt-metal blend at a concentration of 20 mg cobalt/m³ for 3 years caused focal fibrosis, bronchial hyperplasia, and granulomas (Clayton and Clayton 1982, p. 1612). Exposure to a 1-mg/m³ concentration of this blend for 2 years had no adverse effects (Clayton and Clayton 1982, p. 1612).

Administration of intratracheal doses (10, 25, or 50 mg) of cobalt metal dust causes obliterative bronchiolitis adenomatosis in guinea pigs (Schepers 1955/Ex. 1–365). Additional studies in animals have shown that exposure to cobalt dust or fume causes hypersensitivity reactions. Increases in serum A–2 globulin and neuraminic acid occurred in dogs and rabbits exposed by inhalation to cobalt metal, metal fume, or carbide blend; injections of cobalt chloride produced similar reactions (Stokinger and Wagner 1958/Ex. 1–381). Studies conducted in miniswine have shown that inhalation of 0.1 mg/m³ cobalt metal dust (50 percent alpha and 50 percent beta variety, with a size range of from 0.4 um to 3.6 um) has caused early (onset within 3 months) pulmonary disease. Wheezing, which indicates hypersensitivity, occurred in these animals during the fourth week of exposure to 0.1 or 1.0 mg/m³ for 6 hours/day, 5 days/week, for 3 months following a 1-week sensitizing dose (Kerfoot, Fredrick, and Domeier 1975/Ex. 1–145).

Workers exposed to cobalt dust developed allergic sensitization dermatitis, with the most serious eruptions occurring in skin areas in contact with the dust (Clayton and Clayton 1982, p. 1616). Pulmonary disease has been reported frequently in workers exposed to cobalt in the manufacture of cemented tungsten carbide (Miller, Davis, Goldman, and Wyatts 1953/Ex. 1–40; Lundgren and Ohman 1954; Lundgren and Swenson 1953/Ex. 1–816). Chronic exposure generally causes interstitial pneumonitis, but fatalities have occasionally been reported after exposures to cobalt at concentrations of 1 to 2 mg/m³ or less (Fairhall, Castberg, Carrozzo, and Brinton 1947/Ex. 1–954; Fairhall, Keenan, and Brinton 1949/Ex. 1–479). Cobalt-cemented tungsten carbide workers exposed chronically to the dust of this substance develop pneumoconiosis, with pulmonary insufficiency (Clayton and Clayton 1982, p. 1616). An increase in serum A–2 globulin fraction was reported in the case of a welder exposed to fumes containing cobalt; the welder had a history of exertional dyspnea and an abnormal chest x-ray (Siegesmund, Funahashi, and Pintar 1974/Ex. 1–372).

Two epidemiological studies in cobalt-exposed workers have recently been published. A cohort mortality study by Mur et al. (1987) found a statistically significant increase in lung cancer mortality in cobalt and sodium workers in an electrochemical plant, and a study of hard metal workers in Great Britain (Kusaka et al. 1986) reports that occupationally-induced asthma occurred in some cobalt-exposed workers who had average exposure levels below 0.05 mg/m³. In the prior rulemaking, several commenters (Ex. 150; Tr. 7–44/7–46; Tr. 11–108) stated that pulmonary disease and ischemic heart disease may be associated with exposures to cobalt at levels of 0.06 mg/m³ and, perhaps, to levels somewhat below this concentration. In that rulemaking, OSHA expressed concern over these recent findings and stated the Agency's intention to monitor the literature on cobalt carefully to determine whether it should be included in a future PEL update project. OSHA is continuing to monitor the developing literature on cobalt; however, the primary objective of the present rulemaking is to achieve consistency in the Agency's PELs across all sectors.

Accordingly, the Agency is proposing an 8-hour TWA limit for cobalt metal, dust, and fume (measured as cobalt) of 0.05 mg/m³ in the construction, agriculture, and maritime industries. The Agency preliminarily concludes that this limit will reduce the significant risk of respiratory disease, pulmonary sensitization, and allergic dermatitis that have been demonstrated to occur at higher levels of exposure. OSHA preliminarily finds that the proposed limit is necessary to substantially reduce the risk of these material health impairments. In addition, promulgation of these limits will make OSHA's PEL for these substances consistent across all regulated sectors.

ISOPHORONE DIISOCYANATE

CAS: 4098–71–9; Chemical Formula: $C_{12}H_{18}N_2O_2$

H.S. No. 1222

OSHA has no limit for isophorone diisocyanate (IPDI) in the construction, agriculture, and maritime industries. The 1987–1988 ACGIH TLV®–TWA for IPDI was 0.01 ppm, with a skin notation. The NIOSH REL for all diisocyanates is 0.005 ppm as a 10-hour TWA and 0.02 ppm as a 10-minute ceiling limit. The Agency is proposing PELs of 0.005 ppm as a TWA and 0.02 ppm as a STEL, and a skin notation, for IPDI. These limits were recently established by the Agency for IPDI in general industry, and NIOSH concurs (Ex. 8–47, Table N1) that these limits are appropriate.

To date, there is little direct information on the acute toxicity or health effects associated with exposure to this particular isocyanate. However, diisocyanates, in general, cause irritation of the respiratory tract, decreases in pulmonary function, and sensitization. The LC_{50} in rats is 123 mg/m³ for 4 hours, and the dermal LD_{50} in the same species is 1060 mg/kg (RTECS 1990). There are two reports of workers exposed to isophorone diisocyanate or other diisocyanates who developed asthma or dyspnea, although the airborne concentration of IPDI associated with these effects was not specified (Clarke and Aldons 1981/Ex. 1–475; Tyrer 1979/Ex. 1–396). In one case, a 50-year-old spray painter developed a severe case of asthma shortly after beginning to use an IPDI-containing paint (Clark and Aldons 1981). This worker subsequently responded positively to a bronchial challenge (Clarke and Aldons 1981). In the other incident, painters using two-pack paints, i.e., those involving a reaction between diisocyanates and polyhydroxy compounds, are reported to develop asthma from inhaling the resulting vapors (Tyrer 1979).

OSHA is proposing a 0.005-ppm 8-hour TWA limit, a 0.02-ppm 15-minute short-term exposure limit, and a skin notation for IPDI in the construction, agriculture, and maritime industries. The proposed STEL of 0.02 ppm is designed to prevent the severe irritation effects associated with exposure to the diisocyanates even in nonsensitized workers, and the proposed skin notation will prevent dermal absorption of this substance. These are the limits recently established for workplaces in general industry. OSHA preliminarily concludes that these proposed limits will reduce the significant risk of immune-system-mediated pulmonary sensitization, which is associated with isocyanate exposure, among workers in construction, maritime, and agriculture.

PHENOTHIAZINE

CAS: 92–84–2; Chemical Formula: $S(C_6H_4)_2NH$

H.S. No. 1313

OSHA currently has no occupational exposure limit for phenothiazine in the construction, agriculture, and maritime industries. The ACGIH has a TLV®–TWA of 5 mg/m³, with a skin notation; there is no NIOSH REL for this substance. The Agency is proposing a TWA PEL of 5 mg/m³, and a skin notation, for phenothiazine in the construction, agriculture, and maritime industries. OSHA recently established this limit for general industry, and NIOSH (Ex. 8–47, Table N1) concurred with this limit in the prior rulemaking.

Phenothiazine ranges in color from grayish-green to greenish-yellow and can take the form of powder, granules, or flakes. This substance is tasteless but has a slight odor. Phenothiazine is used as an insecticide, antioxidant, polymerization inhibitor, antihelminthic agent, and as a base in the manufacture of pharmaceuticals (HSDB 1984; ACGIH 1986, p. 472). When used in pesticidal applications and in accordance with the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Exposure to phenothiazine causes moderate irritation of the skin (Cralley and Cralley 1985, p. 178). The LD_{50} in mice is 5000 mg/kg; acutely poisoned animals showed behavioral signs (RTECS 1990). Rats given oral doses of 1250 mg/kg during days 1 through 22 of pregnancy showed adverse effects on reproduction (RTECS 1990). After pigs and cattle have been given anti-helminthic doses of phenothiazine, they develop a photosensitization dermatitis with corneal edema (Grant 1986, p. 812).

A study by Mawhinney and Rakow (1988) showed that humans exposed to 15 to 48 mg/m³ of phenothiazine developed skin sensitization; symptoms of sensitization in workers included burning and itching of the skin. Accompanying these sensitization reactions were pinkish-red-colored hair and brown fingernails. Phenothiazine has also been reported to cause photosensitization of the skin, and intense irritation and itching of the skin have been associated with inhalation of phenothiazine spray (ACGIH 1986/Ex. 1–3, p. 472). Workers handling phenothiazine are reported to have developed keratitis, although whether photosensitization or physical irritation was responsible for this effect is now known (Grant 1986, p. 812). Accidental exposures to high but unspecified concentrations of phenothiazine have caused hemolytic anemia, toxic hepatitis, skin photosensitization, and pruritus (Gosselin, Smith, and Hodge 1984, p. II–238).

OSHA is proposing an 8-hour TWA PEL of 5 mg/m³, with a skin notation, for phenothiazine; this limit is below the exposure range that has been shown to cause sensitization reactions in workers. OSHA preliminarily concludes that the uncontrolled exposures that are possible in the construction, agriculture, and maritime industries in the absence of an OSHA limit potentially pose a significant risk of sensitization to workers in these sectors. The Agency believes that the proposed limit is

necessary to reduce this risk among workers in these sectors. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

p-PHENYLENE DIAMINE

CAS: 106–50–3; Chemical Formula: $C_6H_4(NH_2)_2$

H.S. No. 2128

In general industry, construction, and maritime, OSHA's current permissible exposure limit for p-phenylene diamine is 0.1 mg/m³ as an 8-hour TWA; this limit also has a skin notation, which indicates that percutaneous absorption is a significant route of exposure for this substance. There is no limit in agriculture. The ACGIH has an 8-hour TLV®–TWA of 0.1 mg/m³ for p-phenylene diamine, with a skin notation. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA PEL of 0.1 mg/m³, and a skin notation, for p-phenylene diamine in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

p-Phenylene diamine occurs in the form of white to light purple crystals that oxidize to purple or black on exposure to air. This substance is used as a hair dye, a photographic developer, an intermediate in the manufacture of dyes, an ingredient of antioxidants and rubber accelerators, and in photochemical measurements (ACGIH 1986, p. 474.8(89); Hawley's 1987, p. 902).

p-Phenylene diamine causes sensitization of the skin and respiratory tract in exposed workers. The oral LD_{50} in rats is 80mg/kg, and the lowest lethal dose in rabbits by dermal absorption is 5000mg/kg (RTECS 1990). A dose of 5000mg/kg p-phenylene diamine applied to the shaved skin of rabbits was fatal (Burnett, Loehr, and Corbett 1977, in ACGIH 1986, p. 474.8(89)). A dose of 250mg placed on the skin caused moderate irritation in rabbits and mild irritation in guinea pigs (RTECS 1990). In various species (dogs, rabbits, cats, and frogs), p-phenylene diamine was itself only slightly irritating in acute studies; however, the products of its oxidation were found to be strong skin sensitizers (Hanzlik 1923, in ACGIH 1986, p. 474). Groups of male and female rabbits were given topical treatments of 1g/kg of 1.2 percent p-phenylene diamine five times/week for 65 applications, or 10 g per application once every 2 weeks, for a total of seven exposures. The effects observed among the animals were fissures, scab formation, desquamation, acanthosis, and moderate dermal fibroplasia (Cosmetic, Toiletry, and Fragrance Assoc. 1969, in ACGIH 1986, p. 474.9(89)). In chronic studies in which mice, rabbits, and rats were given p-phenylene diamine in various doses by various routes, there were no clinical signs or symptoms and no significant exposure-related changes as compared with controls (ACGIH 1986, p. 474.10(89)). p-Phenylene diamine has been tested for carcinogenicity in mice and rats; however, the International Agency for Research on Cancer has concluded that the evidence in animals is inadequate to evaluate p-phenylene diamine's carcinogenicity (IARC 1978, Vol. 16, p. 136). A recent study (Burnett and Goldenthal 1988, in Food Chem. Toxicol. 26(5):467–474) involving weanling rats given twice weekly skin applications of oxidative hair-coloring formulations that included p-phenylene diamine, however, has shown significant increases in mammary tumors and pituitary adenomas in treated compared with control animals.

In humans, p-phenylene diamine-induced sensitization dermatitis has been reported in workers in the fur-dyeing, photography, and cosmetics industries. Workers exposed to p-phenylene diamine also report experiencing frequent bouts of inflammation of the pharynx and larynx and allergic asthma (Ritter 1921, in ACGIH 1986, p. 474). In reports describing the effects of accidental ingestion of hair dyes containing p-phenylene diamine, symptoms reported included acute renal failure, methemoglobinemia, hemolysis, dyspnea, swelling of the lips, tongue, and neck, and muscular rhabdomyolysis (ACGIH 1986, p. 474.12(89)). Jaundice and subacute atrophy of the liver leading to death were observed in a female hairdresser who had been in contact with hair dyes for over 5 years (Israels and Susman 1934, in ACGIH 1986, p. 474.12(89)). Occupational dermatoses of allergic origin have been confirmed by patch testing in workers exposed to color-developing agents in photographic laboratories; p-phenylene diamine patch tests were positive in several of these workers (Liden, Brehmes, and Andersson 1988, in Acta Dermato-Venerologica 68(6):514–522). Twelve of 13 beauticians who developed occupational allergic contact dermatitis tested positive to patch tests with p-phenylene diamine (Matsunaga, Hosokawa, Suzuki, Arima, and Hayakawa 1988, in Contact Dermatitis 18(2):94–96). Another study has confirmed the cataractogenic effects of hair dyes containing this substance (Jain et al. 1979, in ACGIH 1986, p. 474.12(89)).

Based on this evidence in humans and animals, OSHA preliminarily concludes that p-phenylene diamine causes skin and pulmonary sensitization in exposed individuals. OSHA preliminarily finds that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that establishing the proposed 8-hour TWA limit of 0.1 mg/m³, and a skin notation, for p-phenylene diamine in agriculture is necessary to substantially reduce these risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PHENYL GLYCIDYL ETHER

CAS: 122–60–1; Chemical Formula: $C_6H_5OCH_2CHOCH_2$

H.S. No. 1315

OSHA's current 8-hour TWA limit for phenyl glycidyl ether (PGE) in the construction and maritime industries is 10 ppm. There is no PEL for PGE in agriculture. The ACGIH has a TLV®–TWA of 1 ppm for this substance; the NIOSH REL for PGE is 1 ppm as a 15-minute ceiling limit. The Agency is proposing an 8-hour TWA PEL of 1 ppm for PGE in the construction, agriculture, and maritime industries. This is the limit recently established for PGE in general industry.

Phenyl glycidyl ether is a colorless liquid that is used as a chemical intermediate, a diluent in uncured epoxy resins, and a stabilizer of halogenated compounds (ACGIH 1986, p. 478; HSDB 1986).

Exposure to PGE causes irritation of the skin and respiratory tract and sensitization of the skin in humans and animals. The oral LD_{50} in rats is 2150 mg/kg, and the dermal LD_{50} in rabbits is 1500 mg/kg (RTECS 1990). Acutely poisoned animals showed signs of central nervous system depression and died of respiratory paralysis (Smyth, Carpenter, Weil, and Pozzarin 1954/Ex. 1–440). Applied to the skin of rabbits, PGE caused moderate-to-severe irritation, and irritant effects on the eye of rabbits ranged from mild to severe (RTECS 1990). Studies by Hine, Kodama, Wellington, and colleagues (1956/Ex. 1–331) showed pulmonary inflammation and liver changes in some of the rats exposed to a 100-ppm concentration for 7 hours daily for 50 days; respiratory distress and signs of minimal eye irritation were also observed in the exposed animals. Terrill and Lee (1977/Ex. 1–390) reported kidney, liver, spleen, thymus, and testicular changes in rats exposed to phenyl glycidyl ether at 29 ppm for 4 hours/day, 5 days/week for 2 weeks. At concentrations of 12 or 5 ppm, these authors observed no effects other

than hair loss after exposures of 6 hours/day, 5 days/week for 9 weeks; however, after 18 weeks, 10 percent of male and 25 percent of female rats exhibited alopecia (hair loss). These health effects were believed by the authors to reflect direct irritation of the skin rather than systemic absorption (Terrill and Lee 1977/Ex. 1–390).

Reports of workers using or handling phenyl glycidyl ether have described moderate skin irritation on prolonged or repeated contact. Dermatitis is common in industrially exposed workers. In a group of 20 PGE-exposed workers, 13 had serious signs of contact dermatitis: Rash with papules, second-degree burns, and edema (Hine, Kodama, Wellington et al. 1956/Ex. 1–331). In a second group of PGE-exposed workers with similar signs, eight of 15 tested positive on patch testing (NIOSH Criteria Document 1978). NIOSH (1978d/Ex. 1–232) notes that the glycidyl ethers are biologically reactive compounds because of the presence of the epoxide group; these compounds have also been shown to cause cytotoxic effects and to be mutagenic in short-term bioassays (RTECS 1990).

The Agency is proposing an 8-hour TWA PEL of 1 ppm for phenyl glycidyl ether in the construction, agriculture, and maritime industries. OSHA preliminarily concludes that this limit will protect workers in these sectors from the significant risk of skin sensitization and skin and respiratory tract irritation that is potentially associated with exposure to PGE. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PICRIC ACID

CAS: 88–89–1; Chemical Formula: $HOC_6H_2(NO_2)_3$

H.S. No. 1329

OSHA's current limit for picric acid in the construction and maritime industries is 0.1 mg/m³ as an 8-hour TWA, with a skin notation. There is no PEL for picric acid in agriculture. The 1987–1988 ACGIH TLV®s for this substance were 0.1 mg/m³ as a TWA and 0.3 mg/m³ as a STEL, with a skin notation. NIOSH has no REL for picric acid but concurs (Ex. 8–47, Table N1) with the limit being proposed. The Agency is retaining its 8-hour TWA PEL of 0.1 mg/m³, with a skin notation, in construction and maritime, and is proposing the same PEL for agricultural workplaces. This action will make OSHA's PEL for picric acid consistent across all industry sectors.

Picric acid occurs in the form of colorless to pale yellow, odorless, intensely bitter crystals. It is used in medicine, explosives, rocket fuels, and matches. Picric acid also is used in the leather and glass industries and to etch copper (ACGIH 1986, p. 490).

Picric acid and its salts are toxic by ingestion, skin contact, or inhalation, and these substances also are skin sensitizers (Schwartz 1944/Ex. 1–367). The acute toxicity data for picric acid in animals are sparse. In cats, the lowest lethal oral dose is 250 mg/kg, and in rabbits this value is 120 mg/kg (RTECS 1990). Injected into the corneal stroma of the eyes of rabbits, picric acid caused injury even when the solution had been neutralized (Grant 1986, p. 737). Animals given sublethal doses of picric acid (less than or equal to 50 mg/kg) showed changes to the kidney at autopsy (NRC 1982, Vol. 4, p. 240).

Available reports concerning human exposures describe edema, papules, vesicles, and desquamations of the face, mouth, and nose caused by contact with the dust of this substance (Sunderman, Weidman, and Batson 1945/Ex. 1–383). The symptoms of systemic poisoning following skin absorption include headache, vertigo, vomiting, nausea, diarrhea, and skin and conjunctival discoloration, as well as discoloration of urine and albuminuria; high-dose exposures caused destruction of erythrocytes and produced gastroenteritis, hemorrhagic nephritis, and acute hepatitis (Sunderman, Weidman, and Batson 1945/Ex. 1–383). One worker lapsed into a coma after inhaling a high (not further specified) concentration of picric acid dust (von Oettingen 1941). Picric acid dust causes eye irritation, and sensitization dermatitis may follow dust exposure (Grant 1986, p. 737).

OSHA is retaining its 8-hour TWA PEL of 0.1 mg/m³ and the skin notation for picric acid in the construction and maritime industries and is proposing the same limit and skin notation in agriculture. The Agency preliminarily concludes that this limit is necessary to protect agricultural workers against the dermatitis and sensitization associated with occupational exposures to picric acid. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PLATINUM (soluble salts)

CAS: 7440–06–4; Chemical Formula: Pt

H.S. No. 2133

In general industry, construction, and maritime, OSHA's current permissible exposure limit for the soluble platinum salts (measured as platinum) is 0.002 mg/m³ as an 8-hour TWA. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.002 mg/m³ for the soluble platinum salts. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing an 8-hour TWA limit in agriculture of 0.002 mg/m³ (measured as platinum) for the soluble platinum salts. Promulgation of this limit will make the PEL for these substances consistent across all OSHA-regulated sectors.

Soluble platinum salts include substances such as sodium chloroplatinate, a yellow-orange, odorless solid; ammonium chloroplatinate, a yellow-orange solid; and platinum tetrachloride, which takes the form of brown to red crystals. Platinum's soluble salts are used as catalysts in the production of high-octane gasoline, acids, petrochemicals, pharmaceuticals, and vinyl esters, in the reclamation of platinum ore, in electroplating, and in photographic paper sensitization (NIOSH/OSHA Occupational Health Guideline 1981, p. 3; Merck 1983, pp. 80, 1235).

The soluble salts of platinum are powerful skin and respiratory allergens in humans (Klaassen, Amdur, and Doull 1986, p. 622). Exposure to the soluble platinum salts causes eye irritation and skin and pulmonary sensitization in humans. Ammonium chloroplatinate has an oral LD_{50} in rats of 195 mg/kg, and platinum tetrachloride has an oral LD_{50} in the same species of 276 mg/kg (RTECS 1989). In contact with the skin of rabbits, 100 mg of platinum tetrachloride caused severe irritation (RTECS 1989). Platinum tetrachloride caused slight, reversible irritation when the eyes of rabbits were flooded with 2 to 5 percent solutions of this substance (Grant 1986, p. 748). Acute exposure to ammonium chloroplatinate causes central nervous system effects, including convulsions and coma, before death (Clayton and Clayton 1981, p. 1861). Small non-lethal doses of this salt cause hyperirritability, restlessness, and motor excitement in experimental animals (Clayton and Clayton 1981, p. 1861). Intratesticular or subcutaneous administration of a single 28,951-μg/m³ dose of platinum tetrachloride to rats and mice caused paternal reproductive effects (RTECS 1989).

In humans, the lowest concentrations of ammonium chloroplatinate or sodium chloroplatinate reported to be toxic in humans is 900 ng/m³; at this level, coughing and cyanosis were observed (RTECS 1989). In contact with the eyes, the dusts of soluble platinum salts cause burning, lacrimation, and conjunctival hyperemia associated with photophobia (Grant 1986, p. 748). Severe contact dermatitis due to an allergic reaction may occur after repeated exposure to some platinum salts, such as ammonium

or sodium chloroplatinate or platinic chloride (Grant 1986, p. 748). Of 306 platinum refinery workers, 38 tested positive when challenged with the platinum halide salts (Murdoch et al. 1986, in Proctor, Hughes, and Fischman 1988, p. 417). Exposure to these salts is associated with a syndrome variously referred to as platinum allergy, platinum asthma, or platinosis; the signs and symptoms of platinosis include runny nose, sneezing, tightness of the chest, shortness of breath, cyanosis, wheezing, and coughing (Hunter et al. 1945; Parrot, Herbert, Saindelle, and Ruff 1969, in Proctor, Hughes, and Fischman 1988, p. 417). If exposure to the salts of platinum continues, the symptoms become progressively worse (Parmeggiani 1983, p. 1724). The earliest reported cases of platinosis occurred in 1911, when eight workers handling platinum salts experienced symptoms of throat and respiratory irritation (Karasek and Karasek 1911, in ACGIH 1986, p. 492).

Based on this evidence in humans and animals, OSHA preliminarily concludes that exposure to soluble platinum compounds causes eye irritation, skin sensitization, and asthma in exposed workers. OSHA believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse exposure-related effects. The Agency believes that the proposed 8-hour TWA limit of 0.002 mg/m^3 for the soluble salts of platinum is necessary to substantially reduce these risks of material health impairment. Promulgation of this limit will also make the PEL for these substances consistent across all OSHA-regulated sectors.

RHODIUM METAL, FUME, AND INSOLUBLE COMPOUNDS

CAS: 7440–16–6; Chemical Formula: Rh

H.S. No. 1347

The current OSHA PEL for rhodium metal, fume, and insoluble compounds (measured as Rh) in general industry, construction, and maritime is 0.1 mg/m^3 as an 8-hour TWA. There is no limit in agriculture. The ACGIH recommends a TLV®–TWA of 1 mg/m^3 for rhodium metal and insoluble compounds. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limits being proposed. OSHA is proposing an 8-hour TWA limit of 0.1 mg/m^3 for rhodium metal, fume, and insoluble compounds (measured as Rh) in agriculture. Promulgation of this limit will make the PEL for these substances consistent across all OSHA-regulated sectors.

Rhodium metal is silvery white, hard, ductile, malleable, and odorless. It is available in metal, powder, and spongy forms. Rhodium fume is gray. Insoluble rhodium compounds include rhodium trichloride, rhodium trioxide, and rhodium (II) acetate. Rhodium is used as an alloy with platinum, in furnace windings, laboratory crucibles, spinnerets in the rayon industry, electrical contacts, jewelry, a catalyst, and coatings (Hawley's 1987, p. 1009; ACGIH 1986, p. 512).

Rhodium is a dermal sensitizer. There are no toxicity data for rhodium metal or fume. Instilled into the eye of rabbits, rhodium trichloride caused delayed injury, with opacification and vascularization (Grant 1986, p. 887). In rhodium trichloride, the intravenous LD_{50} in rats and rabbits are 198 and 215 mg/kg, respectively (Landolt, Beck, and Russell 1972). Surviving rats showed no histological lesions at autopsy 100 days later (Landolt, Beck, and Russell 1972). Intravenous injection of a 60 mg/kg dose of rhodium trichloride in rats and rabbits caused no signs of systemic toxicity (Plant 1936). In a carcinogenicity bioassay in mice, rhodium trichloride was administered to mice in drinking water (5 ppm) from weaning until death (Schroeder and Mitchener 1971). The rhodium-treated mice developed a significantly increased number of malignant tumors at multiple sites (Schroeder and Mitchener 1971).

There are no reports of rhodium-related systemic toxicity in humans. Direct contact of the eyes with the insoluble compounds is reported to have caused a mild degree of irritation (Proctor, Hughes, and Fischman 1988, p. 433). Dermal sensitization reactions to rhodium metal have been reported in jewelry workers (Bedello, Goitre, Roncarolo, Bundino, and Cane 1987, in Contact Dermatitis 17(2):111–112) and in precious metal refinery workers (Jirasek et al. 1975, in Ceskoslovenska Dermatologie 50(6):361–368, as reported in NIOSHTIC).

Based on this evidence in humans and animals, OSHA preliminarily concludes that rhodium metal, fume, and insoluble compounds cause sensitization. OSHA therefore preliminarily concludes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing this adverse health effect. The Agency believes that an 8-hour TWA limit of 0.1 mg/m^3 in agriculture is necessary to significantly reduce this risk of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for these substances consistent across all regulated sectors.

RHODIUM, SOLUBLE COMPOUNDS (as Rh)

CAS: 7440–16–6; Chemical Formula: Rh

H.S. No. 1348

OSHA's current permissible exposure limit for the soluble rhodium compounds (measured as Rh) in general industry, construction, and maritime is an 8-hour TWA of 0.001 mg/m^3. There is no limit in agriculture. The ACGIH has a TLV®–TWA of 0.01 mg/m^3 for soluble rhodium compounds. NIOSH has no REL but concurs (Ex. 8–47, Table N1) with the limits being proposed. OSHA is proposing an 8-hour TWA limit of 0.001 mg/m^3 for the soluble rhodium compounds in agriculture. Promulgation of this limit will make the PEL for these substances consistent across all OSHA-regulated sectors.

Soluble rhodium compounds include rhodium nitrate, rhodium sulfate, rhodium carbonyl acetylacetonate, and rhodium sulfite. Soluble rhodium compounds find uses in electroplating, camera fittings, radio equipment, and jewelry, and in laboratory research (NIOSH/OSHA Occupational Health Guideline 1981, pp. 1, 3).

Rhodium is a dermal sensitizer. One of the soluble rhodium compounds, rhodium carbonyl acetylacetonate, is an eye irritant and, in contact with the skin, a sensitizer that produces delayed Type IV sensitization (Johnson, Matthey, and Co., Ltd., 1981, as cited in ACGIH 1986, p. 512). Several rhodium compounds have been found to be potent mutagens in bacterial test systems (Kanematsu, Horn, and Kada 1980, in Mutat. Res. 77(2):109–116).

Based on this evidence in humans and animals, OSHA preliminarily concludes that soluble rhodium compounds cause sensitization. OSHA therefore believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing this adverse health effect. The Agency believes that a limit of 0.001 mg/m^3 for the soluble rhodium compounds (measured as Rh) in agriculture is necessary to significantly reduce the risk of this material health impairment. In addition, promulgation of this limit will make OSHA's PEL for these substances consistent across all regulated sectors.

SUBTILISINS

CAS: 9014–01–1 (Proteolytic enzymes); Chemical Formula: None

H.S. No. 1373

OSHA has no occupational exposure limit for the subtilisins in the construction, agriculture, and maritime industries. The 1987–1988 ACGIH TLV® is a ceiling limit of 0.06 $\mu g/m^3$. NIOSH has no REL for subtilisins but concurs (Ex. 8–47, Table N1) with the limit being proposed. The Agency is proposing a PEL for the construction, agriculture, and maritime industries of 0.06 μ g/m^3

as a 60-minute STEL (measured by a high-volume sampler) for the subtilisins, which is the same PEL recently established by OSHA for the subtilisins in general industry.

The subtilisins are proteolytic bacterial enzymes (produced by various *Bacillus* species) that are used primarily in laundry detergents but also in contact lens cleaners, film processing, and the food industry. They are considered a threat to occupational health because they cause immune-system-mediated bronchoconstriction and respiratory symptoms in addition to primary irritation of the skin and respiratory tract (ACGIH 1986/Ex. 1–3, p. 540; Pepys, Hargreave, Longbottom, and Faux 1969/Ex. 1–568). There are almost no acute toxicity data for subtilisins. The intravenous LD_{50} in mice is 75 mg/kg (RTECS 1990). Instilled into the eyes of rabbits, subtilisins caused severe injury (RTECS 1990).

A study in monkeys (Coate, Busey, Schoenfisch, and Newmann 1978) reports the effects of exposing animals 6 hours/day, 5 days/week for 6 months to atmospheres containing synthetic detergent dust at 1, 10, or 100 mg/m^3 together with enzyme (substilisins) dust at 0.001, 0.01, 0.1, or 1 mg/m^3. Exposures to 10 or 100 mg/m^3 detergent dust together with 0.01 or 1 mg/m^3 enzyme dust produced gross signs of respiratory distress, pulmonary histopathological effects, and pulmonary function impairment in these animals (Coate et al. 1978).

Another study (Thorne, Hillebrand, Magreni et al. 1986) reports that, in guinea pigs, the no-observed-effect level for pulmonary sensitization induced by exposure to the subtilisins for 15 minutes/day for 5 consecutive days was between 0.0083 and 0.041 mg/m^3. Animals exposed on the same regimen at higher levels developed enzyme asthma (Thorne, Hillebrand, Magreni et al. 1986).

In humans, exposure to these proteolytic enzymes causes a feeling of tightness of the chest and breathlessness (Cralley and Cralley 1985, p. 469). There are several documented cases of occupational asthma in workers exposed to subtilisins (Rom 1983, p. 238). That the asthma caused by these substances is immune-system mediated is demonstrated by scratch and intradermal tests that are positive to enzyme extracts of this substance and the presence of IgE antibodies (Rom 1983, p. 239). As many as 40 to 50 percent of workers may become sensitized to subtilisins under conditions of high exposure (Rom 1983, p. 239).

Based on this evidence in humans and animals and available sampling and analytical techniques, OSHA is proposing a 60-minute STEL of 0.06 μg/m^3 for the subtilisins in the construction, agriculture, and maritime industries (using a high-volume sampler). The evidence described above indicates that a short-term limit of 0.06 μg/m^3 for the subtilisins is necessary to reduce the significant risks of respiratory sensitization, skin irritation, and respiratory effects among members of the exposed worker population. OSHA preliminarily concludes that this limit will substantially reduce these significant risks. In addition, promulgation of this limit will make OSHA's PEL for the substilisins consistent across all regulated sectors.

TOLUENE-2,4-DIISOCYANATE
CAS: 584–84–9; Chemical Formula: $CH_3C_6H_3(NCO)_2$
H.S. No. 1398

The current OSHA limit for toluene-2,4-diisocyanate (TDI) in the construction and maritime industries is a ceiling of 0.02 ppm. The Agency has no PEL for TDI in agriculture. The ACGIH has established TLV®s of 0.005 ppm as a TWA and 0.02 ppm as a STEL; NIOSH has RELs of 0.005 ppm as a 10-hour TWA and 0.02 ppm as a 10-minute ceiling for TDI. The Agency is proposing a PEL for TDI in the construction, agriculture, and maritime industries of 0.005 ppm as an 8-hour TWA and 0.02 ppm as a 15-minute STEL. These are the limits recently established for TDI in general industry.

Toluene-2,4-diisocyanate is a clear, colorless liquid that darkens on exposure to sunlight. It has a sharp, irritating, pungent odor. TDI is used in the manufacture of polyurethane foams, elastomers, and coatings (ACGIH 1986, p. 580; Genium MSDS 1989, No. 331).

TDI is one of the most frequently encountered occupational sensitizers, and it is also a known cross-sensitizer. In animals, TDI is a moderate to severe irritant of the skin and eyes (RTECS 1990). The oral LD_{50} in rats is 5800 mg/kg, and the LC_{50} in the same species is 14 ppm for 4 hours (RTECS 1990). In one study, guinea pigs were exposed to TDI concentrations ranging from 0.12 to 10 ppm for 3 hours/day for 5 consecutive days (Karol 1983). No animals in the low-dose group developed antibodies, but 55 percent of the animals exposed to TDI at a concentration greater than 0.36 ppm did develop specific TDI antibodies (Karol 1983). A recent National Toxicology Program lifetime bioassay involving oral gavage of rats with TDI in corn oil showed that this substance produces a statistically significant increase in the incidence of tumors in rats of both sexes and in female mice (NTP 1986). The tumors produced included subcutaneous fibromas and fibrosarcomas, pancreatic adenomas, neoplastic nodules of the liver, and mammary gland fibroadenomas, hemangiomas, hemangiosarcomas, and hepatocellular adenomas (NTP 1986).

The proposed limit, which was recently promulgated by OSHA in general industry, is based on human data showing that some workers can develop sensitization reactions when exposed to TDI at levels below 0.02 ppm. Elkins and colleagues (1962/Ex. 1–138) reviewed the incidence of TDI intoxication in 14 plants in Massachusetts between 1957 and 1962. In eleven instances of TDI intoxication, the average concentration of TDI was 0.015 ppm, and in nine cases the average concentration was below 0.01 ppm. In all plants where the average levels were above 0.01 ppm, TDI had caused respiratory problems. TDI-related respiratory problems were not observed when the average concentration of TDI was maintained below 0.007 ppm (Elkins, McCarl, Brugsch, and Fahy 1962/Ex. 1–138).

Williamson conducted two TDI studies (1964 and 1965) that revealed a 5-percent sensitization rate in 99 workers exposed for 18 months to average TDI concentrations of below 0.02 ppm. The author believed that accidental spills accounted for the high sensitization rate seen in these workers. Williamson also found that six sensitized workers out of 18 exposed to concentrations of TDI below 0.02 ppm for 14 months showed marked decreases in lung function (Williamson 1964 and 1965).

A NOEL (no-observed-effect level) for TDI has been documented. In 1975, Roper and Cromer (Ex. 1–147) failed to observe any symptoms of respiratory illness or changes in pulmonary function in nine employees working in a plant where breathing zone samples showed TDI concentrations of 0.001 to 0.002 ppm. Wegman and colleagues (1974/Ex. 1–112; 1977/Ex. 1–171; 1982/Ex. 1–133) observed a dose-response relationship between exposure and long-term decline in lung function, and this relationship was documented by test results among TDI-exposed employees. Only for those workers exposed to less than 0.002 ppm TDI were the results of lung function tests normal (Wegman, Pagnotto, Fine, and Peters 1974/Ex. 1–112; Wegman, Peters, Pagnotto, and Fine 1977/Ex. 1–171; Wegman, Musk, Main, and Pagnotto 1982/Ex. 1–133).

In the prior rulemaking, the AFL–CIO contended that OSHA had not reduced the PEL for TDI far enough, based on the results of the Wegman et al. study. OSHA responded to the AFL–CIO by noting that other studies in the record suggested that no effects had been seen at this level (Roper and Cromer 1975/Ex. 1–147; Elkins, McCarl, Brugsch, and Fahy 1962/Ex. 1–138). OSHA also noted that the Wegman et al. study (Ex. 1–171, p. 196) described the changes in lung function seen in workers exposed to TDI in the range of 0.002 to 0.005 ppm as "borderline."

The Agency preliminarily concludes that this evidence in humans and animals clearly demonstrates that workers in agriculture, maritime, and construction are at significant risk of pulmonary sensitization reactions, as evidenced by declines in pulmonary function observed among workers exposed to TDI concentrations below this level. OSHA believes that most of the available studies show that a 0.005 TWA PEL and 0.02 ppm STEL will protect these workers against this significant risk; only one of these studies suggests risk at levels below those being proposed. Accordingly, OSHA believes that establishing a 0.005-ppm TWA with a 0.02-ppm STEL is necessary to substantially reduce this significant risk of material health impairment. In addition, promulgation of these limits will make OSHA's PELs for this substance consistent across all regulated sectors.

Preliminary Conclusions for This Group of Sensitizing Toxicants. For the twelve sensitizing agents included in this category of substances, OSHA preliminarily concludes that there are significant occupational risks associated with exposure. The effects caused by such exposures are mediated by the immune system and include skin sensitization, substantial decrements in lung function, bronchoconstriction, asthma, and severe skin irritation, all of which constitute material impairments of health and functional capacity. The reduced or new exposure limits being proposed for these toxic substances will substantially reduce these significant risks in workplaces in construction, maritime, and agriculture and will bring the limits for these sectors into alignment with those recently established in general industry.

14. Substances for Which Proposed Limits Are Based on Avoidance of Cancer

Introduction. For this group of substances, OSHA is proposing limits in construction, maritime, and agriculture on the basis of their potential occupational carcinogenicity. Table C14–1 lists the current OSHA permissible exposure levels (PELs) for these substances in construction and maritime and the PELs being proposed for construction, maritime, and agricultural workplaces. The PELs being proposed are those that are currently in effect in general industry; adoption of the limits being proposed would thus make OSHA's PELs for these substances consistent across all regulated sectors. Table C14–1 also shows the 1987–1988 ACGIH TLV®s, NIOSH RELs, and CAS and HS numbers for these substances.

The following discussion addresses some general aspects of carcinogenicity, together with the methodology used by OSHA in the prior air contaminants and other rulemakings to assess carcinogenic hazards. In this section, quantitative risk models that are widely accepted by the scientific community are used as a means of estimating cancer risks. The multistage model, which is the model primarily used by OSHA, is preferred by most scientists over other models because it is based on a mechanism of cancer that is believed to be biologically more plausible than is the case for the other models.

TABLE C14–1. SUBSTANCE FOR WHICH PROPOSED LIMITS ARE BASED ON AVOIDANCE OF CANCER

H.S. No./chemical name	Cas No.	Current OSHA PEL in construction and maritime*	1987–1988 ACGIH TLV®**	NIOSH REL***	Proposed OSHA PEL in construction, maritime, and agriculture*
1008 Acrylamide	79–06–1	0.3 mg/m³ TWA, Skin	0.03 mg/m³ TWA, Skin, A2.	0.3 mg/m³ TWA	0.03 mg/m³ TWA, Skin
2002 Acrylonitrile	107–13–1	2 ppm TWA, 10 ppm STEL	2 ppm TWA, Skin, A2	1 ppm TWA (8-hour) 10 ppm Ceiling (15-min), Skin†.	2 ppm TWA 10 ppm STEL
1020 Amitrole (3-Amino-1,2,4-triazole).	61–82–5		0.2 mg/m³ TWA		0.2 mg/m³ TWA
2009 ANTU	86–88–4	0.3 mg/m³ TWA	0.3 mg/m³ TWA		0.3 mg/m³ TWA
2011 Arsenic, inorganic (measured as As).	7440–37–1	10 μg/m³ TWA	0.2 mg/m³ TWA (Arsenic and soluble compounds).	2 μg/m³ Ceiling (15-min)†.	10 μg/m³ TWA
2013 Asbestos	Varies	0.2 Fiber/cm³ TWA 1 Fiber/cm³ STEL (30-min)	TLV varies with specific form, A1.	100,000 Fibers/m³, (fibers > 5 μm long), 8-hour TWA (400 Liter sample)†.	0.2 Fiber/cm³ TWA 1 Fiber/cm³ STEL (30-min)
2016 Benzene	71–43–2	1 ppm TWA 5 ppm STEL	10 ppm TWA, A2	0.1 ppm (8-hour) 1ppm ceiling (15-min)†	1 ppm TWA 5 ppm STEL
1033 Beryllium & compounds (measured as Be)****.	7440–41–7	0.002 mg/m³ TWA	0.002 mg/m³ TWA, A2	Do not exceed 0.5 μg/m³†.	2 μg/m³ TWA 5 μg/m³ STEL (30-min) 25 μg/m³ Peak
1073 Carbon tetrachloride	56–23–5	10 ppm TWA, Skin	5 ppm TWA, Skin, A2	2 ppm Ceiling† (60 min, 45-liter sample).	2 ppm TWA
1086 Chloroform Trichloromethane	67–66–3	50 ppm Ceiling	10 ppm TWA, A2	2 ppm Ceiling † (60-min, 45 liter sample).	2 ppm TWA

TABLE C14-1. SUBSTANCE FOR WHICH PROPOSED LIMITS ARE BASED ON AVOIDANCE OF CANCER—Continued

H.S. No./chemical name	Cas No.	Current OSHA PEL in construction and maritime*	1987-1988 ACGIH TLV®**	NIOSH REL***	Proposed OSHA PEL in construction, maritime, and agriculture*
1092 Chromic acid & chromates (measured as CrO_3).	Varies with compound	0.1 mg/m³ TWA	0.05 mg/m³ TWA (measured as Cr).	1 μg/m³ TWA (for carcinogenic hexavalent chromium compounds)†; 25 μg/m³ TWA 50 μg/m³ Ceiling (15-min) (for non-carcinogenic hexavalent chromium compounds, which include chromic acid).	0.1 mg/m³ Ceiling
2039 Coal tar pitch volatiles	65996-93-2	0.2 mg/m³ TWA	0.2 mg/m³ TWA, A1	0.1 mg/m³ TWA (cyclohexane extractable fraction)†.	0.2 mg/m³ TWA
2055 1,2-Dibromo-3-chloropropane (DBCP).	96-12-8	0.001 ppm TWA, Skin		0.01 ppm TWA	0.001 ppm TWA, Skin
1142 Dimethyl sulfate	77-78-1	1 ppm TWA, Skin	0.1 ppm TWA, Skin A2		0.1 ppm TWA, Skin
2083 Ethylene oxide	75-21-8	1 ppm TWA 5ppm Excursion Limit	1 ppm TWA, A2	< 0.1 ppm TWA (8-hour). 5 ppm Ceiling (10-min/day)†.	1 ppm TWA 5 ppm Excursion Limit
2085 Formaldehyde	50-00-0	1 ppm TWA 2 ppm STEL	1 ppm TWA 2 ppm STEL, A2	0.016 ppm TWA (8-hour) 0.1 ppm Ceiling (15-min)†.	1 ppm TWA 2 ppm STEL
1291 2-Nitropropane	79-46-9	25 ppm TWA	10 ppm TWA, A2	*	10 ppm TWA
1308 Perchloroethylene (Tetrachloroethylene).	127-18-4	100 ppm TWA	50 ppm TWA 200 ppm STEL	*	25 ppm TWA
1399 o-Toluidine	95-53-4	5 ppm TWA	2 ppm TWA, Skin, A2		5 ppm TWA, Skin
1400 p-Toluidine	106-49-0		2 ppm TWA, Skin, A2		2 ppm TWA, Skin
1425 Vinyl bromide	593-60-2		5 ppm TWA, A2	*	5 ppm TWA
2167 Vinyl chloride	75-01-4	1 ppm TWA 5 ppm STEL	5 ppm TWA, A1	Lowest reliable detectable concentration†.	1 ppm TWA 5 ppm STEL
1426 Vinyl cyclohexene dioxide	106-67-6		10 ppm TWA, Skin, A2		10 ppm TWA, Skin
1436 Zinc chromates (measured as CrO_3).	Vanes with compound		0.05 mg/m³ TWA, A2 (measured as Cr).	1 μg/m³ TWA†	0.1 mg/m³ Ceiling

*OSHA's PELs do not currently apply in agriculture; OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

**The ACGIH TLV®-TWA is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time. An A1 designation means that the ACGIH has designated this substance a confirmed human carcinogen; an A2 designation means that the substance is classified as a suspected human carcinogen.

***NIOSH TWA limits are for 10 hour/day, 40 hour/week exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

****The standard for beryllium is based on respiratory effects. In this rulemaking, OSHA has not evaluated its carcinogenicity.

†NIOSH considers this substance a potential human carcinogen and recommends that exposures be reduced to the lowest feasible concentration.

Description of the Health Effects. Cancer is an insidious and life-threatening disease that is brought about by the invasion of organ systems by abnormal tissue growth. The abnormal tissue is comprised of cells that have been altered in such a way as to cause unrestricted cell growth. As this unrestricted growth progresses, the abnormal tissue begins to interfere with the vital functions of normal organ systems. In the absence of medical intervention, most forms of cancer are ultimately lethal. In some instances (e.g., colon cancer, breast cancer), life can be prolonged through chemotherapy, radiation treatment, surgery, or some combination of these; however, the quality of life of the victims of cancer is usually severely affected. In other instances, such as lung cancer, there is little hope of survival, even when the tumor is aggressively treated. In past rulemakings on occupational carcinogens (see, for example, Benzene, Ethylene Oxide, Asbestos, and Formaldehyde), OSHA has held that malignant disease clearly constitutes material impairment of health and functional capacity.

An increased risk of developing cancer has been associated with occupational or environmental exposure to a number of chemical substances. The development of chemically induced cancer in humans and animals is a complex and multistep process that is not completely understood. It is currently believed that the mechanism by which cancer develops requires at least two stages: initiation and promotion. Initiation occurs when chemicals interact either directly or indirectly with DNA to cause a heritable mutation. Alterations in DNA structure may cause an incorrect reading of the DNA sequence during replication and result in more altered cells, which may eventually be expressed as a tumor. There is a correlation between substances that are mutagenic in *in vitro* test systems and carcinogenic in *in vivo* situations. Although genotoxic assays are not capable of predicting carcinogenic potential with certainty, these assays are useful for the preliminary identification of substances that may have the potential to cause cancer.

The second stage in the carcinogenic process is promotion. Promotion is considered to be the likely mechanism of action when there is no evidence that a substance interacts with genetic material (e.g., when *in vitro* mutagenicity assays are negative). Peroxisome proliferation, immunosuppression, and hormonal alterations are examples of promotional events; these events facilitate the unrestricted multiplication of initiated cells, leading to the development of cancer. When a substance or its metabolite possesses both initiation and promotion capabilities, it is considered to be a complete carcinogen (i.e., exposure to the substance alone is

sufficient to cause cancer). Examples of such substances recently regulated by OSHA include asbestos, benzene, ethylene oxide, and formaldehyde.

In all of OSHA's past rulemakings for carcinogens, the Agency has used a weight-of-evidence approach to assess the carcinogenic potential of chemical substances. This approach involves examining all available human epidemiologic studies, clinical and case studies, animal studies, mutagenicity studies, and metabolic studies, and then performing a quantitative assessment of the cancer risk to determine the potential of the substance to cause cancer as a result of occupational exposure. OSHA relies heavily on epidemiologic studies of worker populations and on well-conducted animal bioassays to make these determinations. This approach to the promulgation of regulations for carcinogens has been upheld in a number of court decisions. The following discussion summarizes how human and animal studies are used to assess cancer risk.

Epidemiology studies. Epidemiological studies that include detailed exposure data provide the best basis for establishing a causal relationship between exposure to a substance and the onset of cancer in humans. OSHA has relied heavily on epidemiologic evidence in its decisions to promulgate standards for the occupational carcinogens benzene, asbestos, and arsenic. At a minimum, positive epidemiologic studies provide qualitative proof of a causal relationship between exposure to a substance and the development of cancer in humans. A general lack of quantitative exposure data and the long latencies between onset of exposure and appearance of disease may make it difficult to derive quantitative dose-response relationships from epidemiological studies. However, the ability of such studies to link exposures to carcinogens to cancer in humans outweighs these limitations.

Because of the long latency periods associated with chemically induced cancer in humans, these studies cannot be used to detect disease until after irreparable harm has been done. To protect workers or other human populations, it is therefore necessary to assess the risks of such effects before they occur. The data used for this purpose derive from animal bioassays; these data are used to predict human responses and to infer a causal relationship between exposure to a substance and the onset of disease.

Animal data. Animal studies frequently provide the best dose-response data for chemically induced cancer. To use these studies to predict human risk, however, researchers must make certain assumptions; the most important of these assumptions is that physiologic, pharmacokinetic, and biochemical parameters are similar among mammalian species. When data on the metabolic processes of the species of interest (e.g., rats and humans) are available, they may be used to refine the estimates of risk made by extrapolating from animals to humans. Although assumptions must be made before it is possible to use animal data to predict risk in humans, the practice of using animals as surrogates for humans in risk estimation is widely accepted throughout the scientific world. The acceptance of this methodology derives from the observation, after years of conducting thousands of bioassays, that there is a reasonable concordance between carcinogenic effects in animals and carcinogenicity in humans.

Dose-Response and Quantitative Assessment of Risk. A large amount of scientific evidence has accumulated regarding the mechanisms by which carcinogens act and the quantitative relationship between dose and biological response. As a result of these investigations, several mathematical approaches have been developed that permit estimates to be made of the cancer risk that is associated with exposure to low doses of carcinogenic substances.

Since the dominant view of the carcinogenic process holds that most cancer initiators cause irreversible damage to DNA, there is reason to assume that the dose-response of most carcinogens will follow a linear, non-threshold relationship. The Office of Science and Technology Policy (OSTP 1985/Ex. 1–1128) recommends the use of models that incorporate low-dose linearity when the data are limited and there is uncertainty regarding the mechanisms of carcinogenic action. OSHA has observed this practice when conducting risk assessments in prior rulemakings, when the Agency generally relied on the linearized multistage model.

The multistage model used to assess cancer risks associated with exposure to substances in this group is GLOBAL83, a model developed by K.S. Crump and colleagues. If P(d) represents the lifetime risk of cancer at dose d, and A(d) is the extra risk over the background rate at dose d, then the multistage model has the following form:

$$P(d)=1-\exp[-(q_1d+q_2d^2+\ldots+q_kd^k)]$$

where

$$q_i \geq 0$$

$$i=1,2,3,\ldots k$$

and $A(d)=[P(d)-P(0)]/[1-P(0)]$

For a unique set of q_i, this function will adequately describe (or fit) the experimentally derived data. How well the model describes the data may be mathematically determined by what are termed goodness-of-fit tests. Once the model has been fitted to the data, the maximum likelihood estimate (MLE) and the 95-percent upper-confidence limit (UCL) of A(d) are calculated using the 95-percent upper-confidence limit on parameter $q_i(q_i^*)$. The MLE is the point estimate of A(d) and is therefore considered the best estimate of extra risk at dose d.

In the prior air contaminants rulemaking, Dr. Nathan J. Karch, President of Karch Associates and an expert in risk assessment, testified on the appropriateness of using the linearized multistage model to estimate occupational cancer risk:

> The multistage model and the program upon which it is based [GLOBAL83] involves a number of assumptions that are considered unlikely to underestimate risk. At lower doses, the risk is assumed to be linear in dose, and no threshold is assumed to exist.* * * The risk was assumed to be independent of background rates of cancer * * *.
>
> I recognize with growing knowledge of the complexity of various possible mechanisms for cancer induction, that several aspects of the model have come under increasing investigation * * *. Despite what may appear to be conservative assumptions in the use of animal data with the multistage model * * * the multistage model is not likely to be overly conservative at most of the exposures contemplated by this rulemaking. Since the proposed PELs are similar to experimental doses in animals in many cases, the risk estimates from the multistage model tend to be less conservative unless [the PELs] are very high in relation to experimental doses. Moreover, at high doses the risk estimates produced by GLOBAL are similar to those generated by the other commonly used models (Tr. p. 13–50).
>
> An analysis that we perform[ed on] extrapolations from animal data using the multistage model in previous OSHA rulemakings on benzene, ethylene oxide and formaldehyde disclosed that the best, meaning maximum likelihood estimates, from the multistage model were similar and not above the range of estimates from epidemiologic data available * * * [on] workers (Tr. pp. 13–52 to 13–53).

In the prior rulemaking, OSHA asked Dr. Karch to evaluate the scientific literature on the substances in this group to determine whether the data available were suitable for use with the multistage model estimate of the quantitative cancer risk of exposure to these substances. Dr. Karch found the data suitable for the following substances:

acrylamide, amitrole, carbon tetrachloride, chloroform, styrene, o-toluidine, p-toluidine, and vinyl bromide (Ex. 85; Tr. p. 13–50). For the remaining substances examined by Dr. Karch in the earlier rulemaking, the data were judged not to be suitable for risk assessment purposes, and it was therefore not possible to estimate the quantitative cancer risk associated with occupational exposure to these other substances.

For those substances for which suitable data were available, OSHA quantitatively estimated the risk associated with exposure and then relied on these estimates, in part, to make significant risk findings for the air contaminants studied in general industry. OSHA has discussed its approach for making significant risk determinations for cancer-causing substances in a number of rulemakings dealing with carcinogens, and several courts of appeal have upheld OSHA's methodology. For this rulemaking, whose primary purpose is to extend the protection afforded by OSHA's general-industry PELs to workers in construction, maritime, and agriculture, OSHA followed the same methodology as that followed in the prior air contaminants rulemaking and in other rulemakings for carcinogens. [See Arsenic, 48 FR 1816, 1901–1902 (Jan. 14, 1983), upheld *ASARCO* v. *OSHA*, 746 F.2d 483 (9th Cir., 1984); Benzene, 52 FR 34507 (September 11, 1987); Ethylene Oxide, 49 FR 25763 (June 22, 1984), *Public Citizen* v. *Tyson*, 796 F.2d 1479 (D.C. Cir., 1986); Asbestos, 51 FR 22646 (June 20, 1986), *Building and Construction Trades* v. *Brock*, 838 F.2d 1258 (D.C. Cir., 1988).]

The Supreme Court, in the Benzene decision, indicated when a reasonable person might consider a risk significant and take steps to decrease it. The Court stated:

> It is the Agency's responsibility to determine in the first instance what it considers to be a "significant" risk. Some risks are plainly acceptable and others are plainly unacceptable. If, for example, the odds are one in a billion that a person will die from cancer by taking a drink of chlorinated water, the risk clearly could not be considered significant. On the other hand, if the odds are one in a thousand that regular inhalation of gasoline vapors that are 2% benzene will be fatal, a reasonable person might well consider the risk significant and take the appropriate steps to decrease or eliminate it. (*I.U.D.* v. *A.P.I.*, 448 U.S. at 655).

The Court also stated that "while the Agency must support its findings that a certain level of risk exists with substantial evidence, we recognize that its determination that a particular level of risk is 'significant' will be based largely on policy considerations." The Court added that the significant risk determination required by the OSH Act is "not a mathematical straitjacket," and that "OSHA is not required to support its findings with anything approaching scientific certainty." The Court ruled that "a reviewing court [is] to give OSHA some leeway where its findings must be made on the frontiers of scientific knowledge [and that * * *] the Agency is free to use conservative assumptions in interpreting the data with respect to carcinogens, risking error on the side of overprotection rather than under protection" (448 U.S. at 655, 656).

In this proposed rule, OSHA has used the general approach and guidance described above to make the preliminary significant risk determinations for the carcinogens included in this section of the proposal.

The following sections discuss the carcinogenicity evidence for the substances listed in Table C14–1. A brief discussion of the data and a quantitative risk assessment (where appropriate) are included to demonstrate the reduction in cancer risk that could result from lowering OSHA's current PELs or establishing new limits for these potential occupational carcinogens in workplaces in the construction, maritime, and agriculture industries.

ACRYLAMIDE

CAS: 79–06–1; Chemical Formula: $CH_2{=}CHCONH_2$

H.S. No. 1008

OSHA's limit for acrylamide in the construction and maritime industries is 0.3 mg/m³ as an 8-hour TWA, with a skin notation. There is no limit for this substance in agriculture. The ACGIH considers acrylamide a suspected human carcinogen (A2) and has assigned it a TLV®–TWA of 0.03 mg/m³, with a skin notation. NIOSH has a REL of 0.3 mg/m³ as a 10-hour TWA and concurs (Ex. 8–47, Table N6A) with the limit being proposed. The Agency is proposing a TWA limit of 0.03 mg/m³, with a skin notation, for acrylamide in the construction, agriculture, and maritime industries; this is the limit recently established for this substance in general industry.

Acrylamide is a white solid that is widely used as a reactive monomer or intermediate in organic synthesis, and polyacrylamide is a polymer that is used in the manufacture of a host of products, including adhesives, mining chemicals, fibers, pharmaceuticals, animal feed, paper sizing, molded parts, textiles, and coagulant aids (American Cyanamid Company, Ex. 94; ACGIH 1986/Ex. 1–3, p. 12). Acrylamide also is used as a soil stabilizer and conditioner and in sewage and waste treatment (Grant 1986, p. 50; Hawley's 1987, p. 18).

In addition to cancer, acrylamide has caused eye and skin irritation, convulsions and other neurotoxic effects, and reproductive effects in experimental animals. The oral LD_{50} in rats is 124 mg/kg, and the dermal LD_{50} in the same species is 400 mg/kg (RTECS 1990). In rabbits, the lowest lethal dermal dose is 1000 mg/kg (RTECS 1990). Applied to the eyes or skin of rabbits, acrylamide causes mild irritation (RTECS 1990). Neuropathic effects caused by exposure to acrylamide are dose-related and have been seen in rats, cats, and monkeys. Dietary dosing of cats (20 mg/kg/day) for 2 or 3 weeks caused hind-limb weakness and generalized unsteadiness (EPA 1987). Monkeys given 7.1 mg/kg/day orally showed signs of ataxia and motor impairment after 1.5 or 2 months of exposure (EPA 1987). Acrylamide is a cumulative neurotoxin that causes peripheral neuropathy in most experimental animals when the cumulative dose reaches 10 to 50 mg/kg/day; this substance has neuropathic effects regardless of the route of administration: oral, inhalation, intraperitoneal, or dermal (IARC 1986, p. 55). In male mice, oral administration of 35 mg/kg acrylamide twice weekly for 8 weeks caused testicular atrophy and degeneration of the germinal epithelium (IARC 1986, p. 55). A recent study (Zenick, Hope, and Smith 1986) demonstrates that acrylamide has adverse reproductive effects in both male and female rats after oral dosing. This substance also had a significant and adverse effect on the birth weight and the postpartum rate of weight gain in the offspring of treated rats (Zenick, Hope, and Smith 1986).

Two studies are available that demonstrate the carcinogenicity of acrylamide: Johnson, Gorzinsky, Bodner et al. (1986/Ex. 1–825) and Bull, Robinson, Laurie et al. (1984/Ex. 1–252). In the Bull et al. (1984/Ex. 1–252) study, acrylamide was tested as a skin tumor initiator in female Sencar mice; 12-O-tetradecanoylphorbol 3-acetate (TPA) was used as a promoter.

The authors administered six doses of acrylamide that ranged from 0 to 50 mg/kg body weight over a two-week period. A significant dose-related increase in skin tumor incidence was observed for all routes of exposure tested, including topical, gastric intubation, and intraperitoneal injection. The same authors (Bull, Robinson, Laurie et al. 1984/Ex. 1–252) noted a significant dose-related increase in lung adenomas in A/J mice administered acrylamide either by gastric intubation or intraperitoneal injection.

The second study was performed by Johnson et al. (1986/Ex. 1–825) on male and female Fischer 344 rats given 0 to 2.0 mg/kg/day acrylamide in their drinking water for a period of 2 years. During the last 4 months of this study, mortality from cancer was observed at a statistically significant rate in rats exposed at the highest dose level; in addition, tumor incidence increased in animals of both sexes in the highest dose group. In females, tumors of the mammary gland, central nervous system, thyroid gland, oral tissues, uterus, and clitoral gland were seen, while males developed tumors of the central nervous system, thyroid, adrenal gland, and scrotum (Johnson, Gorzinsky, Bodner et al. 1986/Ex. 1–825). Peripheral nerve degeneration was also seen in female rats exposed at the 2-mg/kg/day level (ACGIH 1986/Ex. 1–3, p. 13).

In humans, acrylamide is a demonstrated neuropathogen. Clinical cases involving acrylamide poisoning show that the principal effects of such poisoning include: redness or peeling of the skin of the hands; excessive sweating; loss of weight; limb weakness; sensory loss in the limbs; and urinary incontinence (Le Quesne 1985). In moderate to severe cases, confusion, hallucinations, ataxia, inability to concentrate, and drowsiness occur (Le Quesne 1985). Acute exposure is likely to involve the central nervous system, while cumulative exposure to low doses of acrylamide affects the peripheral nervous system (Proctor, Hughes, and Fischman 1988, p. 56).

A mortality study of 371 acrylamide workers found no statistically significant excesses in cancers of the types hypothesized as likely on the basis of results in animals; however, the number of observed deaths for all cancers was somewhat higher than expected in this cohort (Sobel, Bond, Parsons, and Brenner 1986). A larger cohort study of acrylamide workers (Collins, Swaen, Marsh et al. 1987) found no statistically significant excess in all-cause or cause-specific mortality in these workers.

Risk estimate for acrylamide. For the EPA, Crump et al. (1987) performed a risk assessment for acrylamide, derived in part from the results of the Johnson et al. study (1986/Ex. 1–825). For the purposes of risk assessment, the data for several tumors in female rats were pooled: tumors of the mammary gland, brain and spinal cord, thyroid gland, uterus, and oral cavity. The Maximum Likelihood Estimates (MLEs) and Upper Confidence Limits (UCLs) for the current and proposed exposure limits are presented in Table C14–2. GLOBAL83, a multistage model developed by K.S.Crump, was used to predict these risks. The risk estimate shows that, at OSHA's current PEL in construction and maritime of 0.3 mg/m³, the excess risk of death from cancer for workers exposed over their working lifetimes is 10 per 1,000 workers. At the proposed PEL of 0.03 mg/m³, this number would be reduced to 1 per 1,000 exposed workers.

TABLE C14–2.—MULTISTAGE MODEL ESTIMATES OF CANCER RISK ASSOCIATED WITH WORKING LIFETIME EXPOSURE TO ACRYLAMIDE

Exposure level	Excess cancer deaths per 1,000 workers	
	MLE	UCL
0.3 mg/m³ [a]	10	45
0.03 mg/m³ [b]	1	5

[a] Current OSHA PEL in construction and maritime.
[b] Proposed PEL and current limit in general industry.
MLE = Maximum likelihood estimate of risk.
UCL = 95-percent upper-confidence limit on maximum likelihood estimate of risk.

In the prior rulemaking, a commenter representing the American Cyanamid Company, a major supplier of acrylamide, questioned OSHA's reliance on the Johnson et al. (1986/Ex. 1–825) study and the Bull et al. (1984/Ex. 1–252) study. In response to this commenter, OSHA stated (54 FR 2674, 2675) that (1) it is prudent public health policy to regulate all occupational carcinogens, and not just "complete" carcinogens, to levels that will provide worker protection; (2) the authors of the Bull et al. (1984/Ex. 1–252) study consider acrylamide to be as potent a tumor initiator as ethyl carbamate, a widely recognized tumorigen (Klaassen, Amdur, and Doull 1986/Ex. 1–99, p. 123); and (3) the Bull et al. (1984/Ex. 1–252) study is convincing evidence of acrylamide's carcinogenicity because it shows a dose-related increase in skin tumors in one strain of mouse by three different routes of exposure and the development of lung tumors in another strain of mouse by two routes of administration. OSHA notes further that the International Agency for Research on Cancer (IARC) was also convinced by the evidence presented in these studies; IARC judges the evidence for the carcinogenicity of acrylamide in animals to be sufficient (IARC 1986).

Accordingly, the Agency is proposing an 8-hour TWA PEL of 0.03 mg/m³ for acrylamide in the construction, agriculture, and maritime industries. Because acrylamide is readily absorbed through the skin and can produce systemic effects by this exposure route, OSHA is retaining its skin notation in construction and maritime and proposing it in agriculture. The proposed PEL is based on the significant risk of cancer posed to workers occupationally exposed to this substance. The Agency preliminarily concludes that a 0.03- mg/m³ PEL, and a skin notation, will substantially reduce this significant occupational risk. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ACRYLONITRILE

CAS: 107–13–1; Chemical Formula: CH_2=CHCN

H.S. No. 2002

In general industry, construction, and maritime, OSHA's permissible exposure limits for acrylonitrile (AN) are: 2 ppm as an 8-hour TWA and 10 ppm as a 15-minute short-term limit. Also, the employer shall assure that no employee is exposed to skin contact or eye contact with liquid AN. There are no limits for acrylonitrile in agriculture. A comprehensive OSHA standard for AN was promulgated on October 3, 1978 (43 FR 192). OSHA is proposing limits for AN of 2 ppm as an 8-hour TWA and 10 ppm as a 15-minute STEL in agriculture. Promulgation of these limits will make the PELs for AN consistent across all regulated sectors.

AN is a clear, colorless (when pure), or yellowish liquid with a characteristic odor. Synonyms for AN include acrylon carbaryl, cyanoethylene, fumigrain, 2-propenentrile, VCN, ventox, and vinyl cyanide. The major use of AN is in the production of acrylic and modacrylic fibers by copolymerization with other monomers such as methyl acrylate, methyl methacrylate, styrene, vinyl acetate, vinyl bromide, vinyl chloride, or vinylidene chloride, individually or in combination.

Information regarding the acute toxic effects of acrylonitrile (AN) in both humans and animals is extensive and was discussed at length in the preamble to OSHA's Emergency Temporary Standard (ETS) for AN (42 FR 2586). Based on a careful review of the evidence contained in the 1978 record, OSHA concluded at the time of that rulemaking that acrylonitrile poses a carcinogenic risk to workers. This conclusion was based on the results of experimental studies that showed an increased incidence of tumors in AN-exposed animals and was supplemented by indications of an excess risk of cancer among workers exposed to AN. Moreover, AN has been shown to be capable of inducing mutations, an effect

that has also been observed for many other cancer-causing chemicals.

OSHA's determination that AN is carcinogenic is supported by the findings of other governmental agencies and scientific organizations. Based of the results of industry-sponsored animal studies, the National Cancer Institute's carcinogenicity testing program has "recognized acrylonitrile as carcinogenic to experimental animals" (Ex. 21, Docket H–108). The International Agency for Research on Cancer (IARC) of the World Health Organization stated in a draft report: "* * * the data indicated that acrylonitrile is carcinogenic to rats, producing tumors of the forestomach, brain, and Zymbal gland. Acrylonitrile is also embryotoxic and teratogenic" (Ex. 23, Docket H–108).

In testimony prepared for the 1978 rulemaking, the National Institute for Occupational Safety and Health (NIOSH) concluded: "AN has been shown to be a chemical carcinogen on the basis of animal experiments. The interim results which we have reviewed are significant" (Ex. 42, Docket H–108). Additional information on AN's carcinogenicity is presented in the preamble to OSHA's 1978 AN standard (43 FR 192].

Based on this evidence in humans and animals, OSHA has concluded that exposure to AN can cause cancer in humans and animals. OSHA therefore believes that, in the absence of limits, workers in agriculture are at significant risk of experiencing AN's adverse health effects. The Agency believes that the proposed limits of 2 ppm as an 8-hour TWA and 10 ppm as a 15-minute STEL are necessary to substantially reduce this significant risk of material health impairment among these workers. Promulgation of these limits will also make the PELs for this substance consistent across all OSHA-regulated sectors.

AMITROLE

CAS: 61–82–5; Chemical Formula: $C_2H_4N_4$

H.S. No. 1020

OSHA has no PEL for amitrole in the construction, maritime, or agriculture industries. The level recommended by the ACGIH is a TLV®–TWA of 0.2 mg/m³. There is no NIOSH REL; however, NIOSH concurs (Ex. 8–47, Table N8A) with the PEL OSHA is proposing for this substance. The Agency is proposing an 8-hour TWA limit of 0.2 mg/m³ for amitrole in construction, maritime, and agriculture, which is the limit established by OSHA in the prior rulemaking for general industry.

Amitrole is a white, crystalline powder that is odorless when pure (HSDB 1985). It is used as an herbicide, a plant growth regulator, a cotton defoliant, and a reagent in photography. Amitrole's registered use as an herbicide on food crops was canceled by the EPA in 1971, and it is no longer produced in the United States (HSDB 1985; IARC 1987, p. 295; ACGIH 1986, p. 25); however, this substance is still imported into the United States for use as a specialty herbicide (brush killer) (IARC 1987, p. 295). Amitrole also continues to be used as a post-harvest herbicide around apple and pear trees and as a pre-planting herbicide for kale, maize, potatoes, wheat, and other crops (IARC 1987, p. 295). In addition, this substance is used as a roadside herbicide (IARC 1987, p. 295). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Amitrole is a potent antithyroid agent and has been shown to cause tumors, particularly of the thyroid and pituitary glands, in experimental animals (ACGIH 1986/Ex. 1–3, p. 25). Its tumor-producing activity is thought to be related to its goitrogenic effects, which cause an increase in thyroid-stimulating hormone (TSH). Other antithyroid agents that cause TSH stimulation, such as propylthiouracil, have also been shown to produce thyroid tumors (Guyton 1981/Ex. 1–1002).

The acute toxicity of amitrole is low: The oral LD_{50} in rats is 1100 mg/kg; in mice, this value is 14,700 mg/kg (RTECS 1990). The dermal LD_{50} in rabbits is 10 g/kg (Clayton and Clayton 1981, p. 2702). Repeated exposure of rats to amitrole by intraperitoneal administration at doses of 1 g/kg caused inhibition of liver and kidney catalase; much lower doses (5 mg/kg) sharply reduced iodine uptake by the thyroid gland in the same species (Clayton and Clayton 1981, p. 2703).

Amitrole has been found to be carcinogenic in laboratory animals following dietary exposure to relatively high doses. Attempts to induce tumors by dermal application and subcutaneous injection have been unsuccessful. Studies investigating the carcinogenic potential of amitrole in laboratory animals are reviewed below.

The effects of lifetime exposure to amitrole were investigated in rats, mice, and hamsters fed diets containing 1, 10, or 100 ppm amitrole (Steinhoff, Weber, Mohr, and Boehme 1983/Ex. 1–208). There was a significant increase in the incidence of thyroid tumors in male and female rats and in the incidence of pituitary tumors in female rats exposed to 100 ppm. An excess incidence of tumors was not found in male or female rats exposed to 1 or 10 ppm. The results of this experiment are presented in Table C14–3. Tumor induction was not observed in male or female mice or hamsters. Another study reported negative results for rats fed diets containing 10, 50, or 100 ppm amitrole (Jukes and Schaffer 1960/Ex. 1–213).

TABLE C14–3.—INCIDENCE OF RAT THYROID AND PITUITARY TUMORS ASSOCIATED WITH INGESTION OF AMITROLE

Tumor site	Concentration in diet (ppm)			
	0	1	10	100
Thyroid (Male)				
—Benign	5/75	9/75	4/75	[1] 45/75
—Malignant	3/75	0/75	3/75	[1] 18/75
Thyroid (Female)				
—Benign	7/75	12/75	8/75	[1] 44/75
—Malignant	0/75	1/75	4/75	[1] 28/75
Pituitary (Female)				
—Benign	14/75	20/75	15/75	[1] 36/75
—Malignant	1/75	2/75	4/75	5/75

[1] $p<0.001$, Fisher's Exact Test.

In contrast to the negative results obtained in mice following lifetime dietary exposure to 1, 10, or 100 ppm amitrole, positive results were observed in male and female mice following dietary exposure to higher levels (2192 ppm) of amitrole for 1 year (Innes, Ulland, Valerio et al. 1969/Ex. 1–270). Carcinomas of the thyroid were observed in 89 percent (64/72) of the exposed animals (tumor incidence in the controls was not reported).

Positive results were also observed in mice exposed to 1 percent amitrole in the diet in a lifetime study (alternating exposure for 4 weeks followed by no exposure for 1 week over the lifetime) (Feinstein, Fry, and Staffeld 1978a/Ex. 1–281). Liver tumors developed in 100 percent of the exposed mice; however, the incidence of tumors in unexposed controls was not reported. A small number of thyroid tumors was also seen in these animals. The authors hypothesized that the reason more thyroid tumors were not seen was because the animals died of the high toxic doses before such tumors could be expressed.

The Swedish National Board of Occupational Safety and Health ordered an epidemiological evaluation to assess the incidence of cancer among railroad workers exposed to herbicides (Axelson and Sundell 1974/Ex. 1–812). Amitrole was among the pesticides utilized by these workers. Cohorts were separated into groups according to whether they were exposed to amitrole and

combinations of other herbicides, phenoxy acids and combinations of other herbicides, or other herbicides alone. A statistically significant increase in the incidence of total tumors and lung tumors was found among workers exposed both to amitrole and combinations of other herbicides. Smoking frequency among members of this group was reported to be similar to the frequency of smoking in the general Swedish population.

In a 1980 follow-up to the Axelson and Sundell (1974/Ex. 1–812) study, Axelson and co-workers (Ex. 1–242) combined data from the earlier study with data on workers exposed from 1972 to 1978. Cohorts were divided into the following exposure groups: amitrole alone, phenoxy acids alone, and amitrole and phenoxy acids combined. The reanalyzed data did not show a statistically significant increase in cancer incidence among the workers exposed to amitrole alone; however, the incidence of tumors among workers exposed to amitrole and phenoxy acids together was significantly increased (Axelson, Sundell, Anderson et al. 1980/Ex. 1–242).

Risk estimate for amitrole. The study by Steinhoff et al. (1983/Ex. 1–208) provides sufficient information to use as the basis for the quantitative estimation of the excess cancer risk associated with exposure to amitrole in the workplace. The linearized multistage model was chosen to estimate risk. The incidence of malignant thyroid tumors in female rats was used because these tumors demonstrate a clear monotonic response. Female rats were assumed to weigh 250 g and to consume 25 g of food per day. Human risks were estimated at exposure levels corresponding to the proposed PEL of 0.2 mg/m³, as well as for exposure levels of 0.4 mg/m³ and 1.0 mg/m³. The excess estimated cancer risk, in terms of excess deaths per 1,000 employees, is shown in Table C14–4.

Exposure to 0.2 mg/m³ of amitrole (the limit being proposed) over an occupational lifetime (45 years) is associated with approximately 3 excess cancer deaths per 1,000 employees (0.3 percent) if the MLE is used, and the risk at this level that corresponds to the 95-percent upper-bound estimate is about 4 excess cancer deaths per 1,000 workers. By comparison, the MLEs of risk for lifetime exposure to 0.4 mg/m³ or 1.0 mg/m³ are 5 or 13 excess deaths per 1,000 employees, respectively.

TABLE C14–4.—MULTISTAGE MODEL ESTIMATES OF CANCER RISK ASSOCIATED WITH WORKING LIFETIME EXPOSURE TO AMITROLE

Exposure level	Excess cancer deaths per 1,000 workers	
	MLE	UCL
0.2 mg/m³*	2.7	3.5
0.4 mg/m³	5.3	7.0
1.0 mg/m³	13.0	17.0

* Proposed PEL and current limit in general industry

MLE = Maximum likelihood estimate of risk.
UCL = 95-percent upper-confidence limit on maximum likelihood estimate of risk.

Some commenters in the prior rulemaking urged OSHA to use a different model, i.e., one that incorporates a threshold effect, to estimate the risk of cancer associated with exposure to amitrole (Exs. 3–894; Tr. 3–13, 3–14). Although OSHA recognizes that a threshold-effect level may exist for substances that cause cancer by acting on endocrine-sensitive tissues, the Agency points out that amitrole also has produced liver tumors in mice (Innes, Ulland, Valerio et al. 1969/Ex. 1–270; Feinstein, Fry, and Staffeld 1978a/Ex. 1–281) and, in one instance (Feinstein, Fry, and Staffeld 1978a/Ex. 1–281), the liver tumors appeared at an earlier age and at a higher incidence than did the thyroid tumors. It is not clear from the present data that the mechanism for the development of these amitrole-induced liver tumors is the same as that for amitrole-induced thyroid tumors. OSHA also notes that the proposed 0.2-mg/m³ PEL is, according to the ACGIH's calculations, only a factor of 10 lower than the demonstrated effect level for amitrole-induced effects on thyroid function. OSHA believes that the use of a tenfold uncertainty margin is reasonable when the disease in question is as serious as cancer, whether or not amitrole-induced carcinogenesis follows a dose-threshold pattern. In addition, although the human studies have not been conclusive, they do provide suggestive supporting evidence that exposure to amitrole may increase the risk of cancer among workers.

Accordingly, OSHA preliminarily concludes that the adverse effects resulting from exposure to amitrole potentially include both damage to the thyroid gland and the induction of cancer. OSHA's risk assessment, which is based on animal data, shows that this significant excess cancer risk can be substantially reduced for employees in construction, maritime, or agriculture who may currently be exposed to amitrole at concentrations well above the proposed 0.2-mg/m³ TWA limit. Therefore, OSHA is proposing a 0.2-mg/m³ TWA exposure limit for amitrole in the construction, agriculture, and maritime industries. Promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

alpha-NAPHTHYLTHIOUREA (ANTU)

CAS: 86–88–4; Chemical Formula: $C_{11}H_{10}N_2S$

H.S. No. 2009

The OSHA PEL for alpha-naphthylthiourea (ANTU) in general industry, construction, and maritime is 0.3 mg/m³ as an 8-hour TWA. The Agency has no PEL for ANTU in agriculture. The ACGIH TLV®–TWA for this substance is 0.3 mg/m³. NIOSH has no REL but concurs (Ex. 8–47, Table N3A) with the limit being proposed. OSHA is proposing a 0.3-mg/m³ 8-hour TWA PEL for ANTU in agriculture. This is the same limit as the general industry PEL for ANTU.

ANTU is a colorless to gray crystalline substance that is odorless; it is used as a rodenticide, although it has not been produced in the United States for several years (HSDB 1989). When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

ANTU is more toxic to Norway rats than to animals of other species (Hayes 1982). The lowest reported oral LD_{50} in rats is 6 mg/kg; in monkeys, the oral LD_{50} is 4250 mg/kg (RTECS 1989). ANTU causes pleural effusions and pulmonary edema; before death, acutely poisoned animals had difficulty breathing and developed muscular weakness (Proctor, Hughes, and Fischman 1988, p. 79). Rats fed diets containing 50 to 800 mg/kg ANTU for 2 years showed dose-related hyperplasia of the thyroid (IARC 1983, p. 350). ANTU was tested for carcinogenicity in mice and rats, but these studies were considered inadequate for the purposes of carcinogenicity evaluation (IARC 1983, p. 353).

In humans, exposure to ANTU has been associated with contact dermatitis, destructive changes in the thyroid and adrenal glands, and bladder cancer in exposed workers (ACGIH 1986, p. 36). An impurity in the ANTU, beta-naphthylamine, which is a known human carcinogen, is believed to have been responsible for these cancers. In all, a total of 14 cases of urothelial tumors have been seen since 1982

among rodent exterminators who used ANTU (Orjelick 1975; Davies 1982).

There are no reported cases of accidental ANTU poisoning that have involved ANTU alone, but several poisoning incidents have been attributed to a combination of ANTU and another toxic substance. In one case, a man ingested 80 grams of a rat poison containing 30 percent ANTU and also consumed a large amount of alcohol. This individual subsequently vomited, became cyanotic, and developed fluid in his lungs; he later recovered (Hayes 1982, p. 506). Several poisoning incidents in France involved ANTU and chloralose, another pesticide; all of the poisoned individuals survived but showed tracheobronchial hypersecretions and other ANTU-related signs (Hayes 1982, p. 506).

The evidence described above shows that ANTU causes dose-related pulmonary and endocrine effects in exposed individuals and may cause bladder cancers, although a contaminant in the ANTU, rather than ANTU itself, may have been responsible for these tumors. ANTU can cause these effects when it is ingested, inhaled, and/or absorbed through the skin.

OSHA therefore preliminarily finds that the absence of a PEL for ANTU in agriculture places workers in this sector at significant risk of pulmonary and endocrine damage and perhaps of cancer of the urogenital tract. The Agency believes that the establishment of an 8-hour TWA PEL of 0.3 mg/m^3 will substantially reduce these significant risks of material health impairment. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ARSENIC, INORGANIC COMPOUNDS (as As)

CAS: 7440–38–2; Chemical Formula: Varies with Compound

H.S. No. 2011

In general industry, construction, and maritime, OSHA's permissible exposure limit for inorganic arsenic is 10 μg/m^3 averaged over an 8-hour period. There is no limit in agriculture. The ACGIH has assigned arsenic and soluble compounds a TLV^a–TWA of 0.2 mg/m^3. NIOSH considers these substances potential occupational carcinogens and has issued a REL of 2 μg/m^3 as a 15-minute ceiling. OSHA proposes to establish a PEL of 10 μg/m^3 TWA for inorganic arsenic in agriculture. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

Arsenic (As) is commonly present in amounts ranging from less than 0.001 percent to 6 percent of the sulfide ores mined for their copper, lead, zinc, gold, and silver content. Arsenic is also widely distributed naturally in small amounts (2–5 ppm) throughout the earth's crust. For example, it is found in iron ore and coal. Further, trace amounts of organic arsenic (less than 1 ppm) are naturally present in most living organisms, including those used for food.

Approximately 97 percent of arsenic enters end-product manufacture in the form of arsenic trioxide (As_2O_3 or "white arsenic"). This compound, which is used in the synthesis of many other arsenic compounds, is released by and obtained as a by-product of the smelting of sulfide ores of copper, lead, and zinc. Arsenic is generally regarded, however, as a troublesome impurity in these metals which is eliminated through the smelting process.

Arsenic and its compounds have a variety of applications. The major use (comprising approximately 69 percent of U.S. consumption) is for insecticides and herbicides. Approximately 11 percent of the total U.S. arsenic trioxide consumption is used in glass production as a clarifying and reducing agent. Arsenic is also used as a defoliant in cotton harvesting. When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

OSHA believes that exposure to trivalent arsenic causes respiratory cancer. Studies in the copper smelting industry (Lee and Fraumeni 1969, Pinto and Enterline 1978, Rencher and Carter 1977) have shown a statistically significant increase in lung cancer mortality among workers exposed to arsenic trioxide, sulfur dioxide, and other co-contaminants released during the smelting of copper ores. As Lee and Fraumeni stated, it is impossible to differentiate in these cases between the effects of exposure to arsenic and these other co-contaminants. However, the finding that lung cancer mortality increased consistently with degree and duration of exposure to arsenic trioxide provides substantial evidence that exposure to trivalent arsenic is a cause of respiratory cancer. These data, and particularly those of Lee and Fraumeni and Pinto and Enterline, were discussed in detail in the preamble to the arsenic standard (43 FR 19584, May 5, 1978 and 48 FR 1864, January 14, 1983) and accordingly will not be repeated here. Many experts testified during the hearing for that rulemaking. Both Dr. Fraumeni (Tr. 168, Docket H–037) and Dr. Kraybill (Tr. 151, Docket H–037) stated that inorganic arsenic is strongly incriminated as an occupational carcinogen, and NIOSH recognized that, although each epidemiological study alone has limitations, ". . . when all reports of occupational exposure to inorganic arsenic are considered together,. . . it [is] undeniable that there have been carcinogenic effects which must be attributed to inorganic arsenic" (Tr. 54–5, Docket H–037). Thus, the National Cancer Institute and NIOSH both recommended that pentavalent arsenic be treated as a carcinogen.

On the other hand, the American Wood Preservers Institute (AWPI) argued in that rulemaking that the evidence was not sufficiently persuasive for OSHA to make such a determination. The AWPI suggested that pentavalent arsenic should not be implicated as a causal factor because the studies suggesting its involvement also involved exposure to trivalent arsenic. However, after a thorough review of the record as a whole, OSHA determined that the weight of the evidence clearly pointed to the carcinogenicity of pentavalent arsenic.

Based on this evidence in humans, OSHA concluded in the earlier 6(b) rulemaking that exposure to inorganic arsenic causes cancer in humans. OSHA therefore believes that, in the absence of a limit, workers in agriculture who are exposed to inorganic arsenic are at significant risk of developing cancer. The Agency believes that the proposed 8-hour TWA limit of 10 μg/m^3 is necessary to substantially reduce these risks of material health impairment among this group of workers. Promulgation of this limit will also make the PEL for this substance consistent across all OSHA-regulated sectors.

ASBESTOS

CAS: varies; Chemical Formula: varies

H.S. No. 2013

In general industry, construction, and maritime, OSHA's permissible exposure limits for asbestos are 0.2 fiber per cubic centimeter of air (f/cc) as an 8-hour time weighted average airborne concentration and 1 f/cc as a 30-minute excursion limit. OSHA's most recent comprehensive standard for asbestos in these industries was promulgated in 1986. OSHA has no PEL for asbestos in agriculture. Therefore, OSHA is proposing to establish an 8-hour TWA PEL of 0.2 f/cc and a 30-minute excursion limit of 1 f/cc for asbestos in agriculture. Promulgation of these limits will make the PELs for asbestos consistent across all OSHA-regulated sectors.

Asbestos is the name given to a class of magnesium silicate minerals that occur in fibrous form. Minerals that are included in this group are chrysotile, crocidolite, amosite, anthophyllite asbestos, tremolite asbestos, and actinolite asbestos.

Asbestos, tremolite, anthophyllite, and actinolite have been used in the manufacture of heat-resistant clothing, automobile brake and clutch linings, and a variety of building materials (including floor tiles, roofing felts, ceiling tiles, asbestos-cement pipe and sheet, and fire-resistant drywall). Asbestos, tremolite, anthophyllite, and actinolite are also present in pipe and boiler insulation materials and in sprayed-on materials located in beams, in crawl spaces, and between walls.

OSHA is aware of no instance in which exposure to a toxic substance has more clearly demonstrated detrimental health effects on humans than asbestos exposure. The diseases caused by asbestos exposure are life-threatening or disabling; among these diseases are lung cancer, cancer of the mesothelial lining of the pleura and peritoneum, asbestosis, and gastrointestinal cancer. Of all the diseases caused by asbestos, lung cancer constitutes the greatest health risk for American asbestos workers. Lung cancer has been responsible for more than half of the excess mortality from asbestos exposure in some occupational cohorts.

The relationship between lung cancer and asbestos exposure has been established in numerous epidemiological studies of diverse groups. Asbestos-induced lung cancer usually has a latency period in excess of 20 years, and this cancer may be manifested at a younger age than is true for lung cancer victims who are not exposed to asbestos (Craighead et al., Ex. 84–033, Docket H–033). Few cases of lung cancer are curable, despite advances in medical and surgical oncology. Only 9 percent of lung cancer patients survive for 5 or more years after diagnosis (American Cancer Society, Ex. 84–160, Docket H–033). Asbestos exposure acts synergistically with cigarette smoking to multiply the risk of developing lung cancer.

Many studies also have shown conclusively that mesothelioma is associated with asbestos exposure. In some asbestos-exposed occupational groups, 10 to 18 percent of deaths have been attributable to malignant mesotheliomas of the pleura and peritoneum, a condition that is extremely rare in persons not exposed to asbestos. Generally, a latency period of at least 25 to 30 years is required before mesotheliomas are observed in an occupational cohort, although some victims of mesothelioma have had latency periods exceeding 40 years (Craighead et al., Ex. 84–033, Docket H–033). This form of cancer is usually fatal within a year of diagnosis.

Some epidemiological studies of asbestos-exposed persons have shown increases in esophageal, stomach, colo-rectal, kidney, laryngeal, pharyngeal, and buccal cavity cancers. Although the increased risk of cancers at these sites is not as great as the increased risk of lung cancer and mesothelioma, the increase is of considerable importance because of the high background rates, and therefore the large number of victims, associated with some of these tumors in the general population. For example, a 50 percent increase in a common cancer such as colo-rectal cancer results in many more deaths than a 50 percent increase in a rare cancer.

Asbestosis is pulmonary fibrosis caused by the accumulation of asbestos fibers in the lungs. The adverse effects of asbestosis range from shortness of breath during exertion to cyanosis, effusions of serous fluid, respiratory failure, cardiac hypertrophy, and death. Asbestosis is often a progressive disease, even in the absence of continued exposure. The symptoms of the disease are shortness of breath, cough, fatigue, and vague feelings of sickness. When the fibrosis worsens, shortness of breath occurs even at rest. One clinical feature of early asbestosis as well as other lung diseases is end-respiratory crackles (rales).

Diagnosis of asbestosis is based on the presence of characteristic radiological changes, symptoms, rales, other clinical features of fibrosing lung diseases, and a history of exposure to asbestos.

Asbestos exposure can cause pleural and/or other pulmonary disease. Pleural plaques are a marker of asbestos exposure and may develop within 10 to 20 years after the initial exposure. Plaques are opaque patches visible on chest x-rays that consist of dense strands of collagen (connective tissue protein) lined by mesothelial cells. All commercially used types of asbestos induce pleural plaques. These conclusions about asbestos' health effects are widely accepted both in the U.S. and abroad, and are discussed extensively in the preamble to OSHA's 1986 standard for asbestos, which describes the evidence linking asbestos exposure to lung cancer, mesothelioma, gastrointestinal cancer, and non-malignant respiratory diseases such as asbestosis.

Based on this evidence, OSHA concluded in the earlier asbestos rulemakings that exposure to asbestos can cause cancer in humans. OSHA therefore believes that, in the absence of a PEL, workers in agriculture are at significant risk of experiencing these adverse and often life-threatening health effects. The Agency believes that the proposed limits of 0.2 f/cc as an 8-hour TWA and 1 f/cc as a 30-minute short term limit are necessary to substantially reduce these risks of material health impairment among this group of workers. Promulgation of these limits will make the PELs for asbestos consistent across all OSHA-regulated sectors. Although the current PELs for asbestos are 0.2 f/cc (TWA) and 1 f/cc (30-minute limit), OSHA is currently engaged in rulemaking on asbestos. If a final decision is made to lower one or both of these PELs prior to the issuance of this Air Contaminants standard, OSHA will consider adopting the new limit(s) in agriculture. OSHA requests comments on this issue from interested parties.

BENZENE

CAS No.: 71–43–2; Chemical Formula: C_6H_6

H.S. No. 2016

In general industry, construction, and maritime, OSHA's permissible exposure limits for benzene are 1 ppm as an 8-hour TWA and 5 ppm as a 15-minute STEL. OSHA's comprehensive standard for benzene was promulgated on September 11, 1987 (52 FR 176). There are no PELs for benzene in agriculture, and OSHA is therefore proposing to establish limits in that sector. Promulgation of these limits will make the PELs for benzene consistent across all OSHA-regulated sectors.

Benzene is a clear, colorless, non-corrosive, highly flammable liquid with a strong rather pleasant odor. The low boiling point and high vapor pressure of benzene causes rapid evaporation under ordinary atmospheric conditions and generates vapors that are nearly three times heavier than air.

Benzene is produced primarily by the petrochemical and petroleum refining industries by a process called catalytic reformation, which converts certain lower octane hydrocarbons into higher octane aromatics. These two industries are responsible for 98 percent of the total U.S. production of benzene. Recovery through catalytic reformation and thermal cracking of petroleum hydrocarbons, including the benzene formed from the dealkylation of toluene, accounts for approximately 75percent of the total quantity of benzene produced.

The first major industrial use of benzene was as a solvent in the rubber

industry just before World War I. Large quantities of benzene are used to manufacture other organic compounds such as ethylbenzene, styrene, cumene, and cyclohexanol. This situation led to greatly increased uses of benzene as a solvent in the artificial leather, rubber goods, and rotogravure industries.

Many products contain benzene exclusively as the result of contamination. Benzene is a naturally occurring compound in crude oil and natural gas. Unreacted benzene may also be present in major benzene derivatives (e.g., methylbenzene) or in other specialty chemicals that use benzene as a feedstock (e.g., dicyclopentadiene). The presence of unreacted benzene in major derivatives or other specialty chemicals is undesirable from the producer's point of view and is not generally useful to product users.

Industries and processes currently using benzene or liquids containing benzene include the chemical, printing, lithograph, rubber cement, rubber fabricating, paint, varnish, stain remover, adhesive, and petroleum industries. Benzene is also used extensively in chemical laboratories as a solvent and as a reactant in numerous chemical applications. Where benzene is produced, used, or stored in large amounts, it is generally contained in enclosed systems, although exposures can occur during liquid transfer operations, from equipment leakage and carryover losses, and in maintenance operations.

The scientific literature has documented hundreds of cases of leukemia, aplastic anemia, and other blood abnormalities that have been associated with benzene exposure. Epidemiologic studies of workers exposed to benzene have demonstrated significant excesses of leukemia, multiple myeloma, and lymphatic cancers as well as chromosomal aberrations in these workers. Several of these studies provide a reasonable basis for quantitative cancer risk assessment. More recently, experimental animal studies have demonstrated the induction of cancer, chromosomal damage, and bone marrow toxicity as a result of specific exposures to benzene. All of this information has been used to the extent feasible in OSHA's evaluation of the risk associated with various benzene exposure levels. Information regarding the health effects of benzene on both humans and animals was discussed at length in the preamble to OSHA's Final Rule on Occupational Exposure to Benzene (52 FR 176, September 11, 1987).

Although benzene exposure has been associated with leukemia, aplastic anemia, multiple myeloma, various forms of lymphoma, myelofibrosis, pancytopenia, and depression of singular blood cell lines, OSHA's risk assessments were based preferentially on experimental studies involving the induction of solid tumors in mice and rats. OSHA did not include in its quantitative risk assessments risks of the other conditions mentioned above because they were for the most part identified in case reports and may have underestimated the relative risks associated with benzene exposure.

Based on the evidence in humans and animals described by OSHA in the preamble to the final 6(b) rule for benzene, OSHA concluded that exposure to benzene causes numerous adverse and potentially life-threatening health effects. OSHA therefore believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed limits of 1 ppm as an 8-hour TWA and 5 ppm as a 15-minute STEL are necessary to substantially reduce these risks of material health impairment among these workers. Promulgation of these limits will also make the PEL for benzene consistent across all OSHA-regulated sectors.

BERYLLIUM AND COMPOUNDS (As Be)

CAS No.: 7440–41–7; Chemical Formula: Varies with Compound

H.S. No. 1033

OSHA's current limit for beryllium and its compounds (measured as Be) in the construction and maritime industries is 0.002 mg/m³ (2 μg/m³) as an 8-hour TWA. There is no limit in agriculture for these substances. The ACGIH considers beryllium a suspect human carcinogen (A2) and has assigned a TLV®–TWA for beryllium and compounds of 0.002 mg/m³ (2 μg/m³). NIOSH considers beryllium a potential human carcinogen and recommends that exposure to beryllium and compounds not exceed 0.5 μg/m³. OSHA is proposing to extend its TWA PEL of 0.002 mg/m³ (2 μg/m³) to agriculture and to add a 0.005 mg/m³ (5 μg/m³) 30-minute STEL and a 0.025 mg/m³ (25 μg/m³) peak for beryllium and compounds in the construction, agriculture, and maritime industries. These are the limits for beryllium and its compounds currently in effect in general industry.

Beryllium is a metallic element that is hard, brittle, and grayish-white in color. It is used as a hardening agent in alloys and in non-sparking tools, drill bits, watch balance wheels, airplane brakes, electrical equipment, springs, valves, computers, gyroscopes, and in ceramics manufacturing (Clayton and Clayton 1981, p. 1537). It also has uses in space and nuclear technologies (HSDB 1986; ACGIH 1986, p. 56).

Occupational exposure to beryllium has long been associated with the development of two distinct diseases: Acute chemical pneumonitis and chronic beryllium disease, which is a progressive and debilitating lung disease. Exposure to soluble beryllium salts and to finely divided beryllium oxide adversely affects the skin, mucous membranes, and respiratory tract and leads to chemical pneumonitis; the airborne concentrations of beryllium believed to be associated with acute pulmonary effects are on the order of 100 μg/m³ (Eisenbud et al. 1948). This form of beryllium disease has not occurred in the United States since the 1960s, in large part due to the establishment of a 25 μg/m³ 30-minute short-term limit by the Atomic Energy Commission in 1949 and OSHA's adoption of this limit, through the Section 6(a) process, in 1971.

Chronic beryllium disease is a systemic, granulomatous disease caused by low-level exposure to beryllium over periods ranging from several months to many years. The principal signs and symptoms of chronic beryllium disease are dyspnea, weight loss, chest pain, cough, arthralgia, and fatigue, and clinical manifestations may include interstitial crackles, liver and spleen enlargement, and, in severe cases, clubbing of the fingers. Severe cases may lead to cor pulmonole and right heart failure. Workers with chronic beryllium disease may show one or more of the following ventilatory defects: restrictive lung disease; mixed restrictive and obstructive lung disease; or diminished carbon monoxide diffusing capacity. Three stages of the disease have been identified by lung x-ray: A fine, diffuse granularity; a diffuse reticular pattern; and, finally, distinct nodules (Parkes 1985).

Chronic beryllium disease results from a cell-mediated immune response of the lung to beryllium exposure. Although it had been suggested in the early 1950s that some workers appeared to be allergic to beryllium (Sterner and Eisenbud 1951), the correlation between chronic disease and sensitization to beryllium was not established until 20 years later (Deodhar et al. 1973). Advances in scientists' understanding of immunology and in techniques for the isolation and culture of lymphocytes have led to improvements in the reliability, sensitivity, and specificity of *in vitro* tests for beryllium sensitization. These tests involve analysis of T-cells

recovered either from the blood (Kreiss et al. 1989) or bronchoalveolar lavage fluid (Rossman et al. 1988) of individuals suspected of being sensitized.

At the time NIOSH's criteria document on beryllium was published (NIOSH 1972a), NIOSH judged the evidence that beryllium caused cancer to be equivocal. In testimony at OSHA's 1977 hearing on a standard for beryllium, however, NIOSH presented additional epidemiologic and animal evidence indicating that beryllium is carcinogenic. In particular, NIOSH (1977o; Baier 1977b/Ex. 1–831) cited the studies of Bayliss and Wagoner (1977) and Mancuso (1977), which showed significant increases in bronchogenic cancer among beryllium-exposed workers. NIOSH therefore recommended at the 1977 hearing that exposure to beryllium not exceed the reliable limit of detection of 0.5 $\mu g/m^3$ (NIOSH 1977o; Baier 1977b/Ex. 1–831).

In the recent air contaminants rulemaking for general industry, OSHA determined that, because of the extensive record developed for beryllium following OSHA's 1975 proposed rule for beryllium, the issues to be resolved were too complex to address during the Air Contaminants rulemaking. Accordingly, OSHA retained the Agency's general industry PELs of 0.002 mg/m^3 TWA, 0.005 mg/m^3 as a 30-minute STEL, and 0.025 mg/m^3 as a ceiling to provide workers with continued protection against beryllium-related chronic lung disease. OSHA believes that, in the absence of a limit in agriculture, exposed workers are at significant risk of developing chronic beryllium disease. OSHA preliminarily concludes that extending the 0.002 mg/m^3 TWA PEL, 0.005 mg/m^3 STEL, and 0.025 mg/m^3 peak to construction, maritime, and agriculture will substantially reduce this risk. OSHA believes that the STEL and ceiling are necessary to aid in protecting against initial sensitization to beryllium. By analogy with the effects seen with other inorganic sensitizers such as cobalt, nickel, and chromium (Nemery 1990), short-term high exposure to antigens is believed to be associated with the development of sensitization, and OSHA believes that the STEL and ceiling will ensure that exposures are kept under strict control at all times. Promulgation of these limits in construction, maritime, and agriculture will also make the TWA, STEL, and ceiling for beryllium and its compounds consistent across all regulated sectors.

CARBON TETRACHLORIDE
CAS: 56–23–5; Chemical Formula: CCl_4
H.S. No. 1073

The OSHA PEL for carbon tetrachloride in the construction and maritime industries is 10 ppm as an 8-hour TWA, with a skin notation. There is no limit in agriculture. The ACGIH has established a 5-ppm TLV®–TWA limit, with a skin notation, for this substance. NIOSH considers carbon tetrachloride a potential human carcinogen and has issued a REL of 2 ppm as a ceiling limit (45 liter, 60-minute sample). However, NIOSH concurs (Ex. 8–47, Table N6A) with the limit the Agency is proposing. OSHA is proposing to delete the skin notation and to establish a PEL for carbon tetrachloride of 2 ppm as an 8-hour TWA for workplaces in the construction, agriculture, and maritime industries. This is the limit recently established for carbon tetrachloride in general industry.

Carbon tetrachloride is a heavy, mobile liquid with a sweet odor. Although carbon tetrachloride was once widely used as a dry-cleaning agent and solvent, it has now largely been replaced by less toxic substances in these uses. It is currently used in the manufacture of fluorocarbon propellants, many organic compounds, and in refrigerants and propellants (HSDB 1985).

In addition to cancer, exposure to carbon tetrachloride causes liver, kidney, and lung damage in humans and animals. The oral LD_{50} in rats is 2350 mg/kg, and the LC_{50} in the same species is 8000 ppm for 4 hours (RTECS 1990). A number of studies in experimental animals have shown that single oral doses of carbon tetrachloride ranging from about 100 to 4000 mg/kg produce fatty infiltration of the liver, loss of cytochrome P–450 and other enzymes, and histological changes in the liver (EPA 1985). Hepatocellular necrosis also may occur (EPA 1985). Rats given single oral doses of 4000 mg/kg of carbon tetrachloride showed damage to the proximal tubules of the kidney, clara cells of the lung, and endothelial lining of the lung at autopsy (EPA 1987).

In a subchronic inhalation study, guinea pigs, rats, monkeys, rabbits, and dogs exposed continuously for 90 days to a 61-mg/m^3 concentration of carbon tetrachloride showed signs of liver toxicity, while animals exposed to a 6.1-mg/m^3 concentration on the same regimen showed no adverse effects (Prendergast et al. 1967).

In chronic studies, rats gavaged with carbon tetrachloride at a 1 mg/kg-dose 5 days/week for 12 weeks failed to show adverse effects at autopsy, while those exposed to 10 or 33 mg/kg doses on the same regimen showed centrilobular vacuolization and necrosis of hepatocytes at post mortem (Bruchner et al. 1986).

Human fatalities have occurred in adults who ingested as little as 1.5 ml of carbon tetrachloride, and inhalation exposure to 1500 mg/m^3 has been associated with systemic poisoning and death (EPA 1985). Acute carbon tetrachloride poisoning in humans takes the following course: hepatic and renal failure, followed by pulmonary edema and cardiac failure (Rom 1983, p. 399). The following paragraphs describe the available data on carbon tetrachloride's carcinogenicity in humans and animals.

There have been three case reports in humans of liver tumors developing after carbon tetrachloride exposure (Tracy and Sherlock 1968/Ex. 1–152; Johnstone 1948/Ex. 1–817; Simler, Maurer, and Mandard 1964/Ex. 1–225). In each case, the patient had been acutely overexposed to carbon tetrachloride and had thereafter developed nausea, stomach pains, and signs of severe liver damage.

Blair, Decoufle, and Grauman (1979/Ex. 1–150) studied causes of death in 330 laundry and dry cleaning workers potentially exposed to carbon tetrachloride, as well as to trichloroethylene and tetrachloroethylene. Causes of death based on death certificates were compared to the age, sex, race, and cause-specific distribution of U.S. deaths for the same time period. The proportionate mortality ratio (PMR) for the cohort being studied for all malignant neoplasms was 128, which was statistically significant, indicating that the study group had a 28-percent higher proportion of total deaths due to cancer than the proportion in the U.S. general population. The excess cancer deaths were due to liver, lung, and cervical cancer and leukemia. Although the excess lung and cervical cancer deaths may reflect socioeconomic differences among these workers, the excess liver cancer seen in this study is consistent with findings in animal studies involving carbon tetrachloride.

In animals, carbon tetrachloride has produced hepatocellular carcinomas in all species evaluated (rats, mice, and hamsters). Male rats were given 47 or 94 mg/kg carbon tetrachloride and females were given 80 or 159 mg/kg by gavage for 78 weeks (NCI 1976a/Ex. 1–119; NCI 1976b/Ex. 1–168; NCI 1977b/Ex. 1–169). The incidence of hepatocellular carcinomas was increased in animals exposed to carbon tetrachloride as compared with pooled colony controls but was statistically significant only for low-dose females. The lower incidence of carcinomas in female rats at the high

dose (1/49) compared with that at the low dose (4/49) was attributed by the authors to the increased lethality that occurred among these rats before tumors could be expressed.

In this same study, mice of both sexes received 1250 or 2500 mg/kg carbon tetrachloride by gavage. Hepatocellular carcinomas were found in 49/49 low-dose and 47/48 high-dose males (compared with 5/77 in the control males) and in 40/40 low-dose and 43/45 high-dose females (compared with 1/80 in the control females) (NCI 1976a/Ex. 1–119; NCI 1976b/Ex. 1–168; NCI 1977b/Ex. 1–169).

Edwards, Heston, and Dalton (1942/Ex. 1–68) administered carbon tetrachloride by gavage (64 mg/mouse administered 46 times over four months) to a mouse strain known to have a low incidence of spontaneous hepatomas. The resulting incidence of hepatomas in these animals was 52 percent (28/54) for males and 32 percent (6/19) for females. Previous hepatoma incidence data for untreated mice of this strain were 2/71 for males and 0/81 for females. Carbon tetrachloride administered by gavage has also been shown to produce neoplastic changes in the livers of four additional strains of mice (Andervont 1958/Ex. 1–81; Edwards 1941/Ex. 1–86; Eschenbrenner and Miller 1943/Ex. 1–113).

Della Porta, Terracini, and Shubik (1961/Ex. 1–136) gave weekly gavage treatments of 10 to 20 μg carbon tetrachloride to hamsters for 30 weeks, and the animals were observed for an additional 25 weeks. All 10 hamsters dying or killed between weeks 43 and 55 had liver cell carcinomas, compared with 0/254 among the historical controls.

Risk estimate for carbon tetrachloride. Three animal data sets have sufficient dose-response information to allow quantitative risk estimation: The rat and mouse bioassay data (NCI 1976a/Ex. 1–119; NCI 1976b/Ex. 1–168; NCI 1977b/Ex. 1–169) and the Edwards, Heston, and Dalton (1942/Ex. 1–68) mouse data. To increase sample sizes, the data were pooled for male and female animals in each of the three studies. The estimated risks shown in Table C14–7 are the geometric means of the risk calculated from each of the three data sets.

Inhalation risk in humans was calculated assuming an air intake of 20 m^3 per 24-hour day and a 40-percent absorption rate (EPA 1984a/Ex. 1–1130). All three studies suggest that a common biological mechanism, cell death and regeneration, occurs and leads to the development of the same tumor type.

TABLE C14–7.—MULTISTAGE MODEL ESTIMATES OF CANCER RISK ASSOCIATED WITH WORKING LIFETIME EXPOSURE TO CARBON TETRACHLORIDE

Exposure level	Excess cancer deaths per 1,000 workers	
	MLE	UCL
2 ppm[a]	3.7	5.2
5 ppm[b]	9.2	13.0
10 ppm[c]	17.9	26.0

[a] Proposed OSHA PEL in construction, maritime, and agriculture.
[b] ACGIH TLV®.
[c] OSHA TWA PEL in construction and maritime.
MLE = Maximum likelihood estimate of risk.
UCL = 95-percent upper-confidence limit on the maximum likelihood estimate of risk.

Table C14–7 presents the estimates of lifetime human risk from carbon tetrachloride exposure, calculated by the linearized multistage model (GLOBAL83) at the proposed 2-ppm limit, the ACGIH limit of 5 ppm, and the current 10-ppm OSHA PEL in construction and maritime. Both the maximum likelihood estimates (MLE) and the 95-percent upper-confidence limits of human risk are given, as well as the corresponding expected number of excess cancer deaths per 1,000 workers exposed over a working lifetime. Based on this risk estimate, the MLE at the current OSHA limit of 10 ppm is 17.9 excess deaths per 1,000 exposed workers, an estimate that clearly demonstrates a significant cancer risk at the current PEL in construction and maritime.

Risk at the current ACGIH limit of 5 ppm is estimated to be 9.2 excess deaths per 1,000 workers exposed over their working lifetimes. At the proposed limit of 2 ppm, residual risk continues to be significant, according to the Supreme Court's guidance in the Benzene decision and the analysis presented in the introduction to this section; the risk predicted at 2 ppm is 3.7 excess deaths per 1,000 workers exposed over their working lifetimes. However, risk at the 2 ppm limit is substantially reduced compared with risk at the current OSHA PEL of 10 ppm. Because of the time and resource constraints imposed by the effort to issue a generic air contaminants standard of this magnitude, OSHA did not determine further what alternative lower limit would eliminate or substantially reduce the remaining significant risk and still be feasible. OSHA will reconsider carbon tetrachloride in the next round of PEL updates. In the interim, the risk remaining at the 2 ppm limit is substantially reduced compared to the risk associated with exposure at the current OSHA PEL of 10 ppm.

Based on the evidence presented above and the quantitative estimates of carbon tetrachloride-related cancer risk, OSHA preliminarily concludes that occupational exposure to carbon tetrachloride at the former 10-ppm PEL presents a significant risk of cancer to workers in the construction and maritime industry sectors; because there is no current PEL for carbon tetrachloride in agriculture, OSHA also believes that there is a significant risk of cancer to exposed workers in that sector. OSHA's risk assessment shows the proposed limit of 2 ppm will substantially reduce this risk. Accordingly, OSHA is proposing a PEL of 2 ppm as an 8-hour TWA in construction, maritime, and agriculture. If promulgated as proposed, the permissible exposure limit for carbon tetrachloride would then be the same in all industry sectors. Because the level of residual risk at 2 ppm may be significant, OSHA is committed to reconsidering this PEL in the first update of the Air Contaminants standard. In that rulemaking, OSHA will consider a lesser PEL for all sectors. As discussed in the Legal Authorities section above, OSHA believes that this approach is necessary to provide worker protection in construction, maritime, and agriculture within a reasonable time frame and that this is an appropriate use of the Agency's authority to establish rulemaking priorities.

CHLOROFORM

CAS: 67–66–3; Chemical Formula: $CHCl_3$
H.S. No. 1086

The current OSHA PEL for chloroform in construction and maritime is 50 ppm as a ceiling limit. The Agency has no PEL for this substance in agriculture. The ACGIH has established a TLV®–TWA of 10 ppm and assigned chloroform an A2 (suspect human occupational carcinogen) designation. NIOSH considers chloroform a potential human carcinogen and has issued a REL for this substance of 2 ppm as a ceiling limit (45 liter, 60-minute sample); however, NIOSH concurs (Ex. 8–47, Table N6A) with the limit the Agency is proposing. OSHA is proposing a PEL for chloroform in construction, maritime, and agriculture of 2 ppm as an 8-hour TWA. This is the limit recently established for chloroform in general industry.

Chloroform is a clear, colorless, nonflammable, volatile liquid with a pleasant odor. Chloroform was one of the first inhalation anesthetics used, but its use in anesthesiology has been discontinued; the FDA has banned its use in drugs, cosmetics, and food

packaging. Chloroform is now primarily used in the synthesis of fluorocarbon 22 and other chemicals (Merck 1983, p. 301; Parmeggiani 1983, p. 463).

In addition to cancer, exposure to chloroform causes eye irritation, central nervous system depression, liver and kidney damage, and cardiac effects in humans and animals. The oral LD_{50} in rats is 908 mg/kg, and the LC_{50} in the same species is 75 g/m^3 for 60 minutes (RTECS 1990). Acutely poisoned animals show signs of central nervous system depression, including somnolence and dyspnea; at autopsy, both liver and kidney damage are seen (Gerber and Warner 1983). Rats, rabbits, guinea pigs, and dogs exposed to 25-, 50-, or 80-ppm concentrations of chloroform for 7 hours/day, 5 days/week for 6 months showed, at autopsy, damage to the kidneys and liver (Clayton and Clayton 1982, p. 3464). In contact with the skin of rabbits for 24 hours, chloroform caused redness and a moderate degree of necrosis; the authors of this study reported that chloroform vapor also irritated the eyes of exposed animals (Torkelson, Oyen, and Rowe 1976). In addition, chloroform can be absorbed through the skin in amounts sufficient to cause systemic toxicity (weight loss, degenerative changes in the kidneys, etc.) (Torkelson, Oyen, and Rowe 1976).

In humans, exposure to a chloroform concentration of 10,000 ppm causes clinical anesthesia (Morris 1963). At higher concentrations, cardiovascular depression and ventricular defibrillation follow lethal doses in humans (Morris 1963). Workers exposed to chloroform concentrations of between 80 and 240 ppm during use of this substance as a solvent reported experiencing lassitude, gastrointestinal upsets, and mental dullness; even at a 20- to 70-ppm concentration, these workers reported similar, although milder, reactions (Challen, Hickish, and Bedfor 1958). One-quarter of the workers handling chloroform in a German chemical plant had enlarged livers in clinical examinations; these workers had been occupationally exposed to unspecified concentrations for between 1 and 4 years (Bornski, Sobolewska, and Strakowski 1967).

In the following discussion, the evidence on chloroform's carcinogenicity in humans and animals is described. It is currently believed that the carcinogenicity of chloroform results from the formation of reactive metabolites, such as phosgene, that bind to cellular macromolecules. Although there is some evidence to suggest that chloroform is weakly mutagenic, the results of most mutagenicity tests of this substance are negative.

In humans, there are no epidemiological studies that evaluate populations exposed only to chloroform, although there are several studies that examine populations exposed to chloroform in chlorinated drinking water. However, because chloroform is not the only potential carcinogen present in chlorinated water, OSHA considers the epidemiological data inadequate to use as the basis for a quantitative risk assessment. Thus, a causal relationship between cancer and chloroform exposure cannot be determined based on epidemiological studies alone, although these studies can be used to provide general support for findings in animal studies.

A case-controlled study indicated a significant association between colon cancer and exposure to chlorinated drinking water contaminated with organic material (Young, Kanarek, and Tsiatis 1981/Ex. 1–118). Significant positive associations were also found for chloroform levels in drinking water and the incidence of mortality due to cancer of the bladder, rectum, and large intestine (Hogan, Chi, Hoel, and Mitchell 1979/Ex. 1–159). In addition, similar results have been found by others (Cantor, Hoover, Mason, and McCabe 1978/Ex. 1–50; and Gottlieb, Carr, and Morris 1981/Ex. 1–72). However, although these studies suggest an association between exposure to chloroform and an increased risk of cancer, a definite causal relationship between the development of colon and bladder cancer and exposure to chloroform cannot be determined solely from these studies.

In animals, several long-term studies provide strong evidence for the carcinogenic activity of chloroform. Chloroform has been shown to produce statistically significant increases in renal epithelial tumors in male rats and hepatocellular carcinomas in several strains of mice. The carcinogenic activity of chloroform in these studies is specific to the kidney and liver.

The carcinogenic activity of chloroform was investigated in rats exposed to chloroform by gavage for 78 weeks (NCI 1976a/Ex. 1–119). Male rats were administered doses of 90 or 180 mg/kg/day, and female rats were administered doses of 100 or 200 mg/kg/day. A statistically significant dose-related increase in renal epithelial tumors was observed in treated male rats compared with untreated, matched controls; these tumors were described as carcinomas and adenomas. No increase in the incidence of tumors was observed in chloroform-treated female rats.

In this same study, the carcinogenicity of chloroform was evaluated in mice exposed chronically to chloroform by gavage (NCI 1976a/Ex. 1–119). Male mice were exposed to doses of 138 or 277 mg/kg/day and females to 238 or 477 mg/kg/day for 78 weeks. There were significant dose-related increases in the incidence of hepatocellular carcinomas in chloroform-treated male and female mice. The increase of tumors in male mice for low and high doses was 36 percent and 98 percent, respectively. For female mice, the increases were 80 percent for the low dose and 95 percent for the high dose of chloroform.

The carcinogenic potential of chloroform in mice was further investigated in two other studies (Roe, Palmer, and Worden 1979/Ex. 1–108; Jorgenson, Meierhenry, Rushbrook et al. 1985/Ex. 1–117). Doses of 17, 60, or 100 mg/kg/day were administered to four different strains of male and female mice (C57BL, CBA, CF/1, and ICI) by gavage for 80 weeks (Roe, Palmer, and Worden 1979/Ex. 1–108). The incidence of kidney tumors, described as hypernephromas, was significantly elevated in the ICI strain. Moderate to severe renal changes were observed in the male mice of the other strains, but no significant increase in renal tumors was reported. Tumors were not observed in female mice.

The carcinogenicity of chloroform administered in drinking water was investigated in male rats and female mice (Jorgenson, Meierhenry, Rushbrook et al. 1985/Ex. 1–117). Animals were treated with drinking water containing chloroform concentrations of 200, 400, 900, or 1800 mg/L for 104 weeks. There was a marked increase in the number of kidney tumors (described as tubular cell adenomas and adenocarcinomas) in rats. However, the incidence of tumors in female mice was not significantly increased.

Risk estimate for chloroform. The Jorgenson et al. (1985/Ex. 1–117) rat study, which demonstrated a statistically significant increase in the incidence of renal tumors in male rats, was the data set used as the basis for the quantitative risk estimation. The linearized multistage, one hit, and Weibull models were used. The maximum likelihood estimates of excess cancers over an occupational lifetime for a population of 1,000 workers and the 95-percent upper-bound estimates of these risks are summarized in Table C14–8. The results obtained with the Weibull model are similar to those achieved by using the logit or probit

model. OSHA believes that the estimates based on the use of the multistage model represent the best estimates of risk. OSHA has regularly used this model in previous rulemakings because the model is considered by many to reflect a biologically plausible mechanism of carcinogenesis.

The results of the data analysis presented here are similar to the results of other models described by the EPA (1984f/Ex. 1–216) for chloroform. These three models clearly demonstrate, based on the MLE estimates, that a significant cancer risk exists at the current PEL of 50 ppm. The risks estimated to exist at the current PEL are of the same order of magnitude as the risks associated with exposure to other carcinogens that OSHA has regulated (e.g., benzene, ethylene oxide). Following the approach it has used in other rulemakings, OSHA believes that the MLE estimate derived from the multistage model is the best single estimate of risk.

TABLE C14–8.—MULTISTAGE MODEL ESTIMATES OF CANCER RISK ASSOCIATED WITH WORKING LIFETIME EXPOSURE TO CHLOROFORM

Exposure level	Excess cancer deaths per 1,000 workers	
	MLE	UCL
Multistage:		
2 ppm[a]	0.27	1.80
10 ppm[b]	1.90	8.00
50 ppm[c]	22.40	46.10
One Hit:		
2 ppm[a]	1.40	5.20
10 ppm[b]	7.00	11.10
50 ppm[c]	34.50	54.20
Weibull:		
2 ppm[a]	0.11	0.60
10 ppm[b]	1.60	6.30
50 ppm[c]	24.50	51.30

[a] Proposed PEL in construction, maritime, and agriculture.
[b] ACGIH TLV®.
[c] Current OSHA PEL in construction and maritime.
MLE = Maximum likelihood estimate of risk.
UCL = 95-percent upper-confidence limit on the maximum likelihood estimate of risk.

Based on the evidence presented above, OSHA preliminarily concludes that a significant risk of cancer exists at the current ceiling PEL of 50 ppm, with estimated risks ranging from 22 to 34 excess deaths per 1,000 workers. OSHA also believes that reducing the PEL to 2 ppm will substantially reduce this risk by from 96 to 99 percent, to a residual lifetime risk level of 0.27 to 1.40 deaths per 1,000 exposed workers. Therefore, OSHA is proposing a 2 ppm TWA PEL for chloroform in construction, maritime, and agriculture. Promulgation of this limit will also make OSHA's PEL for this substance consistent across all regulated sectors.

CHROMIC ACID, CHROMATES; ZINC CHROMATES

CAS: Varies with compound; Chemical Formula: Varies with Compound

H.S. No. 1092; 1436

The current OSHA limit for chromic acid and chromates (measured as CrO_3) in the construction and maritime industries is 0.1 mg/m³ as an 8-hour TWA. The Agency has no PEL in agriculture for these substances. The ACGIH has established a TLV®–TWA of 0.05 mg/m³, measured as Cr(VI), for both the soluble and insoluble forms of chromate (except zinc chromate), and has designated insoluble chromates as confirmed human carcinogens (A1). (Note that the 0.05-mg/m³ limit, expressed as Cr(VI), approximates 0.1 mg/m³ expressed as CrO_3.) NIOSH has recommended that exposure to the noncarcinogenic forms of chromium (VI) (which include chromic acid) be limited to 0.025 mg/m³ (measured as CrVI) as a 10-hour TWA and to 0.05 mg/m³ (measured as CrVI) as a 15-minute ceiling. For the carcinogenic (i.e., insoluble) forms of chromium (VI) (which include zinc chromates), NIOSH recommends a 10-hour TWA limit of 0.001 mg/m³ (measured as CrVI). OSHA is proposing a ceiling limit of 0.1 mg/m³ (measured as CrO_3) for chromic acid and chromates, as well as for zinc chromates, in the construction, agriculture, and maritime industries. This is the same PEL as the limit currently in force in general industry.

True chromic acid, H_2CrO_4, exists only in solution form. The chromates are normally crystalline in form, and the majority of these substances are yellowish in color. They have a wide variety of industrial uses, including metal finishing and corrosion control; production of pigments and allied products; use in leather tanning and textiles, wood preservation, and drilling muds; and in chemical synthesis (Grayson 1985, p. 279; Hawley's 1987, pp. 278, 953, 1057, and 1252).

Exposure to chromic acid mists and chromate dust may cause severe irritation of the nose, throat, bronchial tubes, and lungs. Hexavalent chromium compounds have been associated with the development of ulcerated nasal mucosa, perforated nasal septa, rhinitis, nose bleeds, perforated eardrums, pulmonary edema, asthma, kidney damage, erosion and discoloration of the teeth, and skin ulceration. Chromium compounds are known to be allergens, and exposure has been associated with the development of skin sensitization.

Evaluation of workers in the electroplating industry indicates that the current TWA PEL of 0.1 mg/m³ in construction and maritime may not be sufficiently protective against the corrosive and irritating effects of chromates. Franchini et al. (1983, cited in Proctor, Hughes, and Fischman 1988, p. 157) found that workers exposed to chromate concentrations between 0.11 and 0.15 mg/m³ developed ulcers of the nasal septum, asthmatic bronchitis, and mucous membrane irritation. Other studies (Bloomfield and Blum 1928 and U.S. Public Health Service 1953, as cited in ACGIH 1986, p. 139/Ex. 1–3) indicate that concentrations as low as 0.06 mg/m³ are irritating to mucous membranes. The ACGIH TLV® of 0.05 mg/m³ as an 8-hour TWA is based on protecting workers against the corrosive and irritating effects of chromates.

In the rulemaking for general industry, OSHA reviewed some of the data suggesting that exposure to certain insoluble chromate salts is associated with an elevated risk of lung cancer. OSHA determined at that time that the complex issues surrounding this evidence would be more appropriately addressed in a separate comprehensive rulemaking and is considering placing the chromates on its agenda for future rulemaking. Both the AFL–CIO (Ex. 194) and the UAW (Tr. pp. 7–65 to 7–67) agreed that this would be appropriate.

OSHA preliminarily concludes, based on the evidence presented above, that the current 0.1 mg/m³ TWA PEL in construction and maritime, and the absence of any limit for chromates in agriculture, places exposed workers at significant risk of experiencing the corrosive and irritant effects of chromates. OSHA believes that promulgation of a 0.1 mg/m³ ceiling limit will substantially reduce this risk; therefore, the Agency is proposing to apply this limit in construction, maritime, and agriculture. In addition, promulgation of this limit will make OSHA's PEL for these substances consistent across all regulated sectors.

COAL TAR PITCH VOLATILES

CAS No. 65996–32–2

H.S. No. 2039

In 1967, the ACGIH adopted a TLV® of 0.2 mg/m³ for coal tar pitch volatiles (CTPV), measured as the "benzene-soluble" fraction, and listed certain of the carcinogenic components of CTPV. The TLV® was established to assist employers to minimize employee exposures to these listed carcinogenic substances—anthracene, benzo(a)pyrene (BaP), phenanthracene, acridene, chrysene, and pyrene (ACGIH 1986, p. 143). In 1970, when OSHA adopted the ACGIH limits under the Walsh-Healey Act through the OSH

Act's Section 6(a) mechanism, the TLV® for CTPV became the OSHA PEL. This PEL has been in effect in general industry and in the construction and maritime sectors since that time; however, there is no PEL for CTPV in agriculture. At this time, OSHA is proposing an 8-hour time-weighted average PEL of 0.2 mg/m³ for this substance in agricultural workplaces. Promulgation of this limit will make OSHA's PEL for CTPV consistent across all OSHA-regulated sectors.

The principal industrial sources of coal tar are coke-oven plants. The hot gases and vapors produced during the conversion of coal to coke are collected by means of a scrubber, which condenses the effluent into ammonia, water, crude tar, and other byproducts. Crude tar is separated from the remainder of the condensate for further refining and may undergo further processing.

In a study (Aluminum Association, Inc. 1977) of aluminum industry workers, an increase in lung cancer mortality was found in potroom workers using the horizontal Soderberg process. The presence of relatively high amounts of tarry substances and BaP levels in the air of aluminum reduction plants has also been associated with increased lung cancer mortality. Doll et al. (1972) reported high respiratory cancer mortality in coke-oven workers, and Redmond et al. (1976) found that coke-oven workers employed for 5 years or more had a high risk of dying from lung and kidney cancer; non-oven workers had a high risk of developing cancers of the colon, pancreas, buccal cavity, and pharynx, while byproducts workers had no increased risk of dying from any form of cancer (Doll, Vessey, et al. 1972; Reid and Buck 1956). Based on these epidemiologic studies, OSHA preliminarily concludes that occupational exposure to crude coal tar, coal tar pitch, or mixtures containing these substances can cause lung cancer and possibly cancer at a variety of other sites, such as the colon, pancreas, buccal cavity, and pharynx.

Long-term exposure (1–43 years) to coal tar pitch has been reported to cause malignant tumors on the hands, face, and neck of briquette-factory workers (Robillard and Mouchel 1965). However, these investigators did not specify the source or chemical nature of the pitch to which the workers had been exposed. Skin tumors have been reported in many studies (Lane 1937) that involved many different samples of coal tar pitch, leading to the conclusion that coal tar pitch derived from any source should be considered a potent skin tumorigen.

OSHA's preliminary conclusion that exposure to coal tar causes lung cancer is supported by a wealth of animal data, which is discussed in detail by NIOSH (1977).

OSHA preliminarily concludes, based on the epidemiology studies briefly summarized above and on the available animal data, that the absence of any limit for coal tar pitch volatiles in agriculture places exposed workers in this sector at significant risk of developing cancer. In addition, other adverse health effects (photosensitization, respiratory effects, decreased visual acuity) have also been linked to exposure to this substance. OSHA believes that promulgation of a 0.2 mg/m³ 8-hour TWA PEL will substantially reduce this risk, and the Agency is therefore proposing to apply this limit in agriculture. In addition, promulgation of this limit will make OSHA's PEL for CTPV consistent across all regulated sectors.

1,2-DIBROMO-3-CHLOROPROPANE (DBCP)

CAS: 96–12–8; Chemical Formula: $C_3H_5Br_2C1$

H.S. No. 2055

In general industry, construction, and maritime, OSHA's permissible exposure limit for 1,2-dibromo-3-chloropropane (DBCP) is 1 ppb (0.001 ppm) as an 8-hour TWA; this limit also has skin notation, which indicates that percutaneous absorption is a significant route of exposure for DBCP. There is no limit in agriculture. NIOSH has a REL for DBCP of 10 ppb as a 10-hour TWA. OSHA is proposing an 8-hour TWA PEL of 1 ppb (0.001 ppm), and a skin notation, for DBCP in agriculture. Promulgation of this limit will make the PEL for DBCP consistent across all OSHA-regulated sectors.

DBCP, a halogenated hydrocarbon, is produced primarily by the bromination of allyl chloride at room temperature, usually in a vigorous reaction that requires cooling. DBCP is a dense yellow or amber liquid with a pungent odor at high temperatures. It has a low vapor pressure (0.8 mm Hg at 20°C) and is slightly soluble in water (1,000 ppm).

DBCP has been used as an agricultural nematocide since 1955. DBCP is produced in the United States by Dow Chemical Company and Shell Oil company. Mexico, Japan, and Israel also manufacture DBCP and export DBCP to this country. About 12 million pounds of DBCP were consumed in 1972. Following manufacture, DBCP is shipped to formulators who reprocess the chemical into products for consumer emulsifiable concentrates, liquid concentrates, powder, granules, and solid material. Formulating granular DBCP involves spraying liquid DBCP onto inert granules. The formulation of liquid and emulsified DBCP products usually involves the blending of technical grade DBCP with an emulsifier or solvent. The formulators may also distribute the technical grade product.

The complete distribution chain generally includes the manufacture of technical grade DBCP, transportation to the formulator, formulation of DBCP-containing pesticides, distribution of DBCP-containing pesticides, and the agricultural consumption of DBCP pesticides. When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

OSHA has concluded from the evidence in the record (March 17, 1978 final rule on DBCP) that DBCP presents a hazard of cancer and sterility to exposed workers. The results of well-designed animal studies indicate DBCP to be a potent carcinogen in two sexes of two mammalian species at two dose levels. Furthermore, DBCP has been found to cause positive results in microbial assays designed to detect chemicals capable of mutagenesis. This evidence, which was not seriously challenged by any of the participants in the Agency's 1978 proceeding, leads to the conclusion that DBCP must be regulated as a human carcinogen.

Animal studies have also demonstrated that oral doses of DBCP induce degeneration of testicular tissue, accompanied by a reduction of sperm count and abnormal sperm cell development. These testicular effects were confirmed in humans with the discovery of sterility and infertility in a large number of male employees exposed to low levels of DBCP in the manufacture and formulation of pesticides. This evidence was also uncontroverted by hearing participants in the earlier 6(b) rulemaking.

Accordingly, OSHA has concluded that the proven carcinogenic and sterilant potential of DBCP warrants limiting exposure to the lowest level feasible. OSHA has therefore established an eight-hour time-weighted average permissible exposure limit of 1 part per billion (ppb) for this substance. OSHA has concluded, based on evidence presented in the record, that this limit represents the lowest exposure level achievable using present technology.

Based on this evidence, OSHA concluded in 1978 that exposure to DBCP causes cancer and sterility in humans and animals. OSHA therefore

believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed limit of 1 ppb (0.001 ppm) as an 8-hour TWA, and a skin notation, is necessary to significantly reduce these risks of material health impairment among these workers. Promulgation of this limit will make the PEL for this substance consistent across all OSHA-regulated sectors.

DIMETHYL SULFATE

CAS: 77–78–1; Chemical Formula: $(CH_3)_2SO_4$

H.S. No. 1142

OSHA's limit for dimethyl sulfate in the construction and maritime industries is 1 ppm as an 8-hour TWA, with a skin notation. The Agency has no PEL for this substance in agriculture. The ACGIH considers dimethyl sulfate a suspected human carcinogen and has given it an A2 classification. The ACGIH's TLV®–TWA for this substance is 0.1 ppm, with a skin notation. There is no REL for dimethyl sulfate; however, NIOSH (Ex. 8–47, Table N6A) concurred with the selection of this limit. OSHA is proposing a PEL of 0.1 ppm, with a skin notation, as an 8-hour TWA for dimethyl sulfate in the construction, agriculture, and maritime industries. This is the limit recently established in general industry.

Dimethyl sulfate is an oily, colorless liquid with a faint onion-like odor. It is commonly used as a methylating agent in the manufacture of many organic chemicals (ACGIH 1986, p. 212).

In addition to cancer, exposure to dimethyl sulfate causes severe irritation and burns of the eyes, skin, mucous membranes, and lungs in animals and humans. The oral LD_{50} in rats is 205 mg/kg, and the LC_{50} in the same species is 45 mg/m³ for 4 hours RTECS 1990). Mice, rats, and guinea pigs exposed to a 75 ppm concentration of dimethyl sulfate for 17- to 28-minute periods developed pulmonary edema, emphysema, and peribronchitis; autopsy revealed necrosis and fatty infiltration of the liver in these animals (HSDB 1985). Instilled into rabbit eyes and rinsed out almost immediately, dimethyl sulfate nonetheless caused severe irritation; skin contact with the liquid is also severely damaging (RTECS 1990).

Several human fatalities have resulted from industrial accidents involving this substance (Fairhall 1957). Death is caused in these cases by circulatory failure and follows a lengthy interval in which the victim experiences difficult breathing, vomiting, diarrhea, dysuria, and edema of the larynx (Bartalini et al. 1957, Nebelung 1957, Roche et al. 1962). In near-lethal cases, residual effects may persist for years after the incident.

If dimethyl sulfate is splashed into the eye, it causes both acid-type burns and severe, delayed-onset denaturation and necrosis (Grant 1986, p. 350). Contact of the liquid with the skin similarly causes severe necrosis and blistering (HSDB 1985). Dimethyl sulfate is such a severely corrosive substance that it was used as a poison gas during World War I.

The following paragraphs describe dimethyl sulfate's carcinogenicity in animals. The carcinogenic activity of dimethyl sulfate was investigated in male rats chronically exposed to subcutaneous injections of 8 or 16 mg/kg per week (Druckrey, Preussman, Nashed, and Ivanovic 1966/Ex. 1–245). Local sarcomas with metastases to the lung and regional lymph nodes were observed at both dose levels. A single subcutaneous injection of dimethyl sulfate (50 mg/kg) also produced local sarcomas with metastases to the lung (Druckrey, Kruse, Preussman et al. 1970/Ex. 1–246). However, tumors did not develop following chronic weekly intravenous injections of dimethyl sulfate (2 or 4 mg/kg) (Druckrey, Kruse, Preussman et al. 1970/Ex. 1–246). Control data were not reported for either of these studies.

The carcinogenic potential of dimethyl sulfate exposure by inhalation was also evaluated in a bioassay involving male rats (Druckrey, Kruse, Preussman et al. 1970/Ex. 1–246). Animals were exposed to approximately 3 or 10 ppm dimethyl sulfate for 1 hour per day, five times weekly, for 130 days. Malignant tumors developed in 15 percent (3/20) of the rats exposed at 3 ppm and in 18 percent (5/27) of the rats exposed at 10 ppm.

Pregnant rats were exposed to a single intravenous injection of dimethyl sulfate (20 mg/kg body weight) on day 15 of gestation, and the incidence of malignant tumors in the offspring was investigated in litters produced for one year following this exposure; tumors were reported in 7/59 of the offspring. The results of this study are difficult to interpret because several rats in the study died (number of deaths not specified) from the acute toxic effects of dimethyl sulfate, and the incidence of tumors in the control group was not reported.

There is little information available regarding the carcinogenicity of dimethyl sulfate in humans. A case study of workers exposed to dimethyl sulfate reported that three of these workers developed bronchial cancer (Druckrey, Preussman, Nashed, and Ivanovic 1966/Ex. 1–245). However, an epidemiological study by the E.I. du Pont de Nemours Company (1975, as cited in ACGIH 1986/Ex. 1–3, p. 213) demonstrated no increase in the incidence of respiratory cancer among workers exposed to dimethyl sulfate.

In the prior air contaminants rulemaking, OSHA considered the possibility of performing a quantitative risk assessment for dimethyl sulfate and concluded that the studies described above did not have sufficient dose-response data to provide an adequate basis for such a risk assessment (Ex. 85). Dimethyl sulfate induces malignant tumors in animals both by inhalation and ingestion, and there is thus sufficient evidence in animals to predict that workers exposed to dimethyl sulfate are at significant risk of developing cancer; exposures at levels only three times the current PEL (1 ppm) resulted in a significant number of tumors. OSHA also notes that the International Agency for Research on Cancer concluded, on the basis of the evidence described above, that dimethyl sulfate should be considered a carcinogen in animals (IARC 1987, p. 200). Accordingly, OSHA preliminarily concludes that the proposed 0.1-ppm 8-hour TWA PEL, with a skin notation, will substantially reduce the significant risk of cancer mortality associated with exposure to dimethyl sulfate in the construction, agriculture, and maritime industries. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

ETHYLENE OXIDE

CAS: 75–21–8; Chemical Formula: C_2H_4O

H.S. No. 2083

In general industry, construction, and maritime, OSHA's permissible exposure limit for ethylene oxide (EtO) is 1 ppm as an 8-hour TWA and 5 ppm as a 15-minute excursion limit. There is no limit in agriculture. The ACGIH designates EtO as an A2 carcinogen and assigns it an 8-hour TLV®–TWA of 1 ppm. NIOSH considers this substance a potential occupational carcinogen and has a REL of less than 0.1 ppm as an 8-hour TWA and 5 ppm as a 10-minute ceiling. OSHA is proposing an 8-hour TWA of 1 ppm and a 15-minute excursion limit of 5 ppm for EtO in agriculture. Promulgation of these limits will make the PELs for ethylene oxide consistent across all OSHA-regulated sectors.

Ethylene oxide (EtO), also known as 1,2-epoxyethane, oxirane, and dimethylene oxide, is a colorless gas with a characteristic ether-like odor. Although several processes exist for the production of EtO, all United States producers currently manufacture EtO

through the catalytic oxidation of ethylene in the presence of a silver catalyst. EtO is completely miscible with water, alcohol, acetone, benzene, ether, carbon tetrachloride and most organic solvents. It is also highly reactive. In order to reduce explosion hazards when EtO is used as a fumigant or sterilant, it is often used in gaseous mixtures (such as 10% EtO and 90% CO_2 or 12% EtO and 88% halocarbon). Since its first domestic production in 1925, EtO has become a major industrial chemical and is presently one of the 25 highest production volume chemicals in the United States. During the period from 1967 to 1978, for example, the average rate of growth in the EtO industry was 6.7 percent. In 1990, over 5.2 billion pounds of EtO were produced domestically.

The primary use of EtO is as an intermediate in the manufacture of other products. Over 99% of total EtO production is used in the manufacture of other products, and almost 90% is consumed by the EtO manufacturers themselves. On a volume basis, the largest use of EtO is as an intermediate in the production of ethylene glycol, a major component of automotive and other anti-freeze products. Approximately 70 percent of all domestically produced EtO goes into the manufacture of ethylene glycol.

EtO is also widely employed in the production of non-ionic surface-active agents which are used in household detergents and as industrial sufactants. Other products manufactured from EtO include: (1) Ethanolamine, used in sweetening natural gas and in the production of specialty chemicals, detergents and cosmetics; (2) glycol ethers, utilized as a jet fuel additive and in the formulation of coatings, cleaners, automotive brake fluids and inks; (3) diethylene and triethylene glycol, used in drying natural gas and in the manufacture of polyester resins, emulsifiers, lubricants and plasticizer; (4) tetraethylene glycol, utilized to extract aromatic hydrocarbons from nonaromatic hydrocarbons; (5) polyethylene glycol, from which cosmetics, plasticizer, lubricants and dispersants are produced; (6) polyethylene glycol, used for water soluble packaging and for warp sizing, and (7) crown ethers, used for extraction of liquids.

A small fraction of EtO production (less than 0.5 percent) is consumed by sterilant or fumigant users. EtO is utilized as a sterilizing agent by various facets of the health care industry for the sterilization of delicate instruments and heat- or moisture-sensitive devices, and is employed as a fumigant for a number of miscellaneous items, such as spices, black walnut meats, books, furniture, textiles, empty bin equipment, empty cargo holds, cosmetics, and dairy packaging. When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

OSHA has found that EtO can cause several serious adverse health effects. Studies in experimental animals supported by epidemiological studies of working populations indicate that EtO is a potential occupational carcinogen. The evidence suggests that EtO may cause cancers of the blood (leukemia), as well as other organs in humans. In addition, EtO exposure cause mutations, increases the rate of chromosomal aberration and sister chromatid exchange, and causes other undesirable changes in the DNA of mammalian cells. These effects support OSHA's conclusion regarding the carcinogenicity of EtO. EtO exposure has also been associated with an increased risk of spontaneous abortion among pregnant women and is capable of causing other adverse reproductive effects in both men and women. Exposure to high concentrations of EtO causes central nervous system depression and other neurological effects that are thought to be reversible with cessation of exposure. In addition, exposure to EtO gas causes sensitization and irritation of human tissues, including the eyes and respiratory tract.

Three epidemiological studies indicate an association between worker exposure to EtO and a significant increase in the risk of death from cancer. Hogstedt et al. (Ex. 208) found an increased risk of death from leukemia among employees exposed to EtO when this substance was used as a sterilant. In a second study, these investigators confirmed an increased leukemia risk and also observed a significant excess of stomach cancer deaths and total cancer deaths among production workers (Ex. 2–22). Morgan et al. (Ex. 6–5) found an increased risk of mortality from Hodgkin's disease and pancreatic cancer among EtO production workers.

Studies in experimental animals have provided definitive evidence that EtO is carcinogenic in multiple species and by several routes of administration. Leukemia, brain cancer, and mesothelioma have been induced in animals exposed to EtO by inhalation. Cancers of the forestomach have been induced as a result of EtO administration by oral gavage. Injection site sarcomas and skin cancers have been observed in animals exposed to EtO by injection.

Additional evidence regarding the adverse health effects of EtO on both humans and animals was presented in the preamble to OSHA's Final Rule on Occupational Exposure to Ethylene Oxide (FR June 22, 1984).

In the absence of a limit for EtO in agriculture, OSHA preliminarily concludes that exposed workers are at significant risk of developing leukemia and other cancers, as well as neurologic effects. OSHA believes that promulgation of the 1 ppm 8-hour TWA PEL and the 5 ppm 15-minute excursion limit is necessary to reduce this risk. Therefore, OSHA is proposing to apply these limits to agriculture. In addition, promulgation of these limits will make OSHA's PELs for EtO consistent across all regulated sectors.

FORMALDEHYDE

CAS: 30–00–0; Chemical Formula: CH_2O

H.S. No. 2085

In general industry, construction, and maritime, OSHA's permissible exposure limits for formaldehyde are 1 ppm as an 8-hour TWA and 2 ppm as a 15-minute STEL. There is no limit in agriculture. The ACGIH designates this substance an A2 carcinogen and assigns it an 8-hour TLV®–TWA of 1 ppm and a 15-minute STEL of 2 ppm. NIOSH considers formaldehyde a potential occupational carcinogen and has a REL for formaldehyde of 0.016 ppm as an 8-hour TWA and 0.1ppm as a 15-minute ceiling. OSHA is proposing a PEL of 1 ppm as an 8-hour TWA and 2 ppm as a 15-minute STEL for formaldehyde in agriculture. Promulgation of these limits will make the PELs for formaldehyde consistent across all OSHA-regulated sectors.

The chemical called "formaldehyde" is a colorless, pungent gas at room temperature that has an approximate odor threshold of about 1 ppm. The simplest member of the aldehyde class of chemicals, formaldehyde has a molecular weight of 30. Although the term "formaldehyde" is also used to describe various mixtures of formaldehyde, water, and alcohol, the term "formalin" more precisely describes aqueous solutions, particularly those containing 37 to 50 percent formaldehyde and 6 to 15 percent alcohol stabilizer. Most formaldehyde enters commerce as formalin. Alcoholic solutions of formaldehyde are available for processes that require low water content (Ex. 753–53). Para-formaldehyde, a solid, also serves as a source of formaldehyde

gas. Formaldehyde gas, per se, is not available commercially.

Formaldehyde is a major industrial chemical, ranked 24th in production volume in the United States (Ex. 138–F). In 1985, 5.7 billion pounds of 37 percent formaldehyde (by weight) was produced. Formaldehyde is produced in 49 plants in the U.S. by one of two processes: the mixed oxide catalyst process or the silver oxide catalyst process. Both processes use methanol as the product precursor and they differ in catalyst type, operating temperature, and methanol/air ratios.

About two-thirds of annual production is used on-site or at nearby locations. For example, a major use of formaldehyde is in the preparation of adhesive resins for use in the wood products industry. These resin-producing facilities tend to be located in lumber producing areas of the South and West.

Formaldehyde has a high degree of chemical reactivity because of its unique chemical structure, i.e., a carbonyl group attached to hydrogen atoms. As a result it undergoes a wide variety of chemical reactions in three distinct classes: oxidation-reduction reactions, addition or condensation reactions, and self-polymerization.

Formaldehyde has four basic uses: as an intermediate in the production of resins; as an intermediate in the production of industrial chemicals; as a bactericide or fungicide; and as a component in the formulation of end-use consumer items. The manufacture of three types of resins: Urea-formaldehyde, phenol-formaldehyde and melamine formaldehyde, accounts for about 59 percent of total consumption. An additional seven percent is consumed in the production of thermoplastic acetal resins. About one-third is used in the synthesis of high-volume chemical derivatives, including pentaerythritol, hexamethylenetetramine, and butane. Two percent is used in textile treating and amounts of formaldehyde are present as preservatives or bactericides in consumer and industrial products, such as cosmetics, shampoos, and glues. When used in pesticidal applications and as directed on the label, this substance is regulated by the EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

Workers have developed various health effects from inhalation of airborne formaldehyde and from dermal contact with formaldehyde solutions or products containing formaldehyde. At airborne concentrations as low as 0.1 ppm, formaldehyde causes irritation of the eyes, nose, and throat. As airborne concentrations increase, the severity of the irritation increases, and irritation spreads from the nasal passages and throat into the lower respiratory system. Very severe conditions, e.g., exposures above 50 ppm, have caused severe lacrimation (tearing of the eyes) and pulmonary reactions, including pneumonia, bronchial inflammation, and pulmonary edema. Massive inhalation of formaldehyde has caused accidental death in humans. A concentration of 100 ppm is "immediately dangerous to life and health" (IDLH) and is believed to be a potentially fatal level if exposure continues for 30 minutes or more.

Long-term inhalation of formaldehyde gas is associated with nasal cancer in experimental animals. Some studies in humans exposed to formaldehyde have demonstrated increased nasal and nasophrygeal cancer. Thus, formaldehyde should be regarded as an occupational carcinogen.

Skin irritation is a well-known consequence of dermal contact with formaldehyde. Formaldehyde reacts with skin, resulting in a response not unlike the effects seen in the tanning of leather. Employees such as pathologists or undertakers, who handle embalming fluids or other concentrated solutions of formalin, frequently show evidence of skin disease. Dermal contact with formaldehyde-bearing resins has also caused skin disorders (see the NIOSH Health Hazard Evaluations, in Exs.78 and 85, for example).

Dermal sensitization to formaldehyde is a well-known phenomenon. Among atopic individuals seeking assistance at allergy clinics, formaldehyde is among the top 10 sensitizers found in patch testing results. A person sensitized to formaldehyde would have serious difficulties working in industries, such as garment manufacturing or textile finishing, where there is repeated dermal contact with sensitizing materials. Sensitization is not readily reversed, which means that a worker who is sensitized may be unable to continue his or her line of work.

OSHA estimated in the 6(b) rulemaking for formaldehyde that the maximum likelihood estimate (MLE) cancer risk for employees exposed for a lifetime to formaldehyde is 0.6 deaths per 1,000 workers at 1 ppm. This risk estimate and estimates of this risk at other levels were based on the rat study conducted by the CIIT. Information regarding the risk assessment and the health effects of formaldehyde is extensive, and was discussed at length in the preamble to OSHA's Final Rule on Occupational Exposure to Formaldehyde (F.R. publication, December 4, 1987). References cited in this discussion can be found in that preamble.

Based on this evidence in humans and animals, OSHA concluded in that rule that exposure to formaldehyde causes cancer sensitization, respiratory tract irritation, and other systemic effects in humans and animals. OSHA therefore believes that; in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed limits of 1 ppm as an 8-hour TWA and 2 ppm as a 15-minute STEL are necessary to significantly reduce these risks of material health impairment among these workers. Promulgation of these limits will also make the PELs for formaldehyde consistent across all OSHA-regulated sectors.

2–NITROPROPANE

CAS: 79–46–9; Chemical Formula: $CH_3CH(NO_2)CH_3$

H.S. No. 1291

OSHA's limit for 2-nitropropane (2–NP) in the construction and maritime industries is 25 ppm as an 8-hour TWA. The Agency has no PEL for this substance in agriculture. The ACGIH classifies 2-nitropropane as a suspected human carcinogen (A2) and has assigned it a TLV®–TWA of 10 ppm. NIOSH considers 2-nitropropane a potential human carcinogen and recommends that exposure be reduced to the lowest feasible limit. OSHA is proposing a 10 ppm 8-hour TWA PEL for 2-nitropropane in the construction, agriculture, and maritime industries. This is the limit recently established for this substance in general industry.

2-Nitropropane is a colorless liquid. It is used as a chemical intermediate, a solvent, and a component in paint, ink, and varnishes (Fiala, Czenniak, Castonguay et al. 1987/Ex. 1–248).

In addition to cancer, exposure to 2–NP causes pulmonary irritation and, in animals, liver damage. The oral LD_{50} in rats is 720 mg/kg, and the LC_{50} in the same species is 400 ppm for 4 hours (RTECS 1990). Acutely poisoned animals developed cyanosis, convulsed, and became comatose before death (Clayton and Clayton 1982, p. 4159). Pulmonary edema and hemorrhage and damage to the liver were seen in these animals at autopsy (Clayton and Clayton 1982, p. 4159). Cats, rabbits, guinea pigs, and monkeys were exposed to 83 or 328 ppm for 7 hours/day; cats succumbed after a few days of such exposure, but the other animals survived 130 (guinea pigs, rats, and rabbits) or 100 (monkeys) of these exposures (Clayton and Clayton 1982, p. 4159).

Acute effects have been reported in workers exposed either to 2–NP alone or in combination with other substances. A concentration of 20 to 45 ppm xylene and 2–NP caused gastrointestinal symptoms, headache, and anorexia in exposed workers (Skinner 1947). Irritation of the respiratory tract was reported by workers exposed to 2–NP at concentrations ranging from 30 to 300 ppm (AIHA 1978). The following paragraphs describe the carcinogenicity of 2-nitropropane in experimental animals.

The mechanisms of carcinogenicity of 2–NP are thought to involve the release of nitrite and the formation of a reactive azoxy intermediate that can react with cellular macromolecules (Williams and Weisburger 1986/Ex. 1–65). In mutagenicity tests, 2–NP increased the frequency of mutations in all strains of *Salmonella typhimurium* with and without metabolic activation. Positive mutagenicity results were reported in *Salmonella typhimurium* strains TA100, TA1535, and TA98 by Lofroth, Nilsson, and Anderson (1981) and by Speck, Meyer, Zeiger, and Rosenkranz (1982/Ex. 1–290).

The available epidemiology data on the chronic health effects of occupational exposure to 2–NP do not contain sufficient dose-response data to use as a basis for quantitative risk estimation. An unpublished retrospective mortality study of 1,481 potentially exposed workers from a nitropropane production plant found no increase in liver cancer or liver disease mortality. However, lack of exposure data, the small number of workers with long exposures (greater than 15 years), and a short latency period make interpretation of the results of this study difficult (Miller and Temple 1979; Bolender 1983).

There are two studies that report high incidences of liver tumors in male rats exposed to 2–NP by gavage and inhalation. Fiala et al. (1987/Ex. 1–248) administered, by gavage, 1mmol/kg body weight (approximately 27 mg per treatment per 300-gram rat) of 2–NP in a 10-percent aqueous Emulphor EL–620 vehicle to male Sprague-Dawley rats three times weekly for 16 weeks. Dosing was discontinued after 16 weeks because of excessive mortality in the treated rats. Seventy-seven weeks from the first treatment, the surviving rats were sacrificed and subjected to necropsy. All (100 percent) of the treated rats examined had developed hepatocarcinomas (Fiala, Czenniak, Castonguay et al. 1987/Ex. 1–248).

The results of the Fiala et al. (1987/Ex. 1–248) study support the earlier positive results reported by Lewis, Ulrich, and Busey (1979/Ex. 1–826). In the Lewis et al. (1979/Ex. 1–826) study, male Sprague-Dawley rats and male New Zealand White rabbits were exposed via inhalation to 27 ppm or 207 ppm of 2–NP for 7 hours/day, 5 days/week for 6 months. At the end of 6 months, all 10 rats in the high-dose group exhibited hepatocellular carcinomas and neoplastic nodules. No exposure-related lesions were seen in the rats exposed to 27 ppm, and no exposure-related lesions were observed in any of the rabbits.

One high-dose and two low-dose studies reported negative results for rats exposed to 2–NP vapors. Griffin, Benitz, Coulston, and Rosenblum (1978/Ex. 1–243) reported no hepatic carcinomas in male and female rats exposed to 200 ppm of 2–NP by inhalation using a protocol similar to that described by Lewis et al. (1979/Ex. 1–826). Although no hepatic carcinomas were observed, the following effects (generally occurring more extensively in males) were seen: Increased liver weights (both sexes); hepatic nodules; hepatocellular necrosis; and peripheral compression.

Two low-dose studies (Griffin, Coulston, and Stein 1980/Ex. 1–268; Griffin, Stein, and Coulston 1981/Ex. 1–279) also produced negative results. Male and female Sprague-Dawley rats were exposed by inhalation to 25 ppm of 2–NP for seven hours/day, five days/week for 22 months. No pathological changes associated with exposure to 2–NP were seen.

Although the results of both the Lewis et al. (1979/Ex. 1–826) and the Fiala et al. (1987/Ex. 1–248) studies show statistically significant increases in liver carcinomas, neither study provides sufficient dose-response information to use as a basis to quantify the excess cancer risk to humans exposed to 2–NP. Both studies were terminated before the natural lifetime expectancy of the controls, so it is not possible to determine a background incidence of cancer risk. No historical information is provided on tumor incidence for these animals.

2-Nitropropane produced a high incidence of liver tumors in male rats by two routes of administration: inhalation and ingestion. Its ability to cause mutations in *Salmonella typhimurium* further supports the premise that 2–NP is a potential human carcinogen. In the prior rulemaking, OSHA considered whether to perform a quantitative risk assessment on 2–NP and concluded that the studies described above do not contain sufficient dose-response data to use as the basis for quantitative risk estimation using standardized risk assessment models. However, two studies (Fiala, Czenniak, Castonguay et al. 1987/Ex. 1–248; Lewis, Ulrich, and Busey 1979/Ex. 1–826) demonstrate that exposure to 2–NP, either by gavage or inhalation, produced hepatocarcinomas in rats. In addition, this substance produced positive results in two mutagenic assays (Lofroth, Nilsson, and Andersson 1981; Speck, Meyer, Zeiger, and Rosenkranz 1982/Ex. 1–290).

Based on this evidence, OSHA is proposing an 8-hour TWA PEL for 2–NP of 10 ppm in the construction, agriculture, and maritime industries. The Agency preliminarily concludes that a reduction in the PEL is necessary to protect exposed workers in these sectors from the significant risk of cancer potentially associated with exposure to 2–NP at the former PEL. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

PERCHLOROETHYLENE (TETRACHLOROETHYLENE)

CAS: 127–18–4; Chemical Formula: $CCl_2{=}CCl_2$

H.S. No. 1308

OSHA's permissible exposure limit for perchloroethylene (tetrachloroethylene) in the construction and maritime industries is 100 ppm as an 8-hour TWA. The Agency has no PEL in agriculture for this substance. The ACGIH TLV®s are 50 ppm as a TWA and 200 ppm as a STEL. NIOSH considers perchloroethylene a potential human carcinogen and recommends minimizing workplace exposure concentrations to the lowest levels feasible. After reviewing extensive comments in the rulemaking for general industry, OSHA concluded that perchloroethylene is a potential human carcinogen and established a 25 ppm PEL as the lowest level for which the Agency had evidence of feasibility. OSHA is proposing this PEL of 25 ppm as an 8-hour TWA for perchloroethylene in the construction, agriculture, and maritime industries. This is the limit recently established in general industry.

Perchloroethylene is a clear, colorless, nonflammable liquid with an ethereal odor. It is widely used as a solvent in the dry cleaning industry and in industrial degreasing operations.

The oral LD_{50}s of perchloroethylene in rats and mice are 8850 and 8100 mg/kg, respectively. The narcotic effects associated with acute exposure to perchloroethylene are well documented. Four human subjects exposed to a 5000-ppm concentration for 6 minutes experienced vertigo, nausea, and mental confusion for 10 minutes. Mild central nervous system depression results from exposure to 2000 ppm for 5 minutes, and

exposure to 600 ppm for 10 minutes is associated with dizziness, numbness around the mouth, and incoordination. Exposure to 100 ppm for 7 hours has resulted in mild irritation of the nasal passages, eyes, and throat; headache; facial flushing; and slurred speech (Proctor, Hughes, and Fischman, 1988, pp. 399–400). The most comprehensive studies of the effects of prolonged exposure to perchloroethylene vapors on human volunteers were conducted by Stewart and colleagues (Stewart, Hake, LeBrun et al. 1974/Ex. 1–970; Stewart, Hake, Wu et al. 1977/Ex. 1–971); these investigators concluded that prolonged exposure to 200 ppm results in early signs of CNS depression, while no response was elicited in men or women exposed repeatedly to 100 ppm for seven hours/day, except that performance on the Flanagan coordination test was significantly decreased in some exposed subjects (Stewart, Hake, Wu et al. 1977/Ex. 1–971, p. 28).

Chronic exposure to perchloroethylene has been associated with neurological abnormalities, including impaired memory, numbness of the extremities, peripheral neuropathy, and impaired vision. Twenty-one of 40 dry-cleaning workers exposed to perchloroethylene concentrations up to 300 ppm showed abnormalities of the autonomic nervous system. Twenty dry-cleaning workers exposed for an average of 7.5 years to concentrations ranging from 1 to 40 ppm had altered electrodiagnostic and neural rating scores (Proctor, Hughes, and Fischman, 1988, pp. 399–400). In addition to examining the evidence for the chemical's narcotic effects, OSHA reviewed a number of studies on the carcinogenicity of perchloroethylene. These investigations are summarized below.

In a 1977 gavage bioassay for carcinogenicity, perchloroethylene was shown to be a liver carcinogen in mice but not in rats (NCI 1977c, as cited in ACGIH 1986/Ex. 1–3, p. 464). In 1986, the NTP conducted an inhalation bioassay of perchloroethylene (NTP 1986b/Ex. 8–31, Appendix 4), in which groups of 50 male and 50 female F344/N rats and B6C3F1 mice were exposed to perchloroethylene for 6 hours/day, 5 days/week, for 2 years. The exposure concentrations were 0, 200, or 400 ppm for rats and 0, 100, or 200 ppm for mice. Male and female rats exposed to either 200 or 400 ppm developed statistically significant increases in mononuclear cell leukemias. According to the NTP report (NTP 1986b/Ex. 8–31, Appendix 4), the increased incidences of leukemias were responsible for the early deaths observed in male and female rats exposed to perchloroethylene. At autopsy, most of the leukemias were determined to be in an advanced and probably fatal stage. Because of the effect of the leukemias on the early mortality of the exposed rats, a life-table analysis was used to test for the statistical significance of the findings; this analysis revealed that the increased incidence of leukemia was statistically significant in both low- and high-dose male rats and in low-dose female rats, and was marginally significant ($p = 0.053$) in high-dose female rats.

Male rats also developed a significant increase in renal tubular cell adenomas and carcinomas. Perchloroethylene induced a significantly increased incidence of hepatocellular carcinomas at both dose levels in mice of both sexes. The NTP Peer Review Panel concluded that there was "clear evidence of carcinogenicity of tetrachloroethylene" (perchloroethylene) in male rats and in male and female mice, and "some evidence" in female rats (Ex. 8–31, Appendix 4).

In addition, a number of human studies were submitted to the general industry rulemaking record that implicate perchloroethylene as a potential carcinogen (Ex. 8–31). Among these was a study by Brown and Kaplan (1987/Ex. 8–31, Appendix 6), which reported a statistically significant elevation in urinary tract cancer deaths among 1,690 dry cleaning workers exposed to perchloroethylene and other petroleum solvents. However, a subcohort of workers who used perchloroethylene as the primary solvent showed no increase in bladder cancer mortality. Brown and Kaplan concluded that "confounding exposure to petroleum solvents complicates any conclusions regarding the association between * * * [perchloroethylene] and cancer of the urinary tract" (Brown and Kaplan 1987/Ex. 8–31, Appendix 6, p. 540).

Katz and Jowett (1981/Ex. 8–31, Appendix 9) studied the mortality pattern of 671 female dry cleaning workers for the period 1963 through 1977. Elevated incidences of cancers of the kidney and genitals were reported, along with a smaller excess of bladder and skin cancers and lymphosarcomas. The authors concluded that, although results obtained with the methodology used (proportionate mortality ratios) require careful interpretation, "this study raises the possibility that exposure to dry cleaning fluids may increase the risk of certain cancers" (Katz and Jowett 1981/Ex. 8–31, Appendix 9, p. 510). The dry cleaning fluids used by members of the cohort included carbon tetrachloride, trichloroethylene, and perchloroethylene.

Steinhagen et al. (1983/Ex. 8–31, Appendix 8) reported a significant excess of liver cancer among male workers in the laundry and dry cleaning industry in New Jersey. This study was a retrospective case-control study. The liver cancer cases were concentrated among individuals who processed clothes and were exposed to chemicals. The report did not identify the solvents in use (Steinhagen, Slade, Altman, and Bill 1983/Ex. 8–31, Appendix 8).

Duh and Asal (1984/Ex. 8–31, Appendix 7) examined the mortality experience of 440 dry cleaning workers in Oklahoma for the period 1975 through 1981. Elevated standardized mortality odds ratios (SMORs) were found for both lung cancer (SMOR=1.7) and kidney cancer (SMOR=3.8) (Duh and Asal 1984/Ex. 8–31, Appendix 7).

During the general industry rulemaking, the Amalgamated Clothing and Textile Workers Union (ACTWU) submitted a quantitative risk assessment conducted by Dale Hattis of the Center for Technology Policy and Industrial Development at the Massachusetts Institute of Technology (Hattis 1986/Ex. 8–31, Appendix 11–A). This work was conducted in 1986 for the National Institute for Environmental Health Sciences. Hattis used a pharmacokinetic model that incorporated species-specific rates of formation for the metabolites of perchloroethylene. Using the rat leukemia and mouse liver tumor data from the NTP (1986b/Ex. 8–31, Appendix 4) bioassay, Hattis obtained a "best estimate" of the lifetime cancer risk (for workers exposed at the former 100-ppm OSHA limit for 45 years to perchloroethylene) of 45 deaths per 1,000 workers. The best-estimate lifetime risks associated with 45 years of exposure to 50 and 10 ppm of perchloroethylene were 27 and 6.4 deaths per 1,000 workers, respectively.

In its posthearing comments to the general industry rulemaking record, the Halogenated Solvents Industry Alliance (HSIA) (Ex. 186) discussed several aspects of the available data to support its contention that perchloroethylene should not be considered a probable human carcinogen. Specifically, the HSIA raised questions concerning the interpretation of epidemiologic data and the NCI gavage and inhalation studies. OSHA addressed each of these points in the preamble to the final general industry rule (54 FR 2686–2689) and concluded that the evidence for the

carcinogenicity of perchloroethylene "is convincing." Based predominantly on the animal data, NIOSH has also concluded that perchloroethylene is a potential human carcinogen; NIOSH judged the evidence for perchloroethylene's carcinogenicity sufficient to warrant a separate 6(b) rulemaking (Ex. 8–47, Table N6B). In 1987, the International Agency for Research on Cancer (IARC) also classified perchloroethylene as a Category 2B carcinogen (i.e., a substance for which the evidence in animals is sufficient). The EPA's SAB has determined that perchloro-ethylene is a Category C carcinogen (i.e., a possible human carcinogen, and a carcinogen in animals). In addition, a number of human studies suggest elevated cancer risks, particularly of the kidney and bladder, among workers exposed to perchloroethylene and other solvents in dry cleaning facilities.

Based on a review of all of the available evidence on perchloroethylene, including the testimony and briefs submitted by the parties during the rulemaking for general industry, OSHA concluded that perchloroethylene is a potential human carcinogen that presents a significant risk of material health impairment to workers exposed to it in their places of work. The Agency consequently set 25 ppm as the lowest limit for which there was evidence for feasibility. That standard is now in effect for general industry. The IFI and HSIA have challenged the 25 ppm PEL in court as being too low, and the AFL–CIO has challenged on the grounds that it is too high. The matter is now pending before the Eleventh Circuit Court of Appeals.

The risk assessment conducted by Hattis (1986/Ex. 8–31, Appendix 11–A) estimates that there is an excess lifetime cancer mortality risk of 45 deaths per 1,000 workers exposed for 45 years to the current 100-ppm TWA PEL. At the level of 50 ppm, Dr. Hattis estimated the excess lifetime risk to be 27 deaths per 1,000 workers. OSHA preliminarily concludes that this assessment and the underlying evidence indicate that a further reduction in the PEL is necessary. In the general industry rulemaking OSHA concluded that 25 ppm was the lowest level demonstrated by the available evidence to be feasible.

OSHA is proposing an 8-hour TWA PEL of 25 ppm for perchloroethylene in the construction, agriculture, and maritime industries. OSHA preliminarily concludes that this PEL will substantially reduce the significant risk of material impairment of health presented by exposure to this substance. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

o-TOLUIDINE

CAS: 95–53–4; Chemical Formula: $CH_3C_6H_4NH_2$

H.S. No. 1399

OSHA's 8-hour TWA limit for o-toluidine in general industry and in the construction and maritime industries is 5 ppm. The Agency has no PEL in agriculture for this substance. The ACGIH identifies o-toluidine as a suspected human carcinogen (A2) and has assigned it a TLV®–TWA of 2 ppm, with a skin notation. NIOSH has no REL for o-toluidine but concurs (Ex. 8–47, Table N6A) with the limit being proposed. The International Agency for Research on Cancer (IARC 1982b, as cited in ACGIH 1986/Ex. 1–3, p. 586) classifies o-toluidine as a probable carcinogen (category 2A) based on sufficient evidence of its carcinogenicity in rats and mice following oral administration (IARC 1982b, as cited in ACGIH 1986/Ex. 1–3, p. 586). IARC judged the evidence inadequate to establish o-toluidine's carcinogenicity in humans. The Agency is proposing that its current PEL of 5 ppm as a TWA now be applied to the agriculture industry in addition to the construction and maritime industries and that a skin notation be added. This limit, and the skin notation, were recently reaffirmed for general industry.

o-Toluidine is a light yellow liquid that rapidly darkens on exposure to air and light. It is used in textile printing dyes, as a vulcanization accelerator, and in organic synthesis (Hawley's 1987, p. 1165).

o-Toluidine is of moderate acute toxicity; the oral LD_{50}s in rats and mice are 670 mg/kg and 520 mg/kg, respectively. The dermal LD_{50} in rabbits is 3250 mg/kg (RTECS 1990). Acute exposure to o-toluidine causes methemoglobinemia and transient hematuria. The earliest manifestations of exposure are headache and cyanosis of the lips, mucous membranes, fingernail beds, and tongue; with continued exposure, symptoms include weakness and dizziness leading to stupor, unconsciousness, and death. Exposure to a 10-ppm concentration of o-toluidine for a short period of time may lead to symptoms of illness, and exposure to 40 ppm for an hour or more can cause severe toxic effects. Methemoglobinemia has been produced in experimental animals exposed to 6 to 23 ppm for several hours (Proctor, Hughes, and Fischman 1988, pp. 481–482).

o-Toluidine is mutagenic in short-term tests, inducing sister chromatid exchanges and unscheduled DNA synthesis in mammalian cells *in vitro* and chromosomal anomalies in yeast. o-Toluidine was negative in the micronucleus test in mice *in vivo*, but induced cell transformations in the BHK (baby hamster kidney) assay. IARC considers these data to be sufficient evidence of o-toluidine's activity in short-term tests (IARC 1982b).

There are a number of studies that report an excess of bladder tumors in dyestuff workers exposed to o-toluidine and other chemicals; however, there are no studies that examine a population of workers exposed to o-toluidine alone. Workers exposed to toluene, o-nitrotoluene, o-toluidine, and 4,4-methylene bis (2-methylaniline) in manufacturing were observed to have an excess of bladder tumors. However, the concurrent exposures of these workers to these other potential carcinogens make these data inappropriate for use in the quantitative assessment of o-toluidine's carcinogenic risk in human populations. A few reports of bladder tumors in persons exposed primarily to o-toluidine have been reported, but insufficient follow-up time and incomplete data have prevented the establishment of a clear quantitative association between o-toluidine exposure and cancer in humans. For this reason, IARC considers the data from human studies inadequate to establish an association between exposure to o-toluidine and cancer (IARC 1982b).

Several studies have been conducted that implicate o-toluidine as a potential carcinogen. In rats, statistically significant increases in subcutaneous fibromas, fibrosarcomas, and cancers of the urinary bladder have been reported. Studies in mice have resulted in statistically significant increases in hemangiosarcomas and hepatocellular carcinomas. For example, the National Cancer Institute (NCI 1979c, as cited in ACGIH 1986/Ex. 1–3, p. 586) conducted long-term carcinogenicity studies with o-toluidine in rats and mice. Both of these studies were positive for carcinogenicity. The mouse study used groups of 50 female and 50 male B6C3F1 mice fed o-toluidine hydrochloride in the diet at levels of 1000 ppm or 3000 ppm for 102 to 103 weeks. There was no excess mortality in the tested animals. At the 3000-ppm dose level, there was a statistically significant increase in hemangiosarcomas at all sites in males and a statistically significant increase in hepatocellular carcinomas and adenomas in females.

The National Cancer Institute also conducted a 2-year feeding study with 50 male and 50 female Fischer 344 rats. There was a dose-related trend in mortality (which was not caused by cancer); all the males in the high-dose group died by week 100. However, the females at both dose levels were observed to have significant increases in transitional-cell carcinomas or papillomas of the urinary bladder, and the high-dose females developed fibroadenomas of the mammary gland. The males at both dose levels showed significant increases in fibromas of the subcutaneous tissue and mesotheliomas in multiple organs (NCI 1979c, as cited in ACGIH 1986/Ex. 1–3, p. 586). The high mortality in the males complicates the interpretation of these latter findings.

Weisburger, Russfield, Homburger et al. (1978/Ex. 1–535) reported positive findings for o-toluidine in long-term feeding studies in rats and mice. The study in rats was conducted with two groups of 25 male CD rats fed o-toluidine in the diet on one of two regimens: 8000 ppm for three months and then 4000 ppm for an additional 15 months, or 16,000 ppm for three months and then 8000 ppm for an additional 15 months. Statistically significant increases in the incidence of subcutaneous fibromas and fibrosarcomas were observed in both dose groups. In addition, there was a nonstatistically significant increase in the incidence of transitional-cell carcinomas of the urinary bladder in these animals.

Weisburger, Russfield, Homburger et al. (1978/Ex. 1–535) also reported the results of a long-term study in mice. Groups of 25 male and 25 female CD–1 mice were fed diets containing o-toluidine at two dose levels: 16,000 ppm for three months and then 8000 ppm for an additional 15 months, or 32,000 ppm for three months and then 8000 ppm for an additional 15 months. There was a statistically significant, dose-related increase in the incidences of vascular tumors (hemangiosarcomas and hemangiomas of the abdominal viscera) in both sexes of treated mice, compared with results in control mice.

Risk estimate for o-toluidine. Four of these carcinogenicity studies of o-toluidine have yielded sufficient and adequate data for quantitative risk estimation: The two NCI studies (NCI 1979c, as cited in ACGIH 1986/Ex. 1–3, p. 586) and the two Weisburger et al. (1978/Ex. 1–535) studies. OSHA used the NCI (1979c) study in rats as the basis for its quantitative risk assessment because it provides the most appropriate data. Table C14–9 presents the Maximum Likelihood Estimates (MLEs) of excess deaths per 1,000 employees predicted to result from exposure to o-toluidine at the OSHA PEL of 5 ppm and at higher levels to illustrate the potential impact on risk of extending the 5 ppm PEL to agriculture. These data were calculated using a multistage model, GLOBAL83.

TABLE C14–9.—MULTISTAGE MODEL ESTIMATES OF CANCER RISK ASSOCIATED WITH WORKING LIFETIME EXPOSURE TO O-TOLUIDINE

Exposure Level	Excess cancer deaths per 1,000 workers	
	MLE	UCL
50 ppm	1.48	16.0
25 ppm	0.69	8.0
5 ppm	0.14	1.60

MLE = Maximum likelihood estimate of risk.
UCL = Upper-bound (95-percent) confidence limit on maximum likelihood estimate of risk.

Table C14–9 shows an excess MLE estimate of risk of 0.14 per 1,000 workers exposed over their working lifetimes at a PEL of 5 ppm; in contrast, lifetime exposure to 50 ppm is associated with estimated mortality risk levels exceeding 1 in 1,000 workers. Accordingly, OSHA has preliminarily concluded that it is appropriate to retain the existing 5-ppm TWA PEL and to add the skin notation for o-toluidine in the construction and maritime industries and to propose the PEL and skin notation for the agriculture industry as well; adoption of a 5 ppm limit in agriculture is necessary to substantially reduce the significant risk of material impairment of health that would exist in the absence of a limit. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

p-TOLUIDINE

CAS: 106–49–0; Chemical Formula: $CH_3C_6H_4NH_2$

H.S. No. 1400

OSHA has no PEL for p-toluidine in the construction, agriculture, and maritime industries. The ACGIH considers this substance a suspected human carcinogen (A2) and has assigned a TLV®–TWA of 2 ppm, and a skin notation, to p-toluidine. There is no NIOSH REL for this substance. OSHA is proposing a PEL of 2 ppm as an 8-hour TWA, with a skin notation, for p-toluidine in the construction, agriculture, and maritime industries. This is the limit recently established for this substance in general industry.

p-Toluidine is a white solid that is primarily used as an intermediate in the manufacture of dyes (ACGIH 1986, p. 590).

p-Toluidine is somewhat more acutely toxic than the ortho isomer; the oral LD_{50}s in rats and mice are 656 and 330 mg/kg, respectively (RTECS 1984). p-Toluidine is a powerful methemoglobin former, causing anoxia with symptoms of headache, vertigo, and mental confusion. Acute exposures also cause microscopic or macroscopic hematuria, which clears upon cessation of exposure.

One study is available that investigates the carcinogenic potential of lifetime exposure to p-toluidine in experimental animals (Weisburger, Russfield, Homburger et al. 1978/Ex. 1–535). Male and female mice were exposed to p-toluidine in the diet for a total of 18 months. During the first six months of the experiment, mice were exposed to 1000 or 2000 mg p-toluidine/kg diet. As a result of the weight loss that occurred in mice exposed to the 2000 mg/kg diet dose, the concentrations of p-toluidine were reduced to 500 and 1000 mg/kg diet during the last 12 months of exposure. The rate of food consumption by the animals was not reported but was assumed to be 3 g/day. Thus, the average doses of p-toluidine received during the 18-month exposure were calculated to be 80 and 160 mg/kg body weight per day (Weisburger, Russfield, Homburger et al. 1978/Ex. 1–535).

For both the low and high dietary doses of p-toluidine, a significant increase in the incidence of hepatomas was observed. The incidence of tumors in the control, 80, and 160 mg/kg/day groups were 3/38, 10/38, and 12/35, respectively. The same study (Weisburger, Russfield, Homburger et al. 1978/Ex. 1–535) showed negative results in male rats exposed to two doses of p-toluidine in the diet for 18 months (1000- and 2000-mg/kg diet).

Risk estimate for p-toluidine. To assess the quantitative risk of p-toluidine's carcinogenicity, OSHA used the Weisburger et al. (1978/Ex. 1–535) data which, despite some limitations (e.g., changes in dose levels during the experiment and the absence of data concerning the amount of food animals consumed during the exposure period), were considered adequate for risk assessment purposes.

The maximum likelihood estimates (MLE) of excess cancers per 1,000 workers over an occupational lifetime and the 95-percent upper-bound estimates were obtained by using a linearized multistage model (GLOBAL83). These values are summarized in Table C14–10. This table shows the number of cancer deaths potentially associated with working

lifetime exposure to 20, 5, or 2 ppm p-toluidine.

TABLE C14–10.—MULTISTAGE MODEL ESTIMATES OF CANCER RISK ASSOCIATED WITH WORKING LIFETIME EXPOSURE TO P-TOLUIDINE

Exposure Level	Excess cancer deaths per 1,000 workers	
	MLE	UCL
2 ppm*	12	19
5 ppm	29	46
20 ppm	112	172

* Proposed OSHA PEL in construction, maritime, and agriculture.
MLE = Maximum likelihood estimate of risk.
UCL = Upper-bound (95-percent) confidence limit on maximum likelihood estimate of risk.

OSHA preliminarily concludes, as Table C14–10 shows, that workers in construction, maritime, and agriculture who are exposed to p-toluidine are at significant risk of developing hepatomas, an effect that the Agency considers material impairment of health. For example, the MLE at 20 ppm is 112 excess cancer deaths per 1,000 workers exposed over a working lifetime. Promulgating a PEL of 2 ppm will substantially reduce this significant risk of excess cancer deaths by 90 percent. During the general industry rulemaking, NIOSH (Ex. 8–47, Table N6B) judged the evidence on p-toluidine sufficient to warrant a separate 6(b) rulemaking. Additional data may support a further reduction in the PEL. OSHA is proposing an 8-hour TWA limit of 2 ppm for p-toluidine in the construction, agriculture, and maritime industries; a skin notation is included to protect against percutaneous absorption of this substance. The Agency believes this limit will significantly reduce the cancer risks associated with exposures to this substance at the levels permitted by the absence of a limit. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

VINYL BROMIDE

CAS: 593–60–2; Chemical Formula: C_2H_3Br

H.S. 1425

OSHA has no PEL for vinyl bromide in the construction, agriculture, or maritime industries. NIOSH considers vinyl bromide a potential human carcinogen and recommends that all vinyl halides be controlled as specified for vinyl chloride, with an eventual goal of zero exposure. The ACGIH places vinyl bromide on its A2 list of industrial substances suspected of having carcinogenic potential in humans and has assigned it a TLV®–TWA of 5 ppm. The Agency is proposing a PEL of 5 ppm as a TWA for vinyl bromide in the construction, agriculture, and maritime industries. This is the limit recently established for this substance in general industry.

Vinyl bromide is a colorless gas with a characteristic odor and is used as an intermediate in organic synthesis and in the manufacture of polymers, copolymers, and flame retardants. Its principal use is as a flame retardant.

The oral LD_{50} of a vinyl bromide/corn oil solution was found to be 500 mg/kg in rats. Inhalation by rats of a 100,000-ppm concentration of vinyl bromide produced deep anesthesia and death in 15 minutes; however, when exposure was terminated prior to death, the animals recovered and survived. Exposure of rats to a 50,000-ppm concentration caused unconsciousness in 25 minutes and death in 7 hours; minor liver and kidney injury was observed in these animals. No histopathological changes were observed in rats exposed to a 25,000-ppm concentration for 7 hours. In a subchronic study, no changes in food consumption, histopathology, or hematology were noted in rats, rabbits, and monkeys exposed to 250 or 500 ppm for 6 hours/day, 5 days/week, for 6 months (IARC 1979, Vol. 19, pp. 370–371; IARC 1986, Vol. 39, pp. 138–139).

Henschler and Hoos (1982/Ex. 1–818) believe that vinyl bromide undergoes the same mechanism of biotransformation as its structural analog, vinyl chloride, a recognized human carcinogen that has been regulated by OSHA in a Section 6(b) rulemaking. The microsomal oxidation of vinyl bromide leads to epoxide formation, which results, in turn, in the formation of a reactive intermediate. This intermediate has the potential to form covalent bonds with DNA to produce a mutagenic response. Vinyl bromide has been reported to be mutagenic in *Salmonella typhimurium* and *tradescantia* (IARC 1979a/Ex. 1–1125; NIOSH/OSHA 1978/Ex. 1–1119).

No epidemiological studies have been conducted on populations exposed to vinyl bromide. Benya, Busey, Dorato, and Berteau (1982/Ex. 1–244) reported a positive carcinogenic response in an inhalation study of rats exposed to vinyl bromide vapor; this study is important because inhalation is a major mode of occupational exposure. The results of the Van Duuren (1977/Ex. 1–284) study were equivocal (described below), in that female Swiss albino mice were exposed dermally or by subcutaneous injection either to vinyl bromide in acetone or to polymerized vinyl bromide in an aqueous latex solution.

Benya et al. (1982/Ex. 1–244) exposed male and female Sprague-Dawley rats to 0, 9.7, 52, 247, or 1235 ppm vinyl bromide by inhalation for six hours daily, five days per week, for two years. The incidence of angiosarcomas, primarily of the liver, was found to be statistically significant in all dose groups tested except controls. It should be noted that a closely related chemical analog, vinyl chloride, also causes liver angiosarcomas in humans and animals. The combined incidences of hepatic angiosarcomas in the treated male and female rats were 1/288, 17/240, 86/240, 122/240, and 84/240 for these respective dose levels. One female rat in the control group developed an hepatic angiosarcoma. Table C14–11 summarizes the incidence of angiosarcoma in control and treated rats.

Van Duuren (1977/Ex. 1–284) injected a group of female ICR/Ha Swiss mice once weekly for 48 weeks with 0.05 ml of commercial polymerized vinyl bromide aqueous latex suspension; the animals were observed for 420 days. Nineteen of the 30 mice developed sarcomas at the site of injection. Animals in a positive control group that had been injected with b-propriolactone (0.3 mg/.05 ml trioctanoin) developed 18 sarcomas and three squamous cell carcinomas (in 30 mice). No tumors developed in untreated controls or in controls injected with trioctanoin, an organic solvent, alone (Van Duuren 1977/Ex. 1–284).

In another injection study by the same author, a group of female IRC/Ha Swiss mice were treated with 25 mg vinyl bromide per animal in 0.05 ml trioctanoin once weekly for 48 weeks. The mice were observed for 420 days. One control group was given a weekly injection of trioctanoin alone and the other control group was untreated. No local tumors were seen in any of the test groups, although pathological examination of the animals appears to have been incomplete (Van Duuren 1977/Ex. 1–284).

Application of vinyl bromide to the skin of female ICR/Ha Swiss mice at a dose of 15 mg per animal administered in 0.1 ml of acetone three times weekly for 420 days resulted in no tumors. When this solution was applied once and was followed by an application of phorbol myristyl acetate (PMA) three times weekly, one of 30 mice developed a skin papilloma at 412 days, one control treated with PMA developed a tumor after 44 days, and no untreated controls

developed tumors (Van Duuren 1977/Ex. 1–284).

TABLE C14–11.—INCIDENCE OF ANGIOSARCOMAS IN CONTROL AND VINYL-BROMIDE-EXPOSED RATS

Group	Exposure level (ppm)	Males			Females		
		No. of animals	No. with angiosarcoma	p	No. of animals	No. with angiosarcoma	p
1	Control	144	0		144	1	
2	10	120	7	<0.025	120	10	<0.01
3	50	120	38	<0.001	120	50	<0.001
4	250	120	61	<0.001	120	51	<0.001
5	1250	120	43	<0.001	120	41	<0.001

Source: Benya, Busey, Dorato, and Bertsau (1982/Ex. 1–244).

In another dermal study, a dose of 0.1 ml of polymerized vinyl bromide in an aqueous latex suspension was applied three times weekly to the skin of female ICR/Ha Swiss mice for 420 days. No skin tumors developed. When this solution was applied once, followed by an application of PMA three times weekly, one of 30 mice developed a skin tumor at 175 days. No untreated controls developed skin tumors (Van Duuren 1977/Ex. 1–284).

Based on the studies cited above, IARC (1986, Vol. 39, pp. 137–142) concluded that there was sufficient evidence for the carcinogenicity of vinyl bromide in experimental animals, and classified vinyl bromide as a 2A carcinogen.

Risk estimate for vinyl bromide. The Benya et al. (1982/Ex. 1–244) study was a well-designed and -conducted study that yielded sufficient information for quantitative risk estimation. The route of administration used in the study, inhalation, is directly applicable to occupational exposure, and the incidence of hepatic angiosarcoma was significant. Angiosarcoma is a rare and malignant neoplasm that has a very low background incidence in animals and humans. Therefore, its appearance in the exposed rats supports the premise that vinyl bromide is potentially carcinogenic in humans. Also, it is the same tumor that is associated with the exposure of workers and animals to vinyl chloride, a recognized human carcinogen and a compound whose structure is similar to that of vinyl bromide.

To estimate excess cancer risk over background incidence for a chemical, OSHA relied on the rat inhalation study by Benya et al. (1982/Ex. 1–244). Since both male and female rats responded equally to vinyl bromide treatment, data from the two groups were combined by calculating the geometric means of the risk estimates derived from the male and female response data (Anderson 1983/Ex. 1–1009). The high-dose data for each test group were dropped, since their inclusion makes the dose-response curve nonmonotonic and precludes proper fitting of the linearized multistage risk model (EPA 1984d).

Since cancer risk modeling assumes lifetime exposure, adjustments were made to fit the animal data to this criterion. The adjustments made for the data in the Benya et al. (1982/Ex. 1–244) study were: Multiplying dose by 5/7 to adjust for days of exposure per week and by 6/24 to adjust for hours of exposure per day. These adjusted doses were then changed to human equivalent doses.

Three hypothetical occupational exposure limits, 5 ppm, 20 ppm, and 250 ppm, were used to calculate the maximum likelihood estimates of risk of developing angiosarcoma of the liver. Five ppm is OSHA's limit in general industry. Twenty ppm was chosen as an intermediate exposure level, and 250 ppm was the ACGIH TLV® before the ACGIH reduced it in 1978. These occupational dose levels were also adjusted for lifetime exposure. The adjustments made were: multiplying dose by 5/7 to adjust for days worked per week, by 50/52 to adjust for vacation time, by 8/24 to adjust for hours of exposure per day, and by 45/70 to adjust for work years per lifetime.

Because inhalation is the primary route of exposure to vinyl bromide in occupational settings, the occupational dose was calculated assuming that air intake in humans is 20 m^3 per 24-hour day (Anderson 1983/Ex. 1–1009). The fraction of vinyl bromide absorbed was assumed to be 100 percent, because no absorption rate data were available for vinyl bromide. Because the log p (lipid solubility) value for vinyl bromide (1.52) is similar to that for vinyl chloride (1.38), OSHA assumed that the absorption rates of these two compounds would also be similar. The absorption rate for vinyl chloride used in risk estimations is assumed to be 100 percent (IRIS 1988).

The MLE shown in Table C14–12 for an occupational exposure to 250 ppm of vinyl bromide is 870 excess deaths per 1,000 workers. According to the linearized multistage risk model, 870 of 1,000 workers exposed over their working lifetimes to vinyl bromide at 250 ppm are at risk of developing angiosarcoma. The MLE for an occupational exposure to 5 ppm of vinyl bromide is 0.04; this indicates that, at the proposed PEL, 40 workers per 1,000 exposed to this substance over their occupational lifetimes are at risk of developing angiosarcoma.

TABLE C14–12—MULTISTAGE MODEL ESTIMATES OF CANCER RISK ASSOCIATED WITH LIFETIME EXPOSURE TO VINYL BROMIDE

Exposure level	Excess cancer deaths per 1,000 workers	
	MLE[a]	UCL[a]
5 ppm[b]	40	48
20 ppm[c]	155	180
250 ppm[d]	870	930

[a] Geometric mean of male and female rats.
[b] Proposed PEL in construction, maritime, and agriculture.
[c] Intermediate exposure level.
[d] ACGIH limit before 1978.
MLE = Maximum likelihood estimate of risk.
UCL = 95-percent upper-confidence limit on maximum likelihood estimate on risk.

Table C14–12 indicates that workers exposed to this substance, which was formerly not regulated by OSHA in construction, maritime, or agriculture, are clearly at significant risk of developing hepatic angiosarcomas, the same rare type of tumor associated with exposure to vinyl chloride, a structurally similar substance. OSHA determined in its prior rulemaking on vinyl chloride that this disease constitutes a material impairment of health and functional capacity. At the present time, OSHA preliminarily concludes that establishing a PEL of 5 ppm TWA will substantially

reduce the significant risk of cancer potentially associated with exposure at higher levels formerly permitted in the absence of an OSHA limit for this substance in the construction, agriculture, and maritime industries. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

OSHA also concluded in the general industry rulemaking that there is a significant residual risk at vinyl bromide levels of 5 ppm. The AFL–CIO has challenged the 5 ppm limit as being too high in its petition to the Eleventh Circuit. OSHA has stated to the court that it will reevaluate the PEL for vinyl bromide in its first update of the Air Contaminants Standard. OSHA also preliminarily concludes that there is a residual significant risk associated with the proposed 5 ppm PEL for construction, maritime, and agriculture. However, the 5 ppm PEL provides substantially greater protection to workers than do the levels that could exist in the absence of a limit. OSHA believes that this rulemaking, whose principal purpose is to extend to all workers the increased protection that now exists in general industry, could not be completed in a reasonable period if extensive additional information had to be obtained and analyzed to determine what lower level was feasible and necessary to further reduce the risk. Accordingly, in the first Air Contaminants update, OSHA will also consider for construction, maritime, and agriculture whether a lower limit is appropriate for vinyl bromide.

VINYL CHLORIDE

CAS: 75–01–4; Chemical Formula: $CH_2=CHCL$

H.S. No. 2167

In general industry, construction, and maritime, OSHA's permissible exposure limit for vinyl chloride (VC) is 1 ppm as an 8-hour TWA and 5 ppm as a 15-minute STEL. In addition, no employee may be exposed to vinyl chloride by direct contact with this substance in liquid form. There is no limit in agriculture. The ACGIH designates vinyl chloride an A1 carcinogen and assigns it a TLV®–TWA of 5 ppm. NIOSH considers VC a potential human carcinogen and recommends that exposure be reduced to the lowest feasible level. OSHA is proposing an 8-hour TWA PEL of 1 ppm and a 15-minute STEL of 5 ppm in agriculture. Promulgation of these limits will make the PELs for VC consistent across all OSHA-regulated sectors.

Vinyl chloride (chlorethene) is a synthetic organic chemical made from ethylene or acetylene and chloride by any one of several processes. It is the parent compound of a series of thermoplastic resin polymers and copolymers which are widely used for containers, wrapping film, electrical insulation, pipe, conduit, and a variety of other industrial and consumer products. Vinyl chloride is used primarily in the production of polyvinyl chloride (PVC), a resin which is produced through batch processing. The conversion of the VC monomer into a polymer or copolymer is an incomplete process, i.e., not all of the monomer is reacted.

The carcinogenicity of vinyl chloride for three animal species (rat, mouse, hamster) has been documented by Maltoni. Moreover, Maltoni's investigations have demonstrated a dose-dependent relationship for the induction of tumors (i.e., more tumors occur at higher exposure levels), including angiosarcoma of the liver, in rats. Of 200 mice (100 males, 100 females) exposed to 50 ppm of vinyl chloride by inhalation for eleven months, 100 died. Sixty-four animals died without gross postmortem pathologic examination being performed. Of the 36 remaining animals for which a gross postmortem pathologic examination was performed, 13 (36 percent) were found to have liver tumors (including angiosarcomas), 21 (58 percent) lung tumors, 9 (25 percent) skin tumors, and one had a kidney tumor. A similar relationship has been demonstrated in both rats and mice. These investigators have induced angiosarcoma of the liver in rats and mice at exposure concentrations of 50 ppm, and in hamsters at higher concentrations of exposure. Additional tumors involving other organs, including the kidneys, lungs, and skin of exposed animals, were also observed in frequencies much in excess of these in control animals.

According to the 1970 report by the Surgeon General's Ad-Hoc Committee on the Evaluation of Low Levels of Environmental Chemical Carcinogens, the finding of cancer in two or more animal species may be extrapolated to indicate a carcinogenic hazard to humans. Here, such a finding was made in three species that were exposed to VC by inhalation, a route comparable to employee exposure. In addition, there were at least 13 confirmed cases of angiosarcoma of the liver among employees exposed to VC, a particularly significant number in view of the extreme rarity of this cancer in the U.S. adult male population (testimony of Dr. Marcus Key, Director of NIOSH, at the 1974 rulemaking hearing).

The findings of angiosarcoma of the liver in both experimental animals and exposed employees is compelling evidence that exposure of humans to vinyl chloride induces this tumor. Industry spokesmen, at the Agency's 1974 hearing, conceded that VC is carcinogenic in humans (e.g., testimony of Dr. McBurney, VC rulemaking hearing, Tr. 1041). Accordingly, it is concluded that VC must be regarded as a human carcinogen and the probable causal agent of angiosarcoma of the liver, and that exposure of employees to VC must be controlled.

Additional evidence of tumor induction in a variety of other organs, including lung, kidney, brain, and skin, as well as non-malignant alterations, such as fibrosis and connective tissue deterioration, indicates additional oncogenic and toxicologic properties of vinyl chloride.

Based on this evidence in humans and animals, OSHA concluded in the VC rulemaking that exposure to VC causes cancer in humans and animals. OSHA therefore believes that, in the absence of a limit, workers in agriculture are at significant risk of experiencing these adverse health effects. The Agency believes that the proposed limits of 1 ppm as an 8-hour TWA and 5 ppm as a 15-minute STEL are necessary to substantially reduce these risks of material health impairment among these workers. Promulgation of these limits will make the PELs for VC consistent all OSHA-regulated sectors.

VINYL CYCLOHEXENE DIOXIDE

CAS: 106–87–6; Chemical Formula: $C_8H_{12}O_2$

H.S. No. 1426

OSHA has no PEL for vinyl cyclohexene dioxide (VCD) in the construction, agriculture, and maritime industries. The ACGIH classifies VCD as a suspected human carcinogen (A2) and has assigned it a TLV®–TWA of 10 ppm, with a skin notation. NIOSH has no REL for this substance but concurs (Ex. 8–47, Table N6A) with the limit being proposed. OSHA is proposing a 10 ppm TWA PEL, with a skin notation, for this substance in the construction, agriculture, and maritime industries. This is the limit recently established in general industry.

Vinyl cyclohexene dioxide is a colorless liquid used as a chemical intermediate and as a monomer in the manufacture of polyglycols containing unreacted epoxy groups (Hine, Rowe, White, Darmer, and Youngblood 1981/ Ex. 1–976). It is also used as a reactive diluent for other diepoxides and certain epoxy resins (IARC 1976).

Vinyl cyclohexene dioxide is a mild acute toxin when ingested; the oral LD_{50} in rats has been reported to be 2.8 g/kg (ACGIH 1986, p. 627) and the LC_{50} in rats for a 4-hour inhalation period is 800 ppm (RTECS 1990). Dermal exposure to this substance has caused mild-to-moderate skin irritation in workers, with occasional instances of marked skin irritation. Vinyl cyclohexene dioxide can be absorbed through the skin in significant amounts; the dermal LD_{50} in rabbits is 0.62 mg/kg (ACGIH 1986, p. 627).

Turchi, Bonatti, Citti et al. (1981/Ex. 1–282) assayed the mutagenicity of VCD and several other epoxides using the TA100 strain of *S. typhimurium* and V79 Chinese hamster cells; these authors also investigated the alkylating properties of these chemicals. VCD tested positive in both the *S. typhimurium* test (point mutation) and the V79 Chinese hamster cell test (both point mutation and chromosome aberration), and had an intermediate alkylating capacity relative to other epoxide compounds tested.

There are no data concerning the adverse health effects of VCD in humans. There are no reports as a result of industrial experience that reveal carcinogenic effects in workers caused by VCD exposure (ACGIH 1986/Ex. 1–3).

Four studies have reported the development of skin tumors in mice exposed dermally to VCD (Hendry, Homer, and Rose 1951/Ex. 1–250; Kotin and Falk 1963/Ex. 1–287; Weil, Condra, Haun, and Streigel 1963/Ex. 1–257; and Van Duuren, Nelson, Orris, Palmes, and Schmitt 1963/Ex. 1–288). The study of Van Duuren et al. (1963/Ex. 1–288) included controls and is thus particularly well suited for an evaluation of VCD's carcinogenic potential.

These authors painted 30 male Swiss ICR/Ha mice with 0.1 ml of a 10-percent solution of VCD in benzene three times per week (approximately 100 mg of solution per application). Two negative controls were used; one set of 150 mice was treated with benzene alone and another set of 207 mice was not treated with anything. Fourteen of the 30 VCD-treated mice developed skin tumors after an undefined length of time (mean survival time was 326 days). The incidences of skin tumors in the controls were 11/150 and 13/207 for the benzene-treated and untreated mice, respectively. The incidence of skin tumors in the VCD-treated mice was significantly greater (reported in terms of a total tumor index rather than number of tumors) than the incidence observed in either of the controls (Van Duuren, Nelson, Orris, Palmes, and Schmitt 1963/Ex. 1–288).

A recent study by the NTP reported that skin tumors developed in rats exposed to 31.5 g/kg applied to the skin for 2 years. In addition, this same study reported that vinyl cyclohexene dioxide induced mutations in mouse lymphocyte cells and sister chromatid exchanges in hamster ovary cells when administered *in situ* (RTECS 1990).

The studies of Van Duuren et al. (1963/Ex. 1–288) and the NTP (RTECS 1990) demonstrate the carcinogenicity of VCD in experimental animals. OSHA considered the possibility of conducting a quantitative risk assessment for VCD, but the Agency preliminarily concluded that the dose-response data in this study are unsuitable for quantitative risk assessment purposes because the VCD was administered in a solution of benzene, which is itself regulated as a carcinogen and classified as such by several authorities (IARC, NTP, NIOSH, and ACGIH). Even though the Van Duuren et al. (1963/Ex. 1–288) study included a control for the independent carcinogenic effects of benzene, the possibility of a synergistic or additive effect of benzene on VCD cannot be completely ruled out.

Vinyl cyclohexene dioxide has been shown to be carcinogenic by dermal application in mice, and four studies have confirmed these effects. Based on these animal studies showing VCD's carcinogenicity, OSHA preliminarily concludes that exposed employees in agriculture, construction, and maritime are at significant risk of cancer from exposure to VCD at the levels permitted by the absence of an OSHA limit. The Agency considers this effect a material impairment of health and preliminarily concludes that a 10 ppm 8-hour-TWA PEL, with a skin notation, is necessary to substantially reduce the significant occupational risk confronting VCD-exposed employees in these sectors. In addition, promulgation of this limit will make OSHA's PEL for this substance consistent across all regulated sectors.

Preliminary Conclusions for this Group of Substances. The Supreme Court in *I.U.D.* v. *A.P.I.* (*supra*, the *Benzene* decision) gave OSHA directions as to its decisional process; that case involved a carcinogen. OSHA is using the Supreme Court's guidance within the context of this present broader rulemaking. OSHA is also using the approach it has taken in the regulation of arsenic, benzene, EtO, asbestos, and formaldehyde; this approach has been upheld in the Courts of Appeals (see the introduction to this section). For the substances in this section, OSHA has either (1) already conducted (in the case of substance-specific Section 6(b) standards), or (2) considered conducting quantitative risk assessments for each chemical discussed in this section. When adequate dose-response data were available, OSHA performed quantitative appraisals of the significance of the risk, either in the substance-specific rulemakings, the general-industry Air Contaminants rulemaking, or the present rulemaking. These risk assessments follow the approach OSHA has used in prior rulemakings for carcinogens, a process that has repeatedly been upheld by the courts.

OSHA conducted its significant risk analyses using the principles suggested by the Supreme Court and adopted in its carcinogen rulemakings subsequent to *I.U.D.* v. *A.P.I.* OSHA has proposed to establish new or revised exposure limits based on these analyses when they demonstrated that significant risk existed at the former PEL (if any) and that the new limit would substantially reduce significant risk and represent a feasible limit.

In some cases, the available data were not sufficient to conduct quantitative estimates of cancer risk at the same level of detail as in other cases. In these cases, OSHA believes that the analyses performed demonstrate that the new limits are necessary to reduce a significant risk of cancer mortality among workers in construction, maritime, and agriculture.

Overall, OSHA believes its analyses of the proposed new or revised limits for carcinogenic chemicals meet the Agency's legal requirements. OSHA preliminarily concludes that these limits will lead to substantial reductions in the significant risk of material impairment of health among workers in these sectors and are additionally feasible to attain.

15. Substances for Which 1987–1988 ACGIH TLV®s are Less Stringent Than Existing OSHA PELs

OSHA is eliminating this category, which appeared both in the proposed (53 *FR* 20961 et seq.) and final (54 *FR* 2332 et seq.) rules for Air Contaminants in general industry. In re-evaluating this category of substances, which included those for which the TLV®s were less stringent than the corresponding OSHA PELs, the Agency has made a determination that these substances should be reassigned to other categories based on the health effects that provide the basis for establishing their respective limits. These substances have accordingly been reassigned to the following categories:

Substance	New category
Camphor (synthetic)	Sensory Irritation.
Copper fumes (measured as Cu).	Sensory Irritation.
1,1–Dichloroethane	Liver Toxicity.
Fluorine	Sensory Irritation.
Hexachloroethane	Sensory Irritation.
Nickel carbonyl	Cancer.
Rhodium, metal fume & insolubie salts (measured as Rh).	Sensitization
Silica, amorphous, diatomaceous earth.	Respiratory Effects
Silica, amorphous, precipitate & gel.	Respiratory Effects.
Silver, metal dust & fume (measured as Ag).	Systemic Effects.
Tetraethyl lead	Systemic Effects.
Tetramethyl lead–	Systemic Effects.
Uranium, soluble compounds (measured as U).	Kidney Effects.

16. Substances for Which OSHA is Proposing to Establish Short-Term Exposure Limits

Introduction. OSHA is proposing to establish a short-term exposure limit (STEL) for a total of 116 substances; these substances are listed in Table C15–1. OSHA is proposing these PELs for the construction, maritime, and agriculture industries. These STELs were recently adoped in general industry and thus promulgation in the construction, maritime, and agriculture sectors will make these STELs consistent across all OSHA-regulated sectors.

When OSHA adopted the 1970 ACGIH TLV®s for the construction and maritime industries, the ACGIH had not established the short-term TLV® category; as a consequence, none of the limits currently in effect in these industries have STELs. (However, some of the substances on OSHA's previous Z–2 table, whose limits derive from standards established by the American National Standards Institute rather than the ACGIH, have "acceptable ceiling concentrations" that act, in effect, as short-term exposure limits.)

The ACGIH defines a STEL as: a 15-minute time-weighted average exposure which should not be exceeded at any time during a work day even if the eight-hour time-weighted average is within the TLV®. Exposures at the STEL should not be longer than 15 minutes and should not be repeated more than four times per day. There should be at least 60 minutes between successive exposures at the STEL. An averaging period other than 15 minutes may be recommended when this is warranted by observed biological effects (ACGIH 1991).

TABLE C16–1.—SUBSTANCES FOR WHICH OSHA IS PROPOSING TO ESTABLISH STELS TO SUPPLEMENT TWA LIMITS IN CONSTRUCTION, MARITIME, AND AGRICULTURE

H.S. number/chemical name	CAS No.	Proposed STEL
1001 Acetaldehyde	75–07–0	150 ppm.
1004 Acetone	67–64–1	1000 ppm.
1005 Acetonitrile	75–05–8	60 ppm.
1007 Acrolein	107–02–8	0.3 ppm.
1010 Allyl alcohol	107–18–6	4 ppm.
1011 Allyl chloride	107–05–1	2 ppm.
1012 Allyl glycidyl ether (AGE)	106–92–3	10 ppm.
1013 Allyl propyl disulfide	2179–59–1	3 ppm.
1022 Ammonium chloride fume	12125–02–9	20 mg/m³.
1042 Bromine	7726–95–6	0.3 ppm.
1045 2-Butanone (MEK)	78–93–3	300 ppm.
1047 n-Butyl acetate	123–86–4	200 ppm.
1050 tert-Butyl alcohol	75–65–0	150 ppm.
1058 p-tert-Butyltoluene	98–51–1	20 ppm.
1064 Caprolactam Dust	105–60–2	3 mg/m³.
1065 Caprolactam Vapor	105–60–2	40 mg/m³.
1069 Carbon dioxide	124–38–9	30,000 ppm.
1070 Carbon disulfide	75–15–0	12 ppm.
1072 Carbon tetrabromide	558–13–4	0.3 ppm.
1074 Carbonyl fluoride	353–50–4	5 ppm.
1078 Chlorinated camphene	8001–35–2	1 mg/m³.
1079 Chlorine	7782–50–5	1 ppm.
1080 Chlorine dioxide	10049–04–4	0.3 ppm.
1089 o-Chlorostyrene	2039–87–4	75 ppm.
1114 Decaborane	17702–41–9	0.15 ppm.
1116 Di-sec-octyl-phthalate	117–81–7	10 mg/m³.
1119 Dibutyl phosphate	107–66–4	2 ppm.
1122 1,3-Dichloro-5,5-dimethylhydantoin	118–52–5	0.4 mg/m³.
1125 p-Dichlorobenzene	106–46–7	110 ppm.
1127 Dichloroethyl ether	111–44–4	10 ppm.
1137 Diethylamine	109–89–7	25 ppm.
1143 Dimethylaniline	121–69–7	10 ppm.
1149 Dipropylene glycol methyl ether	34590–94–8	150 ppm.
1159 Ethanolamine	141–43–5	6 ppm.
1161 Ethyl acrylate	140–88–5	25 ppm.
1162 Ethyl benzene	100–41–4	125 ppm.
1163 Ethyl bromide	74–96–4	250 ppm.
1164 Ethyl ether	60–29–7	500 ppm.
1168 Ethylene dichloride	107–06–2	2 ppm.
1177 Ferrovanadium dust	12604–58–9	3 mg/m³.
1182 Formamide	75–12–7	30 ppm.
1184 Furfuryl alcohol	98–00–0	15 ppm.
1185 Gasoline	8006–61–9	500 ppm.
1194 n-Heptane	142–82–5	500 ppm.
1201 Hexane isomers	Varies	1000 ppm.
1203 Hexone (Methyl isobutyl ketone)	108–10–1	75 ppm.
1208 Hydrogen fluoride	7664–39–3	6 ppm.
1209 Hydrogen sulfide	7783–06–4	15 ppm.
1216 Iron pentacarbonyl	13463–40–6	0.2 ppm.

TABLE C16–1.—SUBSTANCES FOR WHICH OSHA IS PROPOSING TO ESTABLISH STELS TO SUPPLEMENT TWA LIMITS IN CONSTRUCTION, MARITIME, AND AGRICULTURE—Continued

H.S. number/chemical name	CAS No.	Proposed STEL
1218 Isoamyl alcohol	123–51–3	125 ppm.
1222 Isophorone diisocyanate	4098–71–9	0.02 ppm.
1224 Isopropyl acetate	108–21–4	310 ppm.
1225 Isopropyl alcohol	67–63–0	500 ppm.
1227 Isopropyl glycidyl ether	4016–14–2	75 ppm.
1228 Isopropylamine	75–31–0	10 ppm.
1231 Ketene	463–51–4	1.5 ppm.
1236A Manganese fume	7439–96–5	3 mg/m³.
1242 Mercury (organo), alkyl compounds	7439–97–6	0.03mg/m³.
1243 Mesityl oxide	141–79–7	25 ppm.
1248 Methyl 2-cyanoacrylate	137–05–3	4 ppm.
1249 Methyl acetate	79–20–9	250 ppm.
1250 Methyl acetylene/propadiene mixture	None	1250 ppm.
1252 Methyl alcohol	67–56–1	250 ppm.
1254 Methyl chloride	74–87–3	100 ppm.
1255 Methyl chloroform (1,1,1-trichloroethane)	71–55–6	450 ppm.
1258 Methyl formate	107–31–3	150 ppm.
1261 Methyl isobutyl carbinol	108–11–2	40 ppm.
1267 alpha-Methyl styrene	98–83–9	100 ppm.
1270 o-Methylcyclohexanone	583–60–8	75 ppm.
1281 Morpholine	110–91–8	30 ppm.
1282 Naphthalene	91–20–3	15 ppm.
1286 Nitric acid	7697–37–2	4 ppm.
1295 Octachloronaphthalene	2234–13–1	0.3 mg/m³.
1296 Octane	111–65–9	375 ppm.
1298 Osmium tetroxide	20816–12–0	0.006 mg/m³.
1299 Oxalic acid	144–62–7	2 mg/m³.
1301 Ozone	10028–15–6	0.3 ppm.
1304 Pentaborane	19624–22–7	0.015 ppm.
1306 Pentane	109–66–0	750 ppm.
1307 2-Pentanone (Methyl propyl ketone)	107–87–9	250 ppm.
1309 Perchloryl fluoride	7616–94–6	6 ppm.
1317 Phenylhydrazine	100–63–0	10 ppm.
1319 Phorate (Thimet)	298–02–2	0.2 mg/m³.
1320 Phosdrin (Mevinphos)	7786–34–7	0.3 mg/m³.
1321 Phosphine	7803–51–2	1 ppm.
1322 Phosphoric acid	7664–38–2	3 mg/m³.
1324 Phosphorus pentasulfide	1314–80–3	3 mg/m³.
1325 Phosphorus trichloride	7719–12–2	0.5 ppm.
1338 n-Propyl acetate	109–60–4	250 ppm.
1339 Propyl alcohol	71–23–8	250 ppm.
1340 n-Propyl nitrate	627–13–4	40 ppm.
1341 Propylene dichloride	78–87–5	110 ppm.
1343 Propylene glycol monomethyl ether	107–98–2	150 ppm.
1346 Resorcinol	108–46–3	20 ppm.
1366 Sodium fluoroacetate	62–74–8	0.15 mg/m³.
1372 Styrene (Phenylethylene)	100–42–5	100 ppm.
1375 Sulfur dioxide	7446–09–5	5 ppm.
1379 Sulfuryl fluoride	2699–79–8	10 ppm.
1387 Tetrahydrofuran	109–99–9	250 ppm.
1397 Toluene	108–88–3	150 ppm.
1398 Toluene-2,4-diisocyanate	584–84–9	0.02 ppm.
1403 1,1,2–Trichloro 1,2,2-trifluoroethane	76–13–1	1250 ppm.
1406 Trichloroethylene	79–01–6	200 ppm.
1408 Triethylamine	121–44–8	15 ppm.
1411 Trimethylamine	75–50–3	15 ppm.
1416 Tungsten & compounds(insoluble)	7440–33–7	10 mg/m³.
1417 Tungsten & compounds(soluble)	7440–33–7	3 mg/m³.
1418 Uranium (insoluble compounds)	7440–61–1	0.6 mg/m³.
1424 Vinyl acetate	108–05–4	20 ppm.
1429 VM&P Naphtha	8032–32–4	400 ppm.
1430a, 1430b Wood dust all soft and hardwoods except Western red cedar	None	10 mg/m³.
1431 Xylene (o,m,p-isomers)	1330–20–7	150 ppm.
1435 Zinc chloride fume	7646–85–7	2 mg/m³.
1437 Zinc oxide fume	1314–13–2	10 mg/m³.
1435 Zirconium compounds	7440–67–7	10 mg/m³.

Reasons for ACGIH STELs. The ACGIH establishes STELs for substances that cause a wide variety of acute effects; these effects include irritation, narcosis, lung damage, systemic effects, and organic poisoning. The ACGIH first considered adding STELs to the TLV®–TWAs for some substances in 1971, when it appointed a subcommittee to study the appropriateness of adding such exposure limits to the TLV® list.

In 1973, this subcommittee recommended that the ACGIH establish STELs as a third category (along with TLV®–TWAs and TLV® ceilings) of exposure limits. The STEL was defined as the maximum concentration to which workers can be exposed continuously *for a period of up to 15 minutes* without suffering from

1. Intolerable irritation,
2. Chronic or irreversible tissue change, or
3. Narcosis of sufficient degree to increase accident proneness, impair self-rescue, or materially reduce work efficiency (Supplemental Documentation to the Fourth Edition of the Documentation of the Threshold Limit Values, ACGIH 1984).

The ACGIH stipulated that no more than four such excursions per day were permissible, with at least 60 minutes between exposure periods, and, that these excursions could not cause the daily TLV®-TWA to be exceeded.

In 1974, the ACGIH agreed by consensus that 425 of the 520 compounds in its 1973 list should have STELs assigned to them, but these were not in fact published until 1976, when "Tentative Values" for STELs were listed in the organization's annual booklet. The 1987-1988 ACGIH TLV® booklet states that the TLV®-STEL is "the concentration to which workers can be exposed continuously for a short period of time without suffering from (1) Irritation, (2) chronic or irreversible tissue damage, or (3) narcosis of sufficient degree to increase the likelihood of accidental injury, impair self-rescue or materially reduce work efficiency . . . provided that the daily TLV®-TWA is not exceeded."

In 1982, the ACGIH limited the conditions calling for STELs to "[those situations] where toxic effects have been reported from high short-term exposures in either humans or animals." Since that time, the ACGIH has re-examined the toxicological data and subsequently deleted the STELs for several hundred substances because, in the opinion of the ACGIH, there was insufficient evidence that acute exposures to elevated levels of these substances caused adverse effects. The ACGIH has stressed that STELs should be set on physiological grounds rather than in response to sampling and analytical limitations (ACGIH 1984).

In addition to the STEL category, the ACGIH in the 1970s established a fourth limit, a general "excursion factor" that should always be observed implicitly but is not specifically assigned to each chemical. The "excursion limit" recommended by the ACGIH:

> should exceed three times the TLV-TWA for no more than a total of 30 minutes during a work day and under no circumstances should they exceed five times the TLV-TWA, provided that the TLV-TWA is not exceeded (ACGIH 1987).

The basis for this excursion limit is good industrial hygiene practice: any process that has emissions that exceed the concentrations permitted by this excursion factor is not under good industrial hygiene control, and the ACGIH believes that, in such cases, efforts should be made to restore control (ACGIH 1991). Where a specific STEL exists for a substance, the specific STEL takes precedence over the general excursion limit (ACGIH 1987). Thus *all* ACGIH TLV®-TWAs have implicit excursion limits, but only a few substances (i.e., those for which specific toxicological evidence indicates that a STEL is necessary) have explicit STELs.

Basis for Short-Term Limits Being Proposed by OSHA. The STELs being proposed by OSHA in this rulemaking, which parallel those STELs remaining in the ACGIH's most recent list (ACGIH 1990-1991) are thus limits for substances for which there is toxicological evidence that short-term high exposures cause recognized acute effects. The health effects associated with short-term exposures to some of these substances are shown in Table C15-2.

TABLE C16-2.—HEALTH EFFECTS SUPPORTING PROPOSED STELS

H.S. number/chemical name	Proposed STEL	Health effects
1001 Acetaldehyde	150 ppm	Eye irritation; narcosis; potential injury to respiratory tract.
1004 Acetone	1000 ppm	Eye, nose, and throat irritation; narcosis.
1005 Acetonitrile	60 ppm	Nausea; headache; convulsions.
1007 Acrolein	0.3 ppm	Irritation; lung edema.
1010 Allyl alcohol	4 ppm	Irritation.
1011 Allyl chloride	2 ppm	Mucous membrane irritation.
1012 Allyl glycidyl ether (AGE)	10 ppm	Irritation.
1013 Allyl propyl disulfide	3 ppm	Irritation; lacrimation.
1022 Ammonium chloride fume	20mg/m³	Irritation.
1042 Bromine	0.3 ppm	Respiratory tract irritation.
1045 2-Butanone (MEK)	300 ppm	Eye and nose irritation.
1047 n-Butyl acetate	200 ppm	Throat irritation.
1050 tert-Butyl alcohol	150 ppm	Narcosis.
1056 p-tert-Butyltoluene	20 ppm	Eye, nose, and throat irritation.
1064 Caprolactam dust	3 mg/m³	Irritation.
1065 Caprolactam vapor	40 mg/m³	Irritation.
1069 Carbon dioxide	30,000 ppm	Central nervous system effects; asphyxiation.
1072 Carbon tetrabromide	0.3 ppm	Upper respiratory tract irritation; injury to lungs, liver, and kidney.
1074 Carbonyl fluoride	5 ppm	Respiratory irritation.
1079 Chlorine	1 ppm	Eye, mucous membrane, skin, and pulmonary irritation.
1080 Chlorine dioxide	0.3 ppm	Irritation.
1089 o-Chlorostyrene	75 ppm	Dizziness; nausea; headache.
1114 Decaborane	0.15 ppm	Hyperexcitability; narcosis.
1119 Dibutyl phosphate	2 ppm	Irritation to respiratory tract; headaches.
1122 1,3-Dichloro5,5-dimethylhydantoin	0.4mg/m³	Respiratory irritation.
1125 p-Dichlorobenzene	110 ppm	Acute poisoning.
1127 Dichloroethyl ether	10 ppm	Upper respiratory tract and eye irritation.
1137 Diethylamine	25 ppm	Acute toxicity characterized by strong local irritation.
1143 Dimethylaniline	10 ppm	Methemoglobinemia; CNS depression.
1149 Dipropylene glycol methyl ether	150 ppm	Eye, nose, and throat irritation; central nervous system impairment.
1159 Ethanolamine	6 ppm	Pulmonary irritation.
1161 Ethyl acrylate	25 ppm	Irritation.
1162 Ethyl benzene	125 ppm	Skin and eye irritation.
1163 Ethyl bromide	250 ppm	Narcosis.
1164 Ethyl ether	500 ppm	Narcosis; nasal irritation.
1168 Ethylene dichloride	2 ppm	Central nervous system effects.
1177 Ferrovanadium dust	3 mg/m³	Eye and respiratory irritation.

TABLE C16-2.—HEALTH EFFECTS SUPPORTING PROPOSED STELS—Continued

H.S. number/chemical name	Proposed STEL	Health effects
1184 Furfuryl alcohol	15 ppm	Eye irritation.
1185 Gasoline	500 ppm	Narcosis; irritation.
1194 n-Heptane	500 ppm	Narcosis; respiratory irritation.
1201 Hexane isomers	1000 ppm	Narcotic symptoms; eye and throat irritation; slight nausea, headache.
1203 Hexone (MIBK)	75 ppm	Irritant effects.
1206 Hydrogen fluoride	6 ppm	Eye and respiratory irritation.
1209 Hydrogen sulfide	15 ppm	Eye irritation.
1216 Iron pentacarbonyl	0.2 ppm	Headaches; dizziness.
1218 Isoamyl alcohol	125 ppm	Respiratory and eye irritation.
1222 Isophorone diisocyanate	0.02 ppm	Respiratory effects and sensitization; pulmonary irritation.
1224 Isopropyl acetate	310 ppm	Eye and respiratory irritation.
1225 Isopropyl alcohol	500 ppm	Narcotic effects and irritation.
1227 Isopropyl glycidyl ether	75 ppm	Respiratory tract and eye irritation.
1228 Isopropylamine	10 ppm	Respiratory irritation.
1231 Ketene	1.5 ppm	Respiratory irritation.
1236A Manganese fume	3 mg/m³	Central nervous system effects.
1242 Mercury, (organo) alkyl compounds	0.03 mg/m³	Central nervous system effects; irritation.
1249 Mesityl oxide	25 ppm	Eye and mucous membrane irritation, breathing difficulty, headache and vertigo.
1248 Methyl 2-cyanoacrylate	4 ppm	Nasal and eye irritation.
1249 Methyl acetate	250 ppm	Ocular and nervous disturbances; eye, mucous membrane, upper and lower respiratory tract irritation.
1252 Methyl alcohol	250 ppm	Recurrent headaches; diminution of vision.
1254 Methyl chloride	100 ppm	Narcosis.
1255 Methyl chloroform (1,1,1-trichloroethane)	450 ppm	Anesthesia.
1258 Methyl formate	150 ppm	Visual disturbances (temporary blindness); narcotic symptoms, mucous membrane irritation; dyspnea.
1261 Methyl isobutyl carbinol	40 ppm	Eye irritation.
1267 alpha-Methyl styrene	100 ppm	Eye irritation.
1270 o-Methylcyclohexanone	75 ppm	Eye and respiratory irritation.
1281 Morpholine	30 ppm	Irritation and harmful effects to eyes and vision.
1282 Naphthalene	15 ppm	Ocular effects.
1286 Nitric acid	4 ppm	Respiratory irritation.
1296 Octane	375 ppm	Acute effects on nervous system.
1298 Osmium tetroxide	0.006 mg/m³	Irritation; conjunctivitis.
1299 Oxalic acid	2mg/m³	Severe local burns to eyes, mucous membranes, and skin.
1301 Ozone	0.3 ppm	Pulmonary congestion; eye, nose, and throat irritation.
1304 Pentaborane	0.015 ppm	Central nervous system effects.
1306 Pentane	750 ppm	Narcotic and irritative effects.
1307 2-Pentanone (MPK)	250 ppm	Narcotic effects; irritation.
1309 Perchloryl fluoride	6 ppm	Respiratory irritation; fluorosis.
1317 Phenylhydrazine	10 ppm	Sensitization effects.
1319 Phorate (Thimet)	0.2 mg/m³	Cholinesterase inhibition.
1320 Phosdrin (Mevinphos)	0.3 mg/m³	Cholinesterase inhibition.
1321 Phosphine	1 ppm	Pulmonary irritation.
1322 Phosphoric acid	3 mg/m³	Respiratory irritation.
1324 Phosphorus pentasulfide	3mg/m³	Respiratory irritation.
1325 Phosphorus trichloride	0.5 ppm	Respiratory irritation.
1338 n-Propyl acetate	250 ppm	Irritation; narcosis.
1339 Propyl alcohol	250 ppm	Possible deep narcosis.
1340 n-Propyl nitrate	40 ppm	Irritation; headache, nausea.
1341 Propylene dichloride	110 ppm	Eye irritation; central nervous system effects.
1343 Propylene glycol monomethyl ether	150 ppm	Eye irritation.
1346 Resorcinol	20 ppm	Eye and skin irritation.
1366 Sodium Fluoroacetate	0.15 mg/m³	Metabolic inhibition.
1372 Styrene, monomer	100 ppm	Tremors with subsequent severe convulsions; pulmonary edema may follow severe single exposure.
1375 Sulfur dioxide	5 ppm	Respiratory effects.
1379 Sulfuryl fluoride	10 ppm	Central nervous system effects; pulmonary irritation.
1387 Tetrahydrofuran	250 ppm	Narcotic and irritative effects.
1397 Toluene	150 ppm	Impairment of coordination, momentary memory loss, anorexia.
1398 Toluene-2,4-diisocyanate	0.02 ppm	Sensitization effects.
1403 1,1,2-Trichloro-1,2,2-tri-fluoroethane	1250 ppm	Impairment of psychomotor performance.
1406 Trichloroethylene	200 ppm	Narcosis.
1408 Triethylamine	15 ppm	Acute irritation of eyes, mucous membranes, and lungs.
1411 Trimethylamine	15 ppm	Irritation.
1424 Vinyl acetate	20 ppm	Irritation.
1428 Vinylidene chloride	20 ppm	Overt toxicity.
1430a, 1430b Wood dust, all soft and hardwoods, except Western red cedar	10 mg/m³	Respiratory effects.
1431 Xylene (o,m,p-isomers)	150 ppm	Narcosis, irritant effects.
1435 Zinc chloride (fume)	2mg/m³	Respiratory irritation.
1437 Zinc oxide fume	10 mg/m³	Metal fume fever.

OSHA believes that the STELs and ceilings being proposed reflect the concerns expressed by many in the previous rulemaking that short-term limits be promulgated when a toxicologic basis exists for the short-

term limit. In general, OSHA is proposing STELs or ceiling limits when the toxicologic evidence for a particular substance indicates that the 8-hour TWA PEL alone would be insufficient to protect employees from experiencing adverse effects related to short-term exposure to elevated concentrations of that substance.

OSHA has not proposed establishing a general excursion limit, such as that recommended by the ACGIH, that applies to all regulated substances. However, there are workplace situations where OSHA believes that worker protection requires the implementation of a STEL. For example, OSHA believes that the severity of the health effect caused by exposure and the pattern of exposure prevalent in operations involving a given substance are both factors that should be considered when determining whether a short-term limit is appropriate. OSHA preliminarily concludes that, in these instances, promulgating a STEL is a necessary and appropriate measure for ensuring that workplace conditions will be maintained under a sufficient degree of control to ensure that workers are protected from experiencing serious exposure-related health effects.

17. Substances for Which OSHA Is Proposing to Add Skin Designations

For many of the substances included in this rulemaking, OSHA is proposing either to retain or to add skin designations in recognition of the capacity of these substances to be absorbed through the skin in sufficient quantities to cause toxic effects. Ninety-eight of these substances already have skin designations that apply in the maritime and construction sectors, and OSHA is proposing to add skin designations for an additional 42 substances in these sectors. In agriculture, where OSHA has previously not had limits, skin designations for all 140 substances are being proposed. Table C17–1 shows all of the substances for which the Agency is proposing to establish skin designations; those substances with asterisks already have skin designations in construction and maritime.

The ACGIH began to include skin designations for the chemicals in its list for the first time in 1961 (Stokinger 1962/Ex. 1–998). At that time, the organization stated that:

> This notation is to be interpreted simply as an indicator that skin absorption may contribute to the overall intake from exposure in addition to that from inhalation. It refers mainly to absorption from liquid contamination (Stokinger 1962/Ex. 1–998).

The ACGIH has expanded on its reasoning since the 1960s, and the preface to the most recent Threshold Limit Values and Biological Exposure Indices for 1987–1988 (ACGIH 1990) explains that the skin designation is designed to call attention to the need for "appropriate measures for the prevention of cutaneous absorption so that the threshold limit is not invalidated." Thus, a skin notation warns that exposure via the cutaneous route, including absorption through the eyes or mucous membranes, may contribute substantially to an employee's overall exposure and cause systemic toxicity.

TABLE C17–1.—LIST OF SUBSTANCES FOR WHICH OSHA IS PROPOSING TO ADD SKIN DESIGNATIONS

H.S. number/chemical name	CAS No.
1006 Acrylamide	79–06–1
1009 Acrylic acid	79–10–7
2003 Aldrin*	309–00–2
1010 Allyl alcohol*	107–18–6
1025 Aniline and homologs*	62–53–3
2007 Anisidine*	29191–52–4
2014 Azinphos-methyl*	86–50–0
2021 Bromoform*	75–25–2
1040 2-Butoxyethanol*	111–76–2
1051 n-Butyl alcohol	71–36–3
2024 tert-Butyl chromate (as CrO_3)*	1189–85–1
1055 o-sec-Butylphenol	89–72–5
1070 Carbon disulfide*	75–15–0
1075 Catechol	120–80–9
2026 Chlordane*	57–74–9
1078 Chlorinated camphene*	8001–35–2
1084 o-Chlorobenzylidene malononitrile	2698–41–1
2035 Chlorodiphenyl (42% chlorine) (PCB)*	53469–21–9
2036 Chlorodiphenyl (54% chlorine) (PCB)*	11097–69–1
1088 beta-Chloroprene*	126–99–8
1091 Chlorpyrifos	2921–88–2
2042 Cresol, all isomers*	1319–77–3
2044 Cumene*	98–82–8
1107 Cyclohexanol	108–93–0
1108 Cyclohexanone	108–94–1
1110 Cyclonite	121–82–4
1113 DDT*	50–29–3
1114 Decaborane*	17702–41–9
2049 Demeton (Systox®)*	8065–48–3
1118 Diazinonn	333–41–5
1127 Dichloroethyl ether*	111–44–4
1129 1,3-Dichloropropene	542–75–6
2051 Dichlorvos (DDVP)*	62–73–7
1131 Dicrotophos (Bidrin)	141–66–2
2061 Dieldrin*	60–57–1
2062 2-Diethylaminoethanol*	100–37–8
2054 Diisopropylamine*	108–18–9
2064 Dimethyl acetamide*	127–19–5
1143 Dimethyl aniline (N-Dimethylaniline)*	121–69–7
1141 Dimethyl 1,2-dibromo-2,2-dichloroethyl phosphate	300–76–5
2067 Dimethyl formamide*	68–12–2
2068 1,1-Dimethylhydrazine*	57–14–7
1142 Dimethyl sulfate*	77–78–1
2071 Dinitrobenzene (all isomers)*	528–29–0
	99–65–0
	100–25–4
2070 Dinitro-o-cresol*	534–52–1
2072 Dinitrotoluene*	25321–14–6
1145 Dioxane (Diethylene dioxide)*	123–91–1
1146 Dioxathion (Delnav)	78–34–2

TABLE C17–1.—LIST OF SUBSTANCES FOR WHICH OSHA IS PROPOSING TO ADD SKIN DESIGNATIONS—Continued

H.S. number/chemical name	CAS No.
1149 Dipropylene glycol methyl ether*	34590–94–8
1152 Disulfoton	298–04–4
1156 Endosulfan	115–29–7
2073 Endrin*	72–20–8
1158 Epichlorohydrin*	106–89–8
2074 EPN*	2104–64–5
1160 Ethion (Nialate)	563–12–2
2-Ethoxyethanol*	110–80–5
2-Ethoxyethanol acetate (cellosolve acetate)*	111–15–9
1161 Ethyl acrylate*	140–88–5
1167 Ethylene chlorohydrin*	107–07–3
1170 Ethylene glycol dinitrate*	628–96–6
1172 N-Ethylmorpholine*	100–74–3
1173 Fenamiphos	22224–92–6
1175 Fenthion	55–38–9
1181 Fonofos	944–22–9
1183 Furfural*	98–01–1
1184 Furfuryl alcohol	98–00–0
2088 Heptachlor*	76–44–8
1197 Hexachloroethane*	67–72–1
2089 Hexachloronaphthalene*	1335–87–1
1198 Hexafluoroacetone	684–16–2
1205 Hydrazine*	302–01–2
1207 Hydrogen cyanide*	74–90–8
1211 2-Hydroxypropyl acrylate	999–61–1
1220 Isooctyl alcohol	26952–21–6
1222 Isophorone diisocyanate	4098–71–9
1229 N-Isopropylaniline	768–52–5
2100 Lindane*	58–89–9
1235 Malathion*	121–75–5
1237 Manganese cyclopentadienyl tricarbonyl	12079–65–1
1240 Mercury (aryl and inorganic compounds)*	7439–97–6
1241 Mercury (vapor)*	7439–97–6
1242 Mercury (organic), alkyl compounds*	7439–97–6
1244 Methacrylic acid	79–41–4
2105 Methyl acrylate*	96–33–3
1251 Methylacrylonitrile	126–98–7
1252 Methyl alcohol*	67–56–1
1253 Methyl bromide*	74–83–9
Methyl cellosolve (2-Methoxyethanol)*	109–86–4
Methyl cellosolve acetate (2-Methoxyethyl acetate)*	110–49–6
1270 o-Methylcyclohexanone*	583–60–8
1271 Methylcyclopentadienyl manganese tricarbonyl	12108–13–3
1256 Methyl demeton	8022–00–2
1273 4,4'-Methylene bis(2-chloroaniline)	101–14–4
2112 Methyl hydrazine (monomethyl hydrazine)*	60–34–4
1259 Methyl iodide*	74–88–4
1261 Methyl isobutyl carbinol*	108–11–2
2106 Methyl isocyanate*	624–83–9
1280 Monomethyl aniline*	100–61–8
1281 Morpholine*	110–91–8
2115 Nicotine*	54–11–5
1287 p-Nitroaniline*	100–01–6
2117 Nitrobenzene*	98–95–3
1288 p-Nitrochlorobenzene*	100–00–5
1290 Nitroglycerin*	55–63–0
1292 Nitrotoluene (all isomers)*	88–72–2
	99–08–1
	99–99–0
1295 Octachloronaphthalene*	2234–13–1
1303 Paraquat*	4685–14–7
2122 Parathion*	56–38–2
2123 Pentachlorophenol*	87–86–5
2126 Phenol*	108–95–2
1313 Phenothiazine	92–84–2
1317 Phenylhydrazine*	100–63–0
1319 Phorate (Thimet)	298–02–2

TABLE C17-1.—LIST OF SUBSTANCES FOR WHICH OSHA IS PROPOSING TO ADD SKIN DESIGNATIONS—Continued

H.S. number/chemical name		CAS No.
1320	Phosdrin (Mevinphos®)*	7786-34-7
1329	Picric acid*	88-89-1
1335	Propargyl alcohol	107-19-7
2135	Propyleneimine*	75-55-8
1364	Sodium azide	26628-22-8
1385	Sodium fluoroacetate*	62-74-8
2149	TEDP (Sulfotep)*	3689-24-5
2152	TEPP*	107-49-3
1253	1,1,2,2-Tetrachloroethane*	79-34-5
1255	Tetrachloronaphthalene	1335-88-2
1386	Tetraethyl lead (as Pb)*	78-00-2
1388	Tetramethyl lead (as Pb)*	75-74-1
2156	Tetramethylsuccinonitrile	3333-52-6
2158	Tetryl (2,4,6-Trinitro-phenyl-methyl-nitramine)*	479-45-8
2159	Thallium, soluble compounds (as Tl)*	7440-28-0
1392	Thioglycolic acid	68-11-1
1394	Tin (organic compounds)	7440-31-5
1399	o-Toluidine*	95-53-4
1400	p-Toluidine	106-49-0
1401	m-Toluidine	108-44-1
2162	1,1,2-Trichloroethane*	79-00-5
2163	Trichloronaphthalene*	1321-65-9
1413	2,4,6-Trinitrotoluene (TNT)*	118-96-7
1414	Triorthocresyl phosphate	78-30-8
1426	Vinyl cyclohexene dioxide	106-87-6
1432	m-Xylene-alpha,alpha'-diamine	1477-55-0
1433	Xylidine*	1300-73-8

* Skin designations for these substances already apply in the construction and maritime industry sectors. In these cases, OSHA is proposing to apply skin designations for these substances in the agricultural industry sector as well.

Biological Exposure Indices for 1987–1988 (ACGIH 1990) explains that the skin designation is designed to call attention to the need for "appropriate measures for the prevention of cutaneous absorption so that the threshold limit is not invalidated." Thus, a skin notation warns that exposure via the cutaneous route, including absorption through the eyes or mucous membranes, may contribute substantially to an employee's overall exposure and cause systemic toxicity.

The ACGIH has a policy of using a dermal LD_{50} of 2 g/kg as a general cutoff for determining when to classify a substance as sufficiently absorbable to present a hazard via the percutaneous route; that is, substances having a single-dose dermal LD_{50} of less than 2 g/kg receive a skin notation, while those with dermal LD_{50}s above this cutoff do not (ACGIH 1986/Ex. 1–3, p. 332). The Documentation (ACGIH 1986/Ex. 1–3) contains no cutoff value for chronic dermal exposures (i.e., for toxicity resulting from repeated applications of substances to the skin).

OSHA is proposing to include as paragraph 3[2] of the standard the following language regarding the use of skin notations:

An employee's skin exposure to substances listed in Table Z–1–A with the designation "Skin" following the substance name shall be prevented or reduced to the extent possible through the use of gloves, coveralls, goggles, or other appropriate personal protective equipment, engineering controls or work practices.

OSHA is not proposing to require that engineering controls be used preferentially to protect against skin absorption; the Agency notes that this policy is consistent with 29 CFR 1910.132 and 1910.134, which require the use of engineering controls and work practices in preference to personal protective equipment only when inhalation is the route of entry. The proposed regulatory language that appears here is the same as that adopted in the 1989 Air Contaminants rulemaking for general industry.

As explained in the previous rule, OSHA believes that dermal irritation alone should not warrant a skin designation; instead, OSHA believes that skin designations should be used only in instances where a substance can be percutaneously absorbed in quantities sufficient to cause systemic poisoning. OSHA's decision logic for establishing skin notations derives from the Agency's Hazard Communication Standard [29 CFR 1910.1200]. Appendix A of that regulation defines, in measurable terms, the possible health effects that may occur in the workplace as a result of chemical exposures. These definitions set forth quantitative guidelines for determining if chemicals are "highly toxic" or merely "toxic" by the dermal route of exposure. A chemical is considered *highly toxic* via skin absorption if

* * * [it] has a median lethal dose (LD_{50}) of 200 milligrams or less per kilogram of body weight when administered by continuous contact for 24 hours (or less if death occurs within 24 hours) with the bare skin of albino rabbits weighing between two and three kilograms each.

It is considered *toxic* via skin absorption if

* * * [it] has a median lethal dose (LD_{50}) of more than 200 milligrams per kilogram but not more than 1,000 milligrams per kilogram of body weight when administered by continuous contact for 24 hours (or less if death occurs within 24 hours) with the bare skin of albino rabbits weighing between two and three kilograms each.

Accordingly, OSHA has preliminarily determined that a skin notation is necessary for substances that have median lethal dose (LD_{50}) values in rabbits on single-dose applications of less than 1000 mg/kg. In addition, in very rare cases where available data (for any species) indicate that dermal contact results in a systemic dose that is equivalent to or greater than the dose that would be permitted by the PEL via inhalation, OSHA believes that a skin designation is warranted. In addition to this animal evidence, OSHA believes that the availability of human data demonstrating that systemic injury has occurred as a result of skin absorption is sufficient evidence that a skin notation is warranted. OSHA followed these guidelines in establishing skin designations in the recent rulemaking for general industry and has continued to observe them in the present rulemaking. Promulgation of these skin designations will thus make OSHA's skin designations consistent across all OSHA-regulated sectors.

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V. Preliminary Regulatory Impact Analyses

A. Executive Summary

Affected Industries

The proposed standard addresses employee exposures to airborne substances in the construction, agriculture, and maritime industries. It should be noted that the use of EPA

labelled pesticides pursuant to the label is regulated by the Environmental Protection Agency and therefore is not covered by this standard. Proposed PELs for asphalt fume, fibrous glass, and mineral wool would cover exposures to these substances in all industry sectors.

Activities with potential exposures are common in the construction industry; most firms in SICs 15, 16, and 17 would be affected to some degree. In the maritime industry, the proposed standard would affect establishments involved in shipbuilding and repair and in cargo handling operations. In the agriculture industry, firms likely to be affected include those engaged in silo operations, cotton ginning, and grape harvesting. Potentially affected firms in general industry would include those producing and using fibrous glass and mineral wool, asphalt, paving mixtures, and asphalt roofing products.

Employee Exposures and Benefits

In the construction industry, activities involving potential exposures above the proposed PELs include painting; concrete sealing and coating; abrasive blasting; sanding; batch mixing of concrete, mortar or cement; and burning, cutting, grinding and welding on metal or painted surfaces. Compliance with the proposed standards would protect construction employees from excess exposure to many substances, including lead, petroleum distillates, ethylene glycol, xylene, toluene, butoxyethanol, hexylene glycol, mercury compounds, titanium dioxide, cement dust, and silica.

In the maritime industry lower exposure levels should benefit employees engaged in welding, painting, and bulk cargo handling exposed to metal fumes, solvents, and various dusts. Tank cleaning operations also involve potential exposures to many of the chemicals in the proposed regulation.

In the agriculture industry, employee exposures above the proposed PELs occur during silo operations, grain handling, cotton ginning, and grape harvesting. These operations can involve exposures to grain dust, cotton dust, nuisance dust, or silica.

In general industry, compliance with the proposed regulation would result in lower occupational exposures to asphalt, fibrous glass, RCF, and mineral wool. Employees are exposed to these substances during the production of asphalt and vitreous fibers and during the use or manufacture of products containing these substances.

OSHA estimates that full compliance with the proposed standard across all affected industries would potentially prevent about 8 to 13 fatalities and about 31,000 illnesses (including about 12,000 illnesses involving lost workdays) annually. The primary impact would be in the construction industry, with approximately 7 to 11 fatalities and 21,600 illnesses (including 10,200 illnesses involving lost workdays) potentially prevented each year. In the maritime industry, an estimated 1 to 2 fatalities and 200 illnesses would be avoided each year (including 60 illnesses involving lost workdays); in agriculture full compliance may prevent about 9,000 illnesses annually (including 2,000 illnesses involving lost workdays); and in general industry less than one fatality and about 500 illnesses (including 100 illnesses involving lost workdays) would be avoided annually.

Technological Feasibility and Costs of Compliance

The proposed standard requires employees to be protected against overexposure to regulated substances. The estimated costs of compliance for the proposed rule reflect the incremental costs necessary to reduce exposure levels below the proposed PELs for all employees.

The activity-based approach used to determine exposures and the need for additional controls in construction allowed for explicit consideration of mixed exposures. Exposures to multiple substances were evaluated according to the formula in the proposed regulation; combined exposures exceeding unity were considered to be exposures above the proposed PELs. As a result, the costing methodology included allowances for meeting the mixed exposure limit (MEL) as well as individual PELs.

In many activities engineering controls and work practices will effectively reduce airborne concentrations of hazardous substances. These may include fans, fresh air supply blowers, portable exhaust hoods, enclosures, equipment-mounted dust catchers, vacuum cleaning, and water spraying. In activities for which feasible engineering controls are unavailable or insufficient, respiratory protection would be required. An evaluation of the activities potentially affected by this rulemaking has established that compliance with the proposed regulation can be achieved in all industries through an appropriate combination of engineering controls, work practices, and respiratory protection.

The total estimated annual costs of achieving full compliance with the proposed standard in all industries with the proposed PEL of 5 mg/m^3 for asphalt fume is approximately $103 million. The annual cost for the construction industry would be about $94 million, the annual cost for the maritime industry would be about $5.7 million, the annual cost for the agriculture industry would be about $2.7 million, and the annual cost for general industry would be about $0.6 million.

With a PEL for asphalt fume of 0.2 mg/m^3 the total cost would be approximately $163 million. Compliance costs for the construction industry would be about $51 million higher, and compliance costs in general industry would be about $9 million higher.

In the construction industry, the highest costs are associated with painting, welding, and paint removal, which are concentrated in SIC 1721 (Painting and Paper Hanging), and 1799 (Special Trade Contractors, Not Elsewhere Classified). Respiratory protection accounts for the largest share of compliance costs at $54 million with an asphalt PEL of 5 mg/m^3.

In the maritime industry, most of the compliance costs are associated with painting and welding activities in the shipbuilding and repair industry. In the agriculture industry, the estimated costs of compliance are concentrated in the cotton ginning sector. In general industry the compliance costs are primarily attributable to the proposed PELs for fibers.

Economic Impacts

The costs associated with compliance with the proposed standard would represent less than 0.1 percent of the revenues of the affected industries. The proposed PELs generally would not require drastic or widespread changes. Many employers who currently utilize sound industrial hygiene practices in activities with potential exposures would already be in compliance with the proposed standard.

The overall impact of compliance with the proposed requirements would be an increase in efforts to control employee exposures during activities with recognized potential hazards. The costs would generally be incurred on a per employee basis and on average would represent an increase of less than 0.1 percent of labor costs. These costs should be bearable for all firms and would not create any significant competitive disadvantage for small establishments.

Economic impacts in the maritime and agriculture industries are expected to be minimal. The extent of exposures and necessary control measures would be limited, and the costs of compliance would not represent a substantial burden. In general industry some

additional costs for employee protection would have to be incurred during the production and use of vitreous fibers. The proposed PELs for fibers and for asphalt fume are not expected to produce any significant negative impacts.

B. Preliminary Regulatory Impact Analysis for Construction

1. Summary

This section examines the technological feasibility, costs, benefits, and economic impacts associated with this rulemaking for the construction industry. In addition, an industry profile and an evaluation of nonregulatory alternatives is provided. This chapter is divided into seven parts. Part 1, this summary, briefly introduces the other six parts.

Part 2 provides descriptive statistics about the construction industry and each of its subindustries. The industry employs 5 million workers who construct a wide variety of projects including housing, industrial buildings, roads, and power plants. Each project may employ a combination of contractors and subcontractors that specialize in tasks such as carpentry, painting, or roofing.

Part 3 describes the extent of worker exposures to the substances considered in this rulemaking. Exposures to solvents used in paints and adhesives occur during painting activities, floor work operations, and during the application of single-ply membrane roofing systems. Exposures to lead and other metals can occur when working on painted surfaces, and when welding or soldering. Silica and other particulates may be present during any dusty operation such as abrasive blasting, cement mixing, or earthmoving. Paving and roofing work involve exposures to asphalt fumes. Dozens of other activities, causing significant exposures to over 80 different substances, have been identified.

Virtually all 5 million workers in the construction industry are potentially exposed to one or more of the substances in this proposed rule. Exposures may be intermittent, infrequent, and below the proposed limits, but potential overexposures are not uncommon. OSHA estimates that eliminating all worker exposures above the proposed PELs would result in the prevention of an estimated 7–11 fatalities, 10,200 illnesses involving 214,000 lost workdays, and 11,400 non-lost workday illnesses annually.

Part 4 discusses alternatives to this rulemaking and provides the basis for the conclusion that this proposed regulation is necessary and cost-effective.

Part 5 describes specific activities involving exposures and determines the technological feasibility of meeting the proposed PELs. Engineering controls are available to reduce some exposures but these are not always applicable for all activities. As currently practiced in the construction industry, respiratory protection may be needed to achieve compliance in some high exposure situations. Compliance with the proposed PELs would require an increase in respirator usage but the overall utilization rate would remain relatively low. With an alternative PEL of 0.2 mg/m^3 for asphalt fume, a significant increase in respirator use would be necessary for asphalt roofing and paving operations.

Part 6 presents the estimated costs of compliance. Total annualized compliance costs would be about $94 million with a PEL for asphalt of 5 mg/m^3. The highest costs are associated with painting and welding, which are concentrated in SIC 1721 (Painting and Paper Hanging) and SIC 1799 (Special Trade Contractors, Not Elsewhere Classified). The majority of the compliance costs by type of control is for respiratory protection at $54 million. A PEL of 0.2 mg/m^3 for asphalt would increase total estimated compliance costs by about $51 million, primarily for respiratory protection during asphalt roofing and paving work.

Part 7 assesses the economic impacts of the proposed rule. Costs of compliance are small compared to average revenues for all SIC groups. Overall demand for construction is not expected to be affected because the increased cost of projects would be minimal. Slight cost increases may result for some painting and other activities as the added cost of worker protection is included in the cost of completing the work. The rule would apply to all work done within the United States, and thus international competitiveness would not be directly affected.

2. Industry Profile

Overview. The construction industry is covered by three two-digit Standard Industrial Classification (SIC) codes: SIC 15, Building Construction, General Contractors; SIC 16, Heavy Construction Except Buildings, General Contractors; and SIC 17, Special Trade Contractors. The term construction includes new work, additions, alterations, reconstruction, installations, and repairs.

Special trade contractors are primarily engaged in specialized construction activities, such as plumbing, painting, and electrical work, and work for general contractors under subcontract or directly for property owners. General contractors usually assume responsibility for an entire construction project, but may subcontract to others all of the actual construction work or those portions of the project that require special skills or equipment. General contractors thus may or may not have construction workers on their payroll.

The most recent detailed data on the construction industry are found in the 1987 Census of Construction Industries, issued by the U.S. Department of Commerce in March, 1990. During 1987, establishments with paid employees accounted for $500 billion in total value of construction business. Their payments for construction work subcontracted to others amounted to $132 billion, leaving a net value of construction work of $368 billion. In addition, these establishments paid out $138 billion for the cost of materials, fuels, power, rental of equipment and buildings, and other purchased services. Value added for the construction industry in 1987 was estimated to be $230 billion.

In 1987 there were approximately 540,000 establishments in the construction industry employing a total of over 5 million people. According to the Census of Construction, the total payroll for the construction industry in 1987 was $111 billion. The total hours worked by construction workers during the year were estimated to be 6.7 billion hours.

Residential Building Construction. Residential building construction accounts for over 30 percent of the total value of construction [1]. This category includes SIC 1521, General Building Contractors—Single-Family Houses; SIC 1522, General Building Contractors—Other Than Single-Family; and SIC 1531, Operative Builders. Operative builders are primarily engaged in the construction of single-family houses and other buildings for sale on their own account rather than as contractors.

The residential building construction industry has experienced tremendous growth over the past decade. This is partly due to the substantial decline in mortgage rates since the early 1980s, from a high of 15.14% in 1982 to a low of 9.29% in 1988 [2]. Because of high real interest rates from 1979 to 1982, there was substantial pent-up demand for housing when interest rates began to fall (making the cost of housing more affordable).

The increase in the nominal price of housing due to high inflation rates at the

beginning of the 1980s helped many second-time home buyers, who were able to take advantage of increased equity available in their homes. The Department of Commerce estimates that the cost of construction index (1982 = 100) rose from 63.6 in 1977 to 102.6 in 1983. The first-time home buyer was helped by the decline in interest rates, but hurt by the increase in home prices over this same period. The average sale prices of new homes sold in the United States increased from $89,800 in 1983 to $138,200 in 1988, an increase of 53.9 percent [2]. The improved performance of the economy during this time period led to an increase in residential building activity, due to the increase in real personal income that occurred over this time period, from $9,725 per capita in 1982 to $11,012 per capita in 1987 [3].

However, in 1989, private residential construction decreased by about one percent in constant dollar value. Housing starts fell six percent, from 1.49 million units in 1988 to 1.40 million units in 1989, with declines both in single- and multi-unit structures. While interest rates increased during the first part of 1989, continuing a trend established in 1988, by mid-year of 1989, mortgage rates declined to near 10 percent during the third quarter where they remained through the end of 1989. Mortgage interest rates averaged 10.3 percent during 1989.

Rising interest rates in early 1989 kept new home sales 6.4 percent below 1988 levels, although the sales volume of existing homes increased slightly. Between 1982 and 1989, median new home prices rose 61 percent, while disposable income per capita (in current dollars) increased by about 50 percent. While the decline in interest rates offset rising home prices between 1983 and 1987, both housing prices and interest rates rose in 1988 and early 1989 [4]. As of August 1990, home sales were 10 percent below their level of the previous year.

An area that is likely to grow within the residential building industry is the residential remodeling market. Most home builders that have added remodeling to their line of services have done so because remodeling is less cyclical than new building construction. Last year the top five remodeling markets were Los Angeles/Long Beach, followed by Washington, D.C., New York, Chicago, and San Francisco. From 1984 to 1987, total residential improvements increased by 37.1 percent [5]. However, in 1989, home owners only spent 1.3 percent more than the previous year for upkeep and improvement of residential properties compared to the 7.7 percent increase in 1988. The slower growth in 1989 reflected the country's slower economic growth, higher interest rates, and weaker consumer confidence levels [4].

Residential construction also includes spending for hotels and motels, which represented about $9 billion of the total value of construction in 1987 [1]. It is generally believed that the hotel market has become somewhat overbuilt in the 1980s, and that further construction will occur at a slower pace until some of the excess capacity is absorbed [6]. A new trend in the hotel construction market is a move toward specialization in order to capture a certain market sector, such as business travelers. This may provide for some new hotel construction in certain geographic locations [7].

In 1989, construction of hotels and motels increased substantially over prior years, reflecting an international hotel-building boom. That boom has probably peaked in the United States and a 10 percent decline is likely in 1990 and continuing in 1991 [4].

According to the Census of Construction [1], there were almost 120,000 establishments classified as general contractors or operative builders in residential building construction in 1987. They employed over 600,000 workers and accounted for over $100 billion of the total value of construction work. These figures do not include residential construction work done by special trade contractors. Statistics for each of the 4-digit residential construction SIC codes are presented in Table V–B1.

TABLE V–B1.—RESIDENTIAL CONSTRUCTION

[General Contractors and Operative Builders]

	SIC 1521 [1]	SIC 1522 [2]	SIC 1531 [3]
Number of Establishments	90,376	8,143	20,756
Total Employees	398,291	81,708	168,940
Construction Workers	307,305	61,245	76,105
Construction Workers Hours (Thousands)	470,499	95,455	135,927
Net Value of Construction Work (Thousands of Dollars) [4]	27,319,239	6,257,443	26,837,792
Total Value of Construction Work (Thousands of Dollars)	35,098,146	13,315,544	48,950,839
Single-Family Houses	31,833,307	793,845	42,401,837
Apartment Buildings	638,197	6,337,325	3,174,608
Other Residential	345,309	3,496,516	152,609
Other Construction	6,281,333	2,685,858	3,230,755

[1] General Contractors—Single Family Houses
[2] General Contractors—Residential Buildings Other Than Single-Family.
[3] Operative Builders.
[4] Excludes value of work subcontracted.

Source: Office of Regulatory Analysis, OSHA, U.S. Department of Labor, based on 1987 Census of Construction, U.S. Department of Commerce, March 1990.

Non-Residential Building Construction. The non-residential building industry is divided into two sectors: Industrial buildings and warehouses (SIC 1541) and non-residential buildings, other than industrial buildings and warehouses (SIC 1542). SIC 1541 is primarily made up of manufacturing facilities and warehouses. SIC 1542 consists primarily of commercial, recreational, hospital, religious, and office buildings.

The non-residential building construction industry is dependent upon the economic environment in which it operates. Factors such as interest rates, manufacturing productivity, currency exchange rates, capital expenditures, labor costs, and the stock market all influence the market for non-residential building construction.

The demand for manufacturing facilities in the future will depend upon the trading position of the United States in manufactured goods. If the manufacturing sector in the United States continues to expand, the demand

for new manufacturing facilities (by both domestic and foreign producers) should increase significantly. This increased demand could occur because of one or all of the following reasons: High levels of capacity utilization, increased exports due to the fall in the value of the dollar relative to Japanese and European currencies, and relatively high labor costs overseas. Capacity utilization figures for the manufacturing industry have increased dramatically since 1982, from 70.3 percent in 1982 to 81.1 percent in 1987 [3]; industry experts generally consider 85 to 90 percent to be the viable maximum.

Value of industrial construction increased more than five percent in 1989 in constant dollar value. Further increases in industrial construction are expected because many of the manufacturing industries are operating at a high capacity-utilization rate.

International competition has been constraining the need for United States industries to construct new plants in the past but if the trade deficit continues to be reduced by strong United States exports, industrial construction would benefit from the increased demand. Although the long-term outlook for industrial construction is subject to many uncertainties, it is likely to be one of the stronger construction markets during the next five years [4].

New spending for office building construction has declined every year since 1985, when it reached a high of $31.6 billion. In 1988, office building construction spending was $27.4 billion. Construction of office space is heavily dependent upon the growth and structure of employment. Recently, there has been a decline in employment growth for large corporations (greater than 500 employees) but an increase in employment growth for small firms (less than 100 employees). This change in the structure of employment is likely to have a slight positive effect on spending for office buildings, especially in suburban areas [6].

Overall, new office construction continued to decline in 1989 and 1990 and further declines are expected over the next years due to higher vacancy rates and the elimination of many of the tax benefits of commercial buildings. Demand for office construction in the future will be heavily influenced by the need to absorb vacant office space and the need to raise rents to profitable levels. Other important demand factors will be long-run investment in real estate by pension funds and foreign interests [4].

The commercial building sector primarily includes retail shopping, restaurants, and other retail ventures. During the 1980s, retail construction spending increased by 4.0 percent annually. This rate of growth (much lower than spending growth for offices or hotels) permitted modest expansion without overbuilding in the retail sector. Retail construction is dependent on the housing market which was strong in the 1980s, increasing demand for retail space. Generally, the demand for retail space will lag a change in the housing market by six months to two years.

The relative value of commercial buildings such as shopping centers, warehouses, parking garages, banks, and restaurants declined in 1989 and 1990. The primary causes for the decline are, as in other sectors, overcapacity and the effects of a general decline in the growth rate of the economy. However, the overbuilding problem is not as serious for stores and shopping centers as it is for office buildings and hotels [4].

The commercial building renovation market is expanding as commercial owners face an oversupply of new buildings and industrial owners rein in outlays for new plants. Governments are also renovating an abundance of deteriorating facilities. There has been a steady rise in renovation work over the past two decades. In 1970, contracts for additions and renovations made up only about 16 percent of the non-residential building market but had climbed to 22 percent by 1980 and to 28 percent by 1988. Also, the trend by industrial owners to put the largest share of their capital spending into renovation has intensified in recent years. Today, some of the largest renovation projects can be found in industrial processes and in petroleum refinery markets [8].

General contractors in non-residential building construction (SIC 1541 and SIC 1542) included over 38,000 establishments in 1987. These establishments employed over 600,000 workers. Non-residential construction work done by special trade contractors is not included in these figures. Mean establishment size in SIC 1541 was 20 employees per firm and 16 employees per firm in SIC 1542. Table V–B2 provides a summary of statistics for each nonresidential building construction SIC code.

Total value of non-residential building business done in 1987 was $111 billion, an increase of 56 percent from the 1982 figure of $71 billion. Ownership of these projects was divided between the federal government ($7 billion), state and local governments ($15 billion), and private owners ($89 billion). The five largest sectors within non-residential building construction were the following: office buildings—$28 billion; industrial buildings—$23 billion; other commercial buildings—$22 billion; hospital buildings—$10 billion; and educational buildings—$8 billion. Net value of construction work for SIC 1541 and SIC 1542 combined was over $50 billion in 1987.

TABLE V–B2.—NONRESIDENTIAL BUILDING CONSTRUCTION

[General Contractors]

	SIC 1541 [1]	SIC 1542 [2]
Number of Establishments	7,014	31,337
Total Employees	143,001	488,480
Construction Workers	110,785	342,442
Construction Worker Hours (thousands)	195,425	633,554
Net Value of Construction Work (thosands of dollars) [3]	11,094,502	39,510,241
Total Value of Construction Work (Thousands of Dollars)	21,461,568	89,793,431
Office Buildings	1,688,344	26,438,662
Other Commercial Buildings	1,098,192	20,503,267
Industrial Buildings	15,777,425	7,243,757
Religious Buildings	D	1,975,064
Educational Buildings	358,045	8,048,930
Hospitals	440,284	9,935,109
Other Construction	2,099,278	15,648,642

[1] General Contractors—Industrial Buildings and Warehouses.

[2] General Contractors—Nonresidential Buildings, Other Than SIC 1541.

[3] Excludes value of work subscontracted.

Source: Office of Regulatory Analysis, OSHA, U.S. Department of Labor, based on 1987 Census of Construction, U.S. Department of Commerce, March 1990.

Highway and street construction, except elevated highways. Highway and street construction (SIC 1611) has been a large, steady component of public construction expenditures in the 1980s. While state and local government spending on transportation systems has increased steadily throughout this decade, federal government spending has decreased slightly. Because the majority of public spending expenditures on highway and street construction come from state and local governments, the outlook for this industry sector is dependent upon the budgetary future of state and local governments.

Several states that previously have had large resources to commit to new highway projects are no longer able to sustain this level of spending because of budgetary difficulties. The state of Florida, which has spent large amounts on such infrastructure development in the past, is facing severe cash shortages in the Department of Transportation [6]. This has led to a substantial decline in highway and street construction spending. Other states, such as Texas and Massachusetts, are facing similar

situations due to a decline in tax revenues and high debt payments. The decline in highway and street spending by these states has been offset by increased spending by several states such as North Carolina, Kansas, and California [9].

Table V–B3 shows a summary of statistics for highway and street construction in the United States. In 1987, there were approximately 11,000 establishments in the highway and street construction business (excluding special trade contractors in SIC 17) with 284,000 employees, of whom approximately 239,000 were construction workers. Mean establishment size was 26 workers per firm that year. The value of construction business done in this industry was over $34 billion in 1987, an increase of over $14 billion from 1982.

TABLE V–B3.—HIGHWAY AND STREET CONSTRUCTION, EXCEPT ELEVATED HIGHWAYS

	SIC 1611 [1]
Number of Establishments	10,986
Total Employees	284,380
Construction Workers	239,111
Construction Worker Hours (Thousands)	439,016
Net Value of Construction Work (Thousands of Dollars) [2]	27,983,839
Total Value of Construction Work (Thousands of Dollars)	34,161,427
Highways, Streets	28,123,431
Parking Areas	607,665
Recreational Facilities	183,363
Bridges and Tunnels	1,152,276
Dams and Reservoirs	185,273
Marine Construction	50,755
Sewers and Water Mains	1,112,314
Power Plants	177,092
Other Construction	2,531,228

[1] Highway and Street Construction, Except Elevated Highways.
[2] Excludes value of work subcontracted.

Source: Office of Regulatory Analysis, OSHA, U.S. Department of Labor, based on 1987 Census of Construction, U.S. Department of Commerce, March 1992.

Heavy Construction, Except Highway and Street Construction. This industry category consists of three subcategories: Bridge, Tunnel and Elevated Highway Construction (SIC 1622); Water, Sewer, Pipeline, Communications and Power Line Construction (SIC 1623); and Heavy Construction, Not Elsewhere Classified (SIC 1629).

New construction of bridges, tunnels and elevated highways is highly dependent upon the amount of funding available from state and local government sources. A large percentage of spending on roads and bridges is for maintenance of current infrastructure, an area in which there may be less freedom to decrease spending due to safety concerns. For the first nine months of 1989, total contract awards for bridges and tunnels were $4.5 billion, an increase of 11.0 percent over the first nine months of 1988 [6].

In 1989, 25 percent of value of highway construction consisted of work completed on bridges, overpasses, and tunnels; the other 75 percent consisted of flat work. Bridge work is expected to grow faster than flat work during the next several years because of the need to replace obsolete or unsafe bridges. According to the Federal Highway Administration, 23 percent of the highway bridges in the U.S. in 1989 were structurally deficient and an additional 21 percent were functionally or structurally obsolete.

In 1989, the cost for maintenance and repair work was $22 billion versus $29 billion for new highway construction. While some of the maintenance costs were for routine maintenance such as mowing grass, the largest fraction was spent on repaving roads and repainting bridges [6]. In 1983, there were 259,950 bridges in the Federal-aided highway system and 313,700 in the non-Federal-aided system.

In early 1990, Democratic and Republican leaders of the House of Representatives' Public Works and Transportation Committee began a review of U.S. infrastructure needs in order to develop legislation "to rebuild America." As the country plans for and pursues the huge job of upgrading the infrastructure, an increasing percent of expenditures dedicated to reconstruction, repair and maintenance, especially of bridges, can be expected.

Construction of water, sewer, and power lines (SIC 1623) reflects the expansion of the population and of residential areas. As residential construction increased in the 1980s, construction of these basic services accompanied the new developments. Work in this industry is also dependent on the regulatory atmosphere regarding communication, power, and gas lines. New projects continue to be initiated as demands for water, energy and communications links continues to grow.

SIC 1629 (Heavy Construction, Not Elsewhere Classified) includes activities such as athletic field construction, brush clearing or cutting, dam construction, flood control project construction, land reclamation, marine construction, railroad construction, subway construction, and waste disposal plant construction. Because of the varied nature of these activities, it is difficult to predict the outlook for this industry sector. The overall performance should continue to reflect the general level of activity in the economy as a whole.

Table V–B4 presents summary data about heavy construction, except highway and street construction (SICs 1622, 1623, and 1629). As of 1987, there were approximately 26,000 establishments in these industries, an increase of 8,000 establishments since 1982. The establishments employed approximately 543,000 workers, of whom 444,000 were construction workers. Mean establishment size was 21 employees per firm.

Total value of construction business done in this category in 1987 was over $48 billion. The five largest categories in the heavy construction industry are the following: sewers and water mains—$9.6 billion; bridges and tunnels—$5 billion; blast furnaces—$4.1 billion; sewage treatment plants—$3.5 billion; and power and communication lines—$3.7 billion. The net value of construction work for SIC 1622, SIC 1623, and SIC 1629 was over $40 billion in 1987.

TABLE V–B4.—HEAVY CONSTRUCTION, EXCEPT HIGHWAY AND STREET CONSTRUCTION

	SIC 1622 [1]	SIC 1623 [2]	SIC 1629 [3]
Number of Establishments	1,159	9,919	14,532
Total Employees	47,494	197,632	297,618
Construction Workers	40,092	165,879	238,204
Construction Worker Hours (Thousands)	76,210	309,168	420,660
Net Value of Construction Work (Thousands of Dollars) [4]	4,186,846	15,055,297	21,209,274
Total Value of Construction Work (Thousands of Dollars)	5,480,936	17,010,019	25,632,969
Bridges and Tunnels	4,476,501	127,899	532,104
Marine Construction	29,291	0	1,379,611

TABLE V–B4.—HEAVY CONSTRUCTION, EXCEPT HIGHWAY AND STREET CONSTRUCTION—Continued

	SIC 1622 [1]	SIC 1623 [2]	SIC 1629 [3]
Power and Communication Lines	0	3,473,127	274,727
Sewers and Water Mains	93,450	8,571,756	690,373
Pipelines	0	2,568,987	249,086
Sewage Treatment Plants	113,394	329,756	3,136,903
Other Construction	766,300	1,908,884	19,078,165

[1] Bridge, Tunnel and Elevated Highway Construction.
[2] Water, Sewer, Pipeline, Communications and Power Line Construction.
[3] Heavy Construction, Not Elsewhere Classified.
[4] Excludes value of work subcontracted.
Source: Office of Regulatory Analysis, OSHA, U.S. Department of Labor, based on 1987 Census of Construction, U.S. Department of Commerce, March 1990.

Special Trade Contractors. Establishments in SIC 17 usually specialize in a specific type of construction work, often based on a specific trade. Special trade contractors may work directly for an owner of a project or may work under subcontract to a general contractor, depending on the type of contracting system used. Work is generally performed at the site of construction, but some special trade contractors may also have shops where they perform work incidental to the job site.

The traditional and most common project delivery system in the construction industry is the general contracting system. Under this system the owner of a construction project, who might be an individual, a private corporation, or a government agency, awards the contract to build the project to a general contractor, also commonly called a prime contractor.

The general contractor's prime function is to assemble the specialty contractors and suppliers needed to construct the project according to the plans and specifications. While a general contractor often performs some of the work on the project with his own employees, much of the work may be performed by subcontractors because the prime contractor might not have the expertise, equipment or the trained work force to perform the work. Some of the work may also be subcontracted in order to reduce the general contractor's risk.

Regardless of how much or how little of the construction work is subcontracted, the prime contractor has the responsibility for coordinating, scheduling, and monitoring all of the work done on the construction site. Thus, the subcontractors are dependent both on the general contractor and the owner of the project for the approval of their work.

Tables V–B5 through V–B7 provide summary statistics on each of the special trade contractor categories.

General divisions of special trade contractor activities include: Plumbing, heating and air conditioning work (SIC 1711); painting and paper hanging (SIC 1721); electrical work (SIC 1731); masonry, stone setting, and other stone work (SIC 1741); plastering, drywall, acoustical, and insulation work (SIC 1742); terrazzo, tile, marble, and mosaic work (SIC 1743); carpentry work (SIC 1751); floor laying and other floor work, not elsewhere classified (SIC 1752); roofing, siding, and sheet metal work (SIC 1761); concrete work (SIC 1771); water well drilling (SIC 1781); structural steel erection (SIC 1791); glass and glazing work (SIC 1793); excavation work (SIC 1794); wrecking and demolition work (SIC 1795); installation or erection of building equipment, not elsewhere classified (SIC 1796); and special trade contractors, not elsewhere classified (SIC 1799).

TABLE V–B5.—SPECIAL TRADE CONTRACTORS, SIC GROUPS 1711–1743

	SIC 1711 [1]	SIC 1721 [2]	SIC 1731 [3]	SIC 1741 [4]	SIC 1742 [5]	SIC 1743 [6]
Number of Establishments	89,586	29,857	49,436	23,284	17,809	5,089
Total Employees	617,333	169,968	509,309	168,978	253,563	34,420
Construction Workers	470,793	145,385	405,981	150,308	217,392	27,906
Construction Worker Hours (Thousands)	844,750	223,559	732,100	221,510	350,749	44,916
Net Value of Construction Work (Thousands of Dollars) [*]	44,517,739	7,445,552	34,657,765	8,260,188	15,137,323	2,181,972
Total Value of Construction Work (Thousands of Dollars)	49,503,323	7,953,323	35,836,226	8,714,161	16,426,850	2,271,593
Single-Family Houses	11,394,480	1,929,573	4,164,610	2,738,184	4,486,565	652,962
Office Buildings	6,541,994	1,105,840	7,127,066	1,100,895	2,783,200	325,723
Industrial Buildings	7,117,202	868,916	6,546,566	1,063,764	1,294,473	113,849
Other Building Construction	20,805,493	2,952,507	12,873,331	3,432,954	6,302,394	935,079
Nonbuilding Construction	3,644,154	976,587	5,124,653	378,364	560,215	44,080

[1] Plumbing, Heating and Air Conditioning.
[2] Painting and Paper Hanging.
[3] Electrical Work.
[4] Masonry, Stone Setting, and Other Stone Work.
[5] Plastering, Drywall, Acoustical, and Insulation Work.
[6] Terrazzo, Tile, Marble, and Mosaic Work.
[*] Excludes value of work subcontracted.
Source: Office of Regulatory Analysis, OSHA, U.S. Department of Labor.

TABLE V–B6.—SPECIAL TRADE CONTRACTORS SIC GROUPS 1751–1781

	SIC 1751 [7]	SIC 1752 [8]	SIC 1761 [9]	SIC 1771 [10]	SIC 1781 [11]
Number of Establishments	36,009	8,174	25,673	23,422	3,414
Total Employees	190,431	44,579	231,137	218,194	17,598
Construction Workers	164,191	34,666	186,916	186,840	13,628
Construction Worker Hours (Thousands)	252,984	55,208	293,990	297,487	25,724
Net Value of Construction Work (Thousands of Dollars)*	10,038,947	3,371,200	14,182,802	13,853,510	1,299,288
Total Value of Construction Work (Thousands of Dollars)	11,243,863	3,651,435	15,027,806	15,055,670	1,330,056
Single-Family Houses	6,024,429	1,327,792	4,000,304	3,402,452	428,005
Office Buildings	815,411	702,996	1,644,814	1,340,326	11,425
Industrial Buildings	603,072	149,642	3,370,775	1,458,714	26,704
Other Building Construction	3,672,316	1,441,540	5,794,467	3,766,921	253,331
Nonbuilding Construction	128,635	29,465	217,446	5,087,257	610,591

[7] Carpentry Work.
[8] Floor Laying and Other Floor Work, not Elswhere Classified.
[9] Roofing, Siding, and Sheet Metal Work.
[10] Concrete Work.
[11] Water Well Drilling.
*Excludes value of work subcontracted.
Source: Office of Regulatory Analysis, OSHA, U.S. Department of Labor.

TABLE V–B7.—SPECIAL TRADE CONTRACTORS SIC GROUPS 1791–1799

	SIC 1791 [12]	SIC 1793 [13]	SIC 1794 [14]	SIC 1795 [15]	SIC 1796 [16]	SIC 1799 [17]
Number of Establishments	4,017	4,636	13,422	1,240	3,777	23,198
Total Employees	65,348	40,511	95,329	14,109	62,151	176,084
Construction Workers	54,729	28,730	79,198	11,686	50,244	141,615
Construction Worker Hours (Thousands)	89,851	49,500	137,093	17,545	91,269	221,784
New Value of Construction Work (Thousands of Dollars) *	4,510,231	3,142,354	7,490,988	844,714	5,009,764	9,832,759
Total Value of Construction Work (Thousands of Dollars)	4,862,655	3,222,472	8,244,398	912,484	5,359,826	10,814,313
Single-Family Houses	68,448	396,351	2,112,521	60,203	45,557	767,914
Office Buildings	930,225	927,580	658,168	92,140	1,356,025	937,438
Industrial Buildings	1,173,121	206,157	611,783	155,531	1,565,427	834,379
Other Building Construction	1,664,693	1,650,389	3,171,494	401,178	1,937,399	4,110,221
Nonbuilding Construction	1,026,168	41,995	1,690,432	203,432	455,418	4,164,361

[12] Structural Steel Erection.
[13] Glass and Glazing Work.
[14] Excavation Work.
[15] Wrecking and Demolition Work.
[16] Installation or Erection of Building Equipment, Not Elsewhere Classified.
[17] Special Trade Contractors, Not Elsewhere Classified.
* Excludes value of work subcontracted.
Source: Office of Regulatory Analysis, OSHA, U.S. Department of Labor.

According to the U.S. Department of Commerce's most recent census of the construction industry, there were approximately 342,000 establishments of special trade contractors (SIC 17) which employed 2.9 million workers in 1987, of whom more than 2.3 million were construction workers. Overall, special trade contractors did over $200 billion in total business volume and net value of construction work was over $185 billion. A discussion of special trade contractors by individual four-digit SIC follows.

Plumbing, Heating, and Air Conditioning. Table V–B5 summarizes market structure and trends of SIC 171 through SIC 174. Industry sector SIC 1711 consists of plumbing, heating, and air conditioning contractors.

The optimistic contracting picture many mechanical contractors in SIC 1711 enjoyed in 1989 changed rapidly because of the major slowdown in commercial building. However, jobs like retooling old plants and building efficient ones to help manufacturers lower costs and compete better have shown modest gains. For the 179 firms responding to a 1990 survey of mechanical contractors by Engineering News Record (ENR), 39 percent of new contracts were for the commercial building market, 30 percent for industrial or power markets, 19 percent for the institutional market, and 16 percent for the renovation market. The remainder of revenues came from the water and sewer markets (7 percent), transportation and residential markets (each 2 percent), and other markets (1 percent). Energy-related mechanical work has good prospects because of the crisis in the Middle East, with much of the new work involving alternative sources of energy [10].

The latest economic census conducted by the U.S. Department of Commerce, Bureau of the Census, indicates that in 1987 there were approximately 70,000 establishments in plumbing, heating and air conditioning contracting (SIC 1711), which is an increase of over 15,000 firms since 1982. These firms employed more than 600,000 workers of whom 470,000 were construction workers. This industry segment did over $49 billion in construction business in 1987, up from $33.5 billion in 1982. Net value of construction work was close to $45 billion in 1987, $15 billion more than in 1982 [1].

Painting and Paperhanging. Competition among painting contractors increased during 1989 as a downturn in commercial construction forced some firms to cut prices in order to stay in business. Dun & Bradstreet reported that industry failures were up in 1988, climbing from 303 failed firms in 1987 to 327 in 1988. Most of the larger painting contractors performed commercial (41.9

percent), industrial (28 percent), and institutional (10.8 percent) work in 1989, while many of the small firms performed residential painting [11].

The painting contractor industry has become increasingly sophisticated and capital costs of equipping each worker for industrial painting can be as high as $500. Much of these costs go into sandblasting equipment, blast material recovery systems, and equipment needed to create a negative air pressure environment to protect workers or production lines from contamination [6].

As the downturn in the commercial building market continued in 1989, competition increased. Jobs that used to have four or five bidders attracted eight or nine; owners began putting off maintenance painting to save money [10].

According to the U.S. Department of Commerce industry census, there were close to 30,000 establishments in painting and paperhanging (SIC 1721) in 1987, up from 25,000 in 1982. These firms employed 170,000 workers in 1987, of whom over 145,000 were construction workers. This is an increase of 35,000 workers since 1982. Dollar value of business done in 1987 almost $8 billion compared to $4.7 billion in 1982. Net value of construction work was $7.4 billion in 1987, an increase of $3 billion since 1982 [1].

Electrical Work. In recent years, mergers and acquisitions have contributed to drastic changes in the electrical industry (SIC 1731). In 1988, 590 electrical contractors dropped from the industry's ranks, up nine percent from 1987 according to Dun & Bradstreet [11]. On the other hand, the most recent industry census shows that the actual number of electrical contracting firms operating in the United States has increased by 25 percent between 1982 and 1987 [1].

In ENR's specialty contractors survey, 179 firms who reported at least some electrical work indicated that only eight percent of their 1989 revenues came from transportation jobs and that the commercial (37 percent) and industrial (32 percent) markets dominated their business. The era of giant electrical contractors may be fading because of legal troubles and declining revenues for some of the market's largest contractors. This has opened the market for new contenders.

The outlook for the early 1990s is a cause for caution however. Slowdowns in military construction, automobile plants, and office buildings are depressing the market; bright spots include transportation and renovation which represented 15 percent of revenues in 1989 [10].

The industry census estimates that in 1987 there were over 49,000 electrical contractors performing electrical work (SIC 1731), up from 39,500 firms in 1982. These contractors employed more than 500,000 workers in 1987, of whom 405,000 were construction workers. Total business done was $36 billion, up 33 percent from 1982. Net value of construction work was $34.7 billion in 1987 [1].

Masonry, Stone Setting and Other Stone Work. Of the respondents to the 1990 *ENR* survey of masonry contractors, 23 firms reported revenues substantial enough to place them on the "Top 600 specialty contractors" list. The downturn in commercial building work may be helping masonry contractors that are specializing in restoration work. The reason is that owners are holding onto what they have. Other leading masonry contractors are side-stepping the downturn in commercial building by tapping markets that tend to favor brick and block building materials, such as hospitals, educational and religious buildings, and other institutional buildings.

The U.S. Department of Commerce industry census estimates that there were over 23,000 establishments engaged in masonry work, stone setting, and other stone work (SIC 1741) in 1987, up from 20,000 in 1982. These establishments employed 169,000 workers in 1987, a 40 percent increase over 1982. More than 150,000 of these workers were construction workers in 1987. Total dollar value of construction business done that year was $8.7 billion, almost twice as much as in 1982. Net value of construction work in 1987 was $8.3 billion, approximately $4 billion more than in 1982 [6].

Plastering, Drywall, Acoustical, and Insulation Work. This industry (SIC 1742) consists primarily of wall and ceiling contractors. Competition is intense in this market because of the relative ease of entry related to low capital requirements. In 1989, business failures among wall and ceiling contractors climbed seven percent with 266 firms going out of business.

In 1989 there was $697.1 million worth of work performed by the top 20 wall and ceiling contractors. This was a 34 percent increase over 1988 work according to *ENR's* 1990 survey. In 1989, the commercial building market accounted for 70 percent of the revenues, followed by the institutional market at 14 percent and the residential market at 7 percent. Only 11 percent of the top 20 firms' business came from renovation work. However, most wall and ceiling contractors expect repair and remodeling work to surpass new construction contracts in the near future [10].

According to the U.S. Department of Commerce's Bureau of the Census, there were about 18,000 wall/ceiling contractors (SIC 1742) in 1987, up from 16,000 in 1982. These establishments employed over 250,000 workers in 1987 (an increase of 50,000 over 1982), of whom 217,000 were construction workers. Total dollar value of business done that year was over $16 billion, $6 billion more than in 1982. Net value of construction work increased by $6 billion, from $9 billion in 1982 to $15 billion in 1989 [1].

Terrazzo, Tile, Marble, and Mosaic Work. The U.S. Department of Commerce census estimates that there were 5,000 establishments engaged in terrazzo, tile, marble and mosaic work (SIC 1743) in 1987, an increase of more than one thousand firms since 1982. These establishments employed 34,000 workers in 1987, of whom 28,000 were construction workers. Total dollar value of business done exceeded $2 billion, up from $1 billion in 1982. Net value of construction work in 1987 was also approximately $2 billion [1].

Carpentry Work. Table V–B6 summarizes market structure of special trade contractors in SICs 1751 through 1781. The most recent census by the U.S. Department of Commerce estimates that there were 36,000 firms engaged in carpentry work (SIC 1751), an increase of more than 5,000 over 1982. These establishments employed 190,000 workers in 1987, of whom 164,000 were construction workers. Dollar value of business done in 1987 was $11 billion, approximately twice as much as in 1982. Total net value of construction work was $10 billion in 1987, up from $4.8 billion in 1982.

Carpentry is one of the major specialty trades in construction. Wood is a preferred and common building material with many applications and its use almost always requires the skill of a carpenter. Over 60 percent of the total value of construction work by carpenters is in residential building construction, reflecting the predominant use of wood in these buildings. About 45 percent of carpentry work is for detached single-family houses, where carpenters are often responsible for completing the whole structure. Carpentry is essential in other construction projects as well; for example, carpenters may construct concrete forms and build platforms and guardrails.

Many workers in the carpentry trade act as independent contractors and may only work part of the year. Thus, the

number of full-time equivalent carpenters employed by establishments (as reported by the Department of Commerce) may significantly underestimate the number of people classifying themselves as carpenters. Based on household data from the Bureau of Labor Statistics Employment and Earnings series, there were an estimated 1.3 million carpenters in the United States in 1989.

Floor Laying and Other Floor Work, NEC. In 1987, there were over 8,000 establishments engaged in floor laying and other floor work (SIC 1752) according to the U.S. Department of Commerce census estimates. This was an increase of approximately 2,000 firms over 1982. These firms employed close to 45,000 workers in 1987, of which more than 34,000 were construction workers. Dollar value of business done rose to $3.7 billion that year, up from $1.9 billion in 1982. Net value of construction work increased by $2 billion during that same period to $3.4 billion.

Roofing, Siding and Sheet Metal Work. Roofing contractors (SIC 1761) are not likely to suffer from construction cyclicality as much as other trades because a substantial part of their market consists of repair and renovation work. Renovation work for five of the top roofing firms represented 75 percent of their business. The National Roofing Contractors Association conducted a survey of roofers which showed that commercial work, particularly renovation, has grown substantially in recent years [11].

The 39 largest roofing firms had $608 million in revenues in 1989, seven percent below 1988. Declines in revenues will have the greatest impact among smaller contractors where competition is the most pronounced. In the single ply roofing market, profit margins are about one percent according to industry sources [6]. In contrast, business for sheet metal firms actually improved in 1989 because rising backlogs from the year before were translated into revenues.

According to the U.S. Department of Commerce, Bureau of the Census, there were an estimated 26,000 establishments in roofing, siding and sheet metal work (SIC 1761) in 1987 which employed 230,000 workers, of whom approximately 187,000 were construction workers. Total dollar value of business done in 1987 was $15 billion and net value of construction work was $14 billion.

Concrete work. Concrete contracting (SIC 1771) is a very competitive business consisting of over 23,000 establishments according to the U.S. Department of Commerce. In such a market, a firm with $3 million per year in revenues is considered big. The two top ranking concrete contractors have revenues of over $100 million each. There has been a growing demand for super-flat industrial floors and much of the revenues of the larger firms come from commercial and industrial work; the remainder consists of residential construction and nonbuilding construction. Also, many of the larger firms offer a "turnkey" service that provides for all the concrete needs of a project.

In 1989, some of the big concrete contractors posted gains in revenues, but market prospects softened in 1990 with the hardest hit firms specializing in commercial building. In other subsectors, high rise and automotive markets were down but food processing and retail work were relatively strong as were prospects for paving work [6].

The Bureau of the Census estimates that the concrete contractor industry (SIC 1711) employed 218,000 workers in 1987, of whom 187,000 were construction workers compared to 157,000 and 138,000 respectively in 1982. In 1987, the industry's total dollar value of business done was $15 billion, up from $8 billion in 1982. Net value of construction work in 1987 was estimated at $14 billion compared to $7 billion in 1982.

Water Well Drilling. There has been little change in the water well drilling industry (SIC 1781) over the past five years. According to U.S. Department of Commerce census estimates, there were 3,400 firms engaged in water well drilling in 1987, 100 establishments fewer than in 1982. These firms employed 18,000 workers in 1987, just one thousand more than in 1982. Of these, 14,000 workers were estimated to be construction workers in 1987. Dollar value of business done in 1987 was $1.3 billion, only a marginal increase over 1982. Net value of construction work rose from $1 billion in 1982 to $1.3 billion in 1987.

Structural Steel Erection. Table V–B7 summarizes market structure and trends of special trade contractors in SIC 179. There have been a number of changes in the steel erection industry (SIC 1791) with a shake out that forced some big companies like Bethlehem Steel Corporation to cease field operations in recent years[6].

To offset revenue losses from the weakening commercial market, the nation's largest steel erection contractors have sought infrastructure work. Just a few years ago the largest firms in the sector found about half their work in the commercial market, with the other half evenly split between the industrial and the bridge and highway markets. By 1990, bridge and highway jobs accounted for half of total revenue with industrial and commercial jobs splitting the remainder. It is believed that the infrastructure market is strong enough to offset the downturn in the commercial market for most large steel erection contractors. However, erectors that cannot get into the infrastructure market will probably experience economic hardship [6].

The U.S. Department of Commerce estimates that there were 4,000 establishments in the steel erection industry (SIC 1791) in 1987, an increase of 300 firms over 1982. The industry employed 65,000 workers in 1987, up from 62,000 workers in 1982. Of these, 55,000 were construction workers in 1987 and the dollar value of business done that year was $5 billion, up from $3.7 billion in 1982. Net value of construction work was $4.5 billion in 1987, an increase of approximately $1 billion from 1982.

Glass and Glazing. The United States market for glazing and curtain wall contractors has weakened as the pace of new construction has slowed. In recent years, more than 80 percent of the work of the 25 largest glazing and curtain wall contractors came from the commercial market. According to Dun and Bradstreet, 66 glazing firms failed in 1989 [6]. Many banks have tightened lending to commercial developers. According to industry sources, some banks now expect projects to be 30–50 percent pre-leased before they will consider financing construction [10].

Overall, the U.S. Department of Commerce's 1987 industry census estimates that there were about 5,000 establishments in the glass and glazing contracting industry in 1987 compared to 4,000 in 1982. These establishments employed 41,000 workers in 1987, of whom 29,000 were construction workers. In 1987, total dollar value of construction business done by SIC 1793 was estimated to be $3.2 billion and net value of construction work was $3.1 billion compared to $2.6 billion and $2.4 billion respectively in 1982.

Excavation Work

There were 6,000 fewer excavation/foundation contractors (SIC 1794) in 1987 than in 1982. This sector has had a particularly large number of failures; even large excavation contractors are having a difficult time maintaining a steady flow of work. There seems to be no particular region of the country that is doing significantly better than the others [6].

Although some of the large general contractors do their own excavation and foundation work, excavation contractors

still dominate the market. Of the $7.4 billion worth of excavation and foundation work performed in 1987 by specialty contractors, 22.1 percent was for residential construction.

According to the U.S. Department of Commerce industry census, there were 13,400 establishments in 1987 in SIC 1794 compared to 19,600 in 1982. These establishments employed 95,000 workers in 1987, down from 136,000 in 1982. Total dollar value of business done in 1987 ($8.2 billion) was approximately the same as in 1982. Net value of construction work in 1987 was estimated at $7.4 billion.

Wrecking and Demolition. Regulation of environmental pollutants, particularly asbestos, has redefined the demolition industry. Those companies who are not gaining experience in asbestos and other hazardous materials are losing market share because developers are looking for demolition firms that can perform the dual function of demolition and abatement.

In 1990, wrecking and demolition firms were squeezed by rising capital requirements, higher insurance costs and stricter environmental regulations. Some smaller demolition contractors were having a difficult time adjusting. However, environmental concerns are creating more work for firms that have the equipment and crews to deal with the more complex environmental clean-up jobs.

In the South, for example, new work is coming from the dismantling of industrial facilities such as petrochemical plants that are environmentally unsound. In the West, infrastructure improvement including freeway work and bridge removal is a major source of work for wrecking contractors. The rest of the country also has a growing need for bridge work over the next several years. Over 40 percent of the country's bridges are in need of rehabilitation or repair. Some of these will be replaced by demolition contractors. High public concern about occupational lead exposure and increased environmental concerns over lead paint abatement should translate into more contracts for firms specializing in removing or working with this heavy metal.

The most recent U.S. Department of Commerce industry census estimates that there were 1,200 firms engaged in wrecking and demolition work (SIC 1795) in 1987, up from 900 firms in 1982. These firms employed over 14,000 workers in 1987, of whom almost 12,000 were construction workers. Dollar value of business done more than doubled from 1982 to 1987, rising from $400 million to approximately $910 million, while net value of construction work changed from $300 million in 1982 to $845 million in 1987.

Installation or Erection of Building Equipment, NEC. These special trade contractors (SIC 1796) primarily engage in the installation or erection of building equipment such as elevators, pneumatic tube systems, and dust collecting equipment. Contractors in this industry also install or dismantle machinery or other industrial equipment.

For example, U.S. elevator contractors are part of SIC 1796. United States elevator contractors are coping with fluctuations in the demand for new facilities by capitalizing on a recent trend toward modernization to fend off the effects of a national slowdown in new multi-story construction. The popular trend in upgrading elevators is to replace the conventional relay-lodging systems of the 1950s and 1960s with today's state-of-the-art, electronic microprocessor systems, which are easier to install, operate, and maintain. However, in order to install and service such sophisticated equipment, the industry will have to undergo a transformation which will affect its workforce. To improve the workforce, the National Association of Elevator Contractors is spearheading a campaign to better educate field personnel among its existing 400 member companies and to recruit more college and trade school graduates with computer backgrounds into elevator contracting [6].

According to the U.S. Department of Commerce industry census, there were close to 4,000 establishments in SIC 1796, a number which has changed little since 1982. These establishments employed 62,000 workers in 1987, of whom 50,000 were construction workers. Dollar value of business done was estimated at $5.4 billion, approximately $1 billion more than in 1982. Net value of construction work was $5 billion in 1987, up $1 billion from 1982.

Miscellaneous Special Trade Contractors, NEC. This industry group consists of special trade contractors who are primarily engaged in construction work and who are not classified under any of the other construction industry categories in SIC 1799. This group includes such diverse firms as swimming pool and fence construction, erection and installation of ornamental metal work, house moving, shoring work, waterproofing, dampproofing, fireproofing, sandblasting, and steam cleaning of building exteriors.

This group represents almost half (47 percent) of all establishments included in the miscellaneous contractor industry (SIC 179). According to the U.S. Department of Commerce most recent census figures, there were an estimated 23,000 establishments in SIC 1799 in 1987, 3,000 more firms than in 1982. These establishments employed 176,000 workers in 1987, of whom 140,000 were construction workers. Total dollar value of business done by these firms was $11 billion in 1987, up from $6 billion in 1982. Net value of construction work in 1987 was estimated at $10 billion.

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3. Employee Exposures and Benefits

Introduction. Employee exposures to the substances included in the scope of this rulemaking are associated with a wide variety of acute and chronic conditions and illnesses. These include sensory irritation, narcosis, systemic toxicity, respiratory disease, neuropathy, and cancer.

Since the adoption of the existing limits twenty years ago, toxicological evidence has become available that shows that adverse health effects can occur as a consequence of exposure to the substances regulated, and that such health effects occur even when exposures are maintained at the current limits. In addition, many substances that have come into widespread use or been introduced since 1970 have been shown to be potentially hazardous in workplace environments. OSHA thus believes that reducing worker exposures

to such substances by lowering existing exposure limits or by adding limits for previously unregulated substances will result in a significantly reduced risk of illness.

This chapter describes both the methodology used to identify construction workers potentially exposed to hazardous substances included in this proposed rulemaking and the expected benefits to those workers resulting from lowering permissible exposure limits.

Data sources and methodology. The extent of exposures to the substances included in this proposed rulemaking for the construction industry was evaluated by first identifying specific activities with potential exposures.

For each activity, the extent of exposures to the various substances present was determined. Activities involving substances for which OSHA is adding a skin designation were assessed for dermal exposure.

OSHA used exposure monitoring data from several sources to determine worker exposure levels during construction activities. One source for monitoring data is OSHA's Integrated Management Information System (IMIS) data base. This data base contains exposure measurements obtained by OSHA compliance officers during inspections of worksites. For each site inspected, the IMIS file includes information on the number of employees involved, results of employee air monitoring, and the number of employees potentially exposed to each substance monitored.

Another valuable source of information was the National Institute for Occupational Safety and Health (NIOSH). NIOSH regularly conducts studies on potential occupational health hazards throughout the United States, including the construction industry. NIOSH's health hazard evaluation reports give detailed descriptions of working conditions and activities at a particular site. The chemicals present are identified, recommended controls and work practices are discussed, and exposure monitoring results are analyzed. Although the conclusions in the report concerning possible health hazards are related to a particular site, the data provide a link between a certain set of circumstances and the resulting exposure levels, and can be effectively applied to similar situations for the relevant activity throughout the industry.

OSHA collected additional exposure monitoring data specifically for this rulemaking. A series of site visits was conducted by industrial hygienists working for a contractor hired by OSHA. Establishments were assured of confidentiality, allowed their employees to be monitored, and provided various information such as the duration and frequency of activities, the numbers of employees involved, and the value of the construction work at the project. In return, the establishments received the consulting services of the health professionals for the duration of the visit. Data gathered from this effort served to confirm data from other sources and provided definitive information for principal activities and activities that otherwise would not be as well documented.

OSHA also conducted a major computer-assisted telephone interview (CATI) survey to develop a comprehensive profile of employee exposures in the construction industry. Over 1,100 establishments provided complete responses. The firms were selected at random within 48 survey cells. The survey cells were defined by a combination of SIC code and establishment size. Establishments with fewer than twenty employees were considered small and establishments with twenty or more employees were considered large.

Each respondent supplied specific information on the demographic characteristics of the firm, the type of construction project covered, and the role of the firm and its employees in the project. Respondents were asked to identify all activities with potential exposures. For each category of construction work, an initial list of activities and associated substances and products served as prompts. The substances and products involved in each of the respondent's activities were then determined and confirmed. In cases where the presence of specific products is easier to identify than the presence of individual chemicals, the names of the products and the manufacturers were solicited and material safety data sheets (MSDSs) were used to identify the relevant chemicals.

The survey requested details about each activity to determine the nature of potential exposures, including information on the work environment, the controls used, the scale of the activity, and the number of workers involved. The data from each respondent were evaluated in combination with documented exposure monitoring results and other research concerning the chemicals present, such as the concentration and quantity involved, the likelihood of becoming airborne, and the stringency of the proposed PEL. The number of workers likely to be exposed and overexposed to various chemicals during each activity was determined with computer algorithms developed by experienced industrial hygienists using all available data associated with the activity.

The estimates generated from the survey data were derived from the responses of a representative sample of construction establishments within each survey cell. These data were scaled up to national levels through the use of published statistics on total employment by firm size and SIC code. The 1987 Census of Construction Industries, published by the U.S. Department of Commerce in 1990, provides these and other detailed statistics on the construction industry.

Number of Exposed Employees. Table V–B8 presents the total estimated full-time equivalent number of employees exposed to substances found in the construction industry and considered in this proposed rulemaking. A full-time equivalent exposed employee is comparable to a person-year of exposure, which represents one shift each working day for one year. Due to the nature of some construction activities, the actual number of people who may experience an exposure over the course of a year would be larger. For example, if 100,000 workers were exposed to a chemical for an average of two hours per week, their combined exposure would equal that of 5,000 full-time equivalent employees.

TABLE V–B8.—EMPLOYEE EXPOSURES TO CHEMICALS IN CONSTRUCTION ADDRESSED BY THE PROPOSED RULE

Chemical		Full-time equivalent employees	
Code	Name	Exposed	Exposed above proposed PEL and unprotected
0040	Acetone	94130	670
0160	Alpha-Alumina	1	0
0170	Ammonia	66969	316
0290	Asphalt fumes	300786	1643
0420	Butane	37	0
0430	2-Butanone (MEK)	35204	285
0435	Butoxyethanol	64368	1299
0440	n-Butyl-acetate	17651	200
0460	n-Butyl alcohol	1022	23
0477	n-Butyl glycidyl ether	1485	0
0515	Calcium hydroxide	2759	0
0560	Carbon monoxide	143708	0
0577	Portland cement	409291	3905
0640	Chlorine	8601	54
0645	Epichlorohydrin	326	0
0686	Chromic acid and chromates	7764	591
0850	Cyclohexanone	40587	0
0910	Diethylamine	2181	0
0921	Diethylene triamine	57707	0
1030	Ethanolamine	1207	0
1050	Ethyl acrylate	753	0
1060	Ethyl benzene	79998	844

TABLE V-B8.—EMPLOYEE EXPOSURES TO CHEMICALS IN CONSTRUCTION ADDRESSED BY THE PROPOSED RULE—Continued

Chemical		Full-time equivalent employees	
Code	Name	Exposed	Exposed above proposed PEL and unprotected
1285	Fluorotrichloromethane	4692	0
1300	Fibrous glass	160484	490
1340	Gasoline	46234	0
1371	n-Heptane	34926	55
1380	n-Hexane	63986	2397
1385	Hexone (methyl isobutyl ketone)	34830	200
1389	Hexylene glycol	77681	1549
1480	Hydrogen sulfide	5801	54
1560	Isopropyl alcohol	137103	200
1591	Lead	105050	6266
1630	Mercury, organo alkyl compounds	45236	0
1631	Mercury, aryl and inorganic compounds	93164	1471
1660	Methyl alcohol	64716	68
1720	Methyl chloroform (1,1,1-trichloroethane)	112119	1431
1781	Mineral wool fiber	4456	0
1782	Alpha-methyl styrene	99591	0
1790	Molybdenum	4974	1698
1840	Nickel, soluble compounds	20137	2139
1900	Nitrogen dioxide	136823	10
1910	Ethylene glycol dinitrate	310	0
1912	Nitroglycerine	310	0
1913	Ethylene glycol vapor and mist	314657	7950
1941	2-nitropropane	1022	25
1980	Ozone	130123	0
2037	Petroleum distillates (naphtha)	473443	16917
2085	Phosphoric acid	1265	0
2160	n-Propyl acetate	3905	0
2210	Propylene glycol monomethyl ether	55369	1103
2227	Rosin core solder pyrolysis (as HCHO)	27821	0
2260	Sodium hydroxide	7	0
2270	Stoddard solvent	12522	0
2280	Styrene	721	0
2390	Tetrahydrofuran	31323	0
2431	Tin organic compounds	47344	0
2440	Titanium dioxide	222499	5355
2460	Toluene	314940	2291
2470	Toluene-2,4-diisocyanate	20563	401
2480	Triethylamine	2558	591
2490	Trichloroethylene	67359	68
2530	2,4,6-Trinitrotoluene (TNT)	3	0
2584	VM & P naphtha	24221	473
2587	Welding fume	106735	11088
2590	Xylene (o-, m-, p-isomers)	376742	4357
2610	Zinc oxide fume	15322	235
2611	Zinc chloride fume	3190	0
2612	Zinc chromate, as CR	594	0
9010	Silica quartz (crystalline), respirable	852083	30583
9015	Silica cristobalite, respirable dust	68938	11783
9050	Silica, amorphous (diatomaceous earth)	1982	0
9075	Mica	55500	591
9210	Wood dust, hard wood	152322	804
9211	Wood dust, soft wood	395217	189
A102	Aluminum: Welding fumes	17822	0
H146	Hexane isomers	1000	0
W102	Wood dust, western red cedar	9363	0
W103	Wood dust, soft & hard woods exc W. red	43525	30
Z102	Zinc oxide dust	18537	0

Source: Office of Regulatory Analysis, OSHA; based on CONSAD [1].

The total number of employees potentially exposed to one or more substances annually would be over 4 million, the total number of construction workers in the industry. This is due to several factors. Workers in every occupational group in the construction industry engage in one or more of the activities identified as involving exposures to the substances associated with this proposed rule. The workforce in the construction industry is relatively transient and mobile within the industry and workers have a greater likelihood of being assigned to a variety of different tasks over the course of a year. Each firm in the industry may work on several projects with varying activities and exposures. A construction worker engaged in one activity faces potential exposures from other activities occurring in the vicinity.

Substances for which exposures above the proposed PELs are likely to occur most frequently include acetone, butoxyethanol, n-heptane, lead, methyl chloroform, nickel, ethylene glycol, petroleum distillates, portland cement, titanium dioxide, toluene, welding fume, xylene, crystalline silica quartz, and silica cristobalite. Specific chemicals and activities with overexposures are discussed below.

Acetone is a solvent and may be a component in paints and varnishes, rubber adhesives, and vinyl cements. Although this chemical will be present in many construction activities, most of the overexposures to acetone are expected to occur during the application of single ply membrane roofing systems. Overexposures have also been found during the application of floor coatings and while laying floors, as well as during steel painting and bonding and gluing operations.

N-heptane is a solvent that can be present in many construction applications. Overexposures to n-heptane can occur during the application of single ply membrane roofing systems and during the exterior painting of steel. Through the use of adequate ventilation, exposures can be kept below the PEL and the short-term exposure limit during floor work (patching and resurfacing) and other gluing operations.

Lead exposure is a serious health hazard faced by many construction employees. The risks are compounded because employers may not always be aware of the presence of lead or may underestimate the potential for overexposure. Old lead-based paints may have a lead content of over 30 percent. Severe overexposures can occur when the paint is grinded, burned, or otherwise dispersed into the air. Soldering, brazing, and welding may also involve lead exposures. The proposed PEL for total welding fumes is 100 times the proposed PEL for lead, making specific precautions for lead exposure a necessary consideration when it is present.

Activities where overexposures to lead are most common include abrasive blasting; burning, cutting, or welding on painted steel, welding or cutting galvanized steel; grinding, drilling, chipping, or cutting on any painted surfaces; and soldering or brazing. Over 500,000 construction employees are estimated to be engaged in these activities on a regular basis, which may often be done by inexperienced laborers and helpers.

Potential exposures to lead are expected to become more widespread in the construction industry in the near future. The Department of Housing and Urban Development (HUD) is requiring public housing agencies to inspect housing projects for lead paints by 1994. Abatement is required if lead is present above specified concentrations. The lead abatement industry is expected to grow significantly as the awareness of potential lead hazards increases among all building owners.

A second source of increased exposures to lead will result from increases in infrastructure development efforts. Tens of thousands of bridges, neglected in the past, are in need of

rebuilding, repair, or repainting, and major rehabilitation projects are being initiated or planned throughout the country. The removal of old lead paints and other activities associated with bridge work involve potentially severe exposures even in outdoor settings. Compliance with Environmental Protection Agency (EPA) regulations, which are designed to protect the environment by requiring such operations to be enclosed, further concentrates exposures for workers within the enclosures.

Methyl chloroform is a solvent used in construction activities such as laying floors, surface cleaning and stripping, insulation installation, and other bonding and gluing operations. Overexposures can be avoided by providing adequate ventilation, but have been found to occur in situations with extended exposures in relatively confined spaces. Surface cleaning and stripping operations are especially prone to result in worker overexposures due to the proximity of the chemical to the breathing zone.

Exposures to airborne concentrations of soluble compounds of nickel are possible during burning, cutting, or welding on painted steel and also during welding or cutting stainless steel.

Ethylene glycol is a component of many latex paints. It can become airborne as either a vapor or a mist during painting operations. When painting is done indoors and concentrations are allowed to build up, overexposures may occur. Workers engaged in interior spray painting for extended periods of time are most likely to experience overexposures.

The presence of petroleum distillates is widespread in the construction industry. It can be a component of such products as laquers, paints, polyester coatings, asphalt products, caulks, sealants, and rubber cements. Often the presence of petroleum distillates will not be a significant hazard, but several activities have been identified where the potential for overexposure exists. These include exterior painting of wood finishes, exterior painting of steel, concrete sealing and coating operations, interior application of floor coatings, interior painting, surface stripping, and preparing concrete forms.

Toluene and xylene are common solvents found in many paints, coatings, caulks, epoxies, adhesives, sealants, and curing agent mixtures. Although most of the construction workforce may be regularly exposed to these agents to some degree, overexposures are relatively limited. Activities with potential for overexposure include the application of single ply membrane roofing systems, concrete sealing and coating operations, interior application of floor coatings, interior painting, preparing and installing concrete forms, and traffic line painting.

Welding and cutting are common activities that are the main sources of exposure to welding fumes. Overexposures can occur if appropriate controls are not employed. Based on the CONSAD survey of construction establishments, approximately 10 percent of workers exposed to welding fumes are potentially overexposed.

Exposures to various forms of silica are among the most common exposures to airborne contaminants in the construction industry. Incidences of overexposure are likely to be limited to circumstances involving extended periods of work in dusty environments. These are most likely to occur during earthmoving, trench excavation, abrasive blasting, batch mixing of concrete or mortar, grinding or cutting on concrete and other surfaces, and tunnel blasting and excavation.

Estimated reductions in fatalities and illnesses. The reduction of employee exposures to hazardous substances to a level below that associated with adverse health effects will result in a decrease in the number of fatalities and illness cases among affected construction workers. This section provides estimates of the numbers of fatalities and illnesses that can be expected to be avoided in the construction industry through compliance with the proposed rule.

As presented in Table V–B8, OSHA has estimated the extent of exposures in the construction industry as the number of full-time equivalent employees exposed. Construction workers performing activities contributing to these exposures are currently at risk of experiencing a variety of adverse health effects brought about by overexposures to the substances included in this rulemaking. Many of these adverse effects, in particular cancer, systemic toxicity, chronic respiratory disease, neuropathy, and chronic liver and kidney damage, can result in lethal outcomes. Employees who are excessively exposed to substances causing organ damage, neurological impairment, or metabolic effects may also be at excess risk of incurring a fatal condition. A description of the risks associated with overexposures to specific chemicals can be found in the health effects section of this preamble.

Table V–B9 presents the extent of exposures above the proposed PELs in construction by substance and type of health effect. Substances for which compliance with the proposed PEL would be equivalent to compliance with existing requirements are not included in this table, and the benefits of compliance with these limits are not attributed to the proposed rule. Such substances include nuisance particulates, limestone, plaster, gypsum, and calcium silicate.

The major health effects category includes respiratory effects, which are primarily due to exposures to silica, soluble nickel compounds, asphalt fume, mica, fibrous glass, and wood dust. Petroleum distillates, lead, n-hexane, and mercury cause neuropathy effects among exposed workers; welding fume, butoxyethanol, and zinc oxide fume are major contributors to systemic toxicity effects; and toluene, methyl chloroform, and n-heptane exposures are associated with narcosis effects. Substances with exposures above the proposed PELs causing increased cancer risk to construction workers include chromic acid and chromates. Sensory irritation results from exposure to ethylene, xylene, hexylene, and propylene; and physical irritation results from exposure to titanium dioxide, portland cement, and molybdenum.

TABLE V–B9.—EXPOSURES IN CONSTRUCTION ABOVE PROPOSED LIMITS BY TYPE OF HEALTH EFFECT

Type of health effect and substance	Exposures above proposed limits (person-years)
Sensory irritation:	
Ethylene Glycol	7,950
Xylene	4,357
Hexylene Glycol	1,549
Propylene G.M.E.	1,103
Ethyl Benzene	844
Acetone	670
Triethylamine	591
VM & P Naphtha	473
Ammonia	316
2–Butanone	205
N–Butyl Acetate	200
Isopropyl Alcohol	200
Chlorine	54
Subtotal	18,592
Physical irritation:	
Titanium Dioxide	5,355
Portland Cement	3,905
Molybdenum	1,696
Toluene-2,4-Diisocyanate	401
Subtotal	11,357
Respiratory:	
Silica Quartz (Crystalline)	30,583
Silica Cristobalite	11,783
Nickel, Soluble Compounds	2,139
Asphalt Fume	1,643
Mica	591
Fibrous Glass	490
Wood Dust, Hard Wood	304
Wood Dust, Soft Wood	169
Wood Dust, general	30

TABLE V–B9.—EXPOSURES IN CONSTRUCTION ABOVE PROPOSED LIMITS BY TYPE OF HEALTH EFFECT—Continued

Type of health effect and substance	Exposures above proposed limits (person-years)
Nitrogen Dioxide	10
Subtotal	47,770
Narcosis:	
Toluene	2,291
Methyl Chloroform	1,431
N–Heptane	85
Trichloroethylene	68
Subtotal	3,875
Neuropathy:	
Petroleum Distillates	16,387
Lead	6,266
N–Hexane	2,397
Mercury, Aryl and Inorganic	1,471
N–Butyl Alcohol	23
Subtotal	26,544
Cancer:	
Chromic Acid/Chromates	591
2–Nitropropane	23
Subtotal	614
Systemic toxicity:	
Welding Fume	11,088
Butoxyethanol	1,299
Zinc Oxide Fume	235
Subtotal	12,622
Ocular:	
Methyl Alcohol	68
Hydrogen Sulfide	54
Subtotal	122
Liver/Kidney: Hexone	200
All	121,695

NOTE: Summation of exposures to different substances may include double counting of employees simultaneously exposed to more than one substance.

Source: Office of Regulatory Analysis, OSHA, U.S. Department of Labor.

OSHA has estimated the expected reductions in illnesses and fatalities resulting from this rulemaking based on full-time equivalent exposures. The PELs are established based on the ability to protect workers from all potential exposure over a full working lifetime, and the risks associated with excess exposure are based on daily full-time exposure for 45 years. Actual total excess risk to construction workers also depends on the overall dose.

As a best estimate of excess risk, it is assumed that the aggregate amount of overexposure will determine the aggregate amount of excess risk, and that this will be a linear (proportional) relationship. There are two offsetting arguments which indicate that the actual total risk for the construction work force could be higher or lower. If the total risk depends positively on the total number of people exposed as well as on the total amount of exposure, then the actual total risks could be considerably higher. On the other hand, if less frequent exposures result in lower than proportional risk, then the actual risk would be lower than that estimated.

Based on the assumption of proportional risk, and a full-time equivalent population exposed above the proposed PELs continuously over a working lifetime, OSHA estimates that an excess mortality rate in the range of 0.5 to 2 percent (with a best estimate of 1 percent) would result. Fatalities would result from continuous occupational exposure above the proposed PELs for substances associated with cancer and adverse respiratory, narcosis, neuropathy, systemic toxicity, liver, and kidney effects. Substances causing these health effects may also cause other life-threatening conditions and indirectly contribute to an increased risk of premature death by increasing susceptibility to diseases, weakening immune systems, or through interactions with other chemicals.

Estimates of the extent of exposures above the proposed PELs are classified by type of health effect and presented in Table V–B9. If exposure to each chemical is counted independently, then the estimated total number of person-years of exposure above the relevant proposed PELs is about 90,000 annually. However, total person-years of overexposure may be greater than the number of total full-time equivalent overexposed employees if exposure to more than one substance occurs simultaneously. Since the estimated mortality rate applies to numbers of workers, the number of person-years of overexposure was adjusted downward by 10 percent to account for this possibility. As a result, approximately 81,000 full-time equivalent workers are estimated to be exposed above the proposed PELs with an increased risk of premature death.

Based on the assumption of a 1 percent excess mortality rate among this cohort, an average of 18 fatalities per year would be addressed by this rulemaking. OSHA assumes that reductions in exposures resulting from full compliance with the proposed rule could be expected to prevent about half (7–11) of these fatalities.

OSHA's estimated excess mortality rate is conservative compared with other mortality studies and epidemiological research in the construction industry. An occupational mortality study conducted by the California Department of Health Services revealed that laborers and other construction workers experience significantly higher mortality rates than the general population [6]. Mortality rates were an average of over 50 percent higher for lung cancer, other cancers, circulatory system diseases, cerebrovascular disease, chronic obstructive pulmonary disease, cirrhosis and other liver disease, and diseases of the urinary system. These data indicate that the excess mortality due to overexposures among construction workers may be far greater than estimated above. However, a portion of this excess mortality may be related to causes other than exposures to substances in this proposed rulemaking (e.g. cigarette smoking).

On the basis of exposure data delineated further in Section V–B–6 (Costs of Compliance), OSHA estimates that about 84,000 full time equivalent construction employees may be exposed to an average of 0.5 mg/m^3 asphalt fume. The quantitative risk assessment for asphalt fume presented in this preamble indicates that this exposure may produce approximately 4 to 5 excess fatalities due to lung cancer annually. Although exposures may be reduced for some employees by using applicable engineering controls and work practices (such as insulating pipes and keeping asphalt temperatures low), OSHA expects that 80,000 FTE employees would need respiratory protection in addition to such controls to comply with a PEL of 0.2 mg/m^3. According to the results of the quantitative risk assessment, about 3 to 4 fatalities per year may thus be avoided by reducing the average exposure to 0.05 mg/m^3.

The number of illnesses associated with overexposures in construction that are potentially preventable by this proposed rulemaking is difficult to assess. The Bureau of Labor Statistics (BLS), in its annual survey of occupational injuries and illnesses in the United States, found that 7,700 illnesses were documented and recorded in the construction industry in 1989 [7]. It is widely recognized that these data significantly underestimate the prevalence of occupational illness because only cases recorded and reported by employers are counted.

In the construction industry it is likely that many occupationally related illnesses are never documented as such. One reason may be that neither the employer nor the employee may be aware that an illness is occupationally related. In addition, chronic and disabling illnesses may only become

apparent after the exposure has ceased, and none of these cases are likely to be included in employer records. Relatively high rates of worker turnover experienced in construction make tracking of exposures more problematical; subsequent employers would probably not be aware of exposures with previous employers. Underreporting of illnesses with latency periods would be greater than the underreporting of illnesses with toxic or acute effects.

In order to compensate for the underreporting of occupationally related illnesses, the BLS estimates were multiplied by a factor of 5. With this adjustment the estimated annual number of illnesses in construction is 38,500.

Some of these illnesses would not be addressed by the proposed standard. Illnesses in construction may include effects associated with exposures to hazardous agents or physical stress, such as radiation, noise, and ergonomic stress. However, illnesses which are covered include skin disorders; the proposed standard adds skin designations for specific substances as appropriate, and costs of providing dermal protection are included in the estimated compliance costs. Although the PELs set by this standard address airborne concentrations, a general reduction in exposure through absorption and ingestion can be expected through increased awareness and improved work and hygiene practices.

The data from the BLS survey indicate that about 30 percent of reported illnesses in construction are due to repeated trauma, physical agents, and poisonings [7]. Overall, 70 percent of the total illnesses in construction, or approximately 27,000 cases, are considered potentially preventable by the proposed standard.

As compliance with the proposed rule would offer protection from excessive dermal and airborne exposures to hazardous chemicals encountered by construction workers in a comprehensive manner, an 80 percent reduction in the incidence of occupationally related illnesses is projected, with a range of 70 to 90 percent. Thus, OSHA estimates that compliance with the proposed rule would potentially prevent about 21,600 illnesses annually.

BLS reported that about 47 percent of the recorded illnesses involved lost workdays, and that an average of 21 lost workdays resulted from lost workday cases [7]. OSHA assumes these same rates apply to unrecorded illnesses. Compliance with the proposed rule is thus estimated to prevent about 10,200 illnesses involving lost workdays, and reduce the number of lost workdays in construction by about 214,000 workdays.

Based on research by Kip Viscusi, the implicit average value of avoiding each lost workday case would be about $40,000 [11]. This value is based on a willingness-to-pay methodology and takes existing compensation programs into account. The total potential benefit associated with the prevention of lost workday illnesses alone would be $408 million.

In addition, excess exposures to some substances in the proposed rule cause material impairments of health that may not always be considered illnesses. Cases of physical and sensory irritation are likely to occur regularly among hundreds of thousands of construction workers. Eliminating these material impairments of health should further reduce the prevalence of illnesses among these workers. Compliance with the proposed PELs would also produce benefits that cannot be quantified, such as the increased comfort and productivity of employees.

OSHA requests comments and data on the prevalence of illnesses and health-related fatalities in construction, as well as on the potential effectiveness of the proposed standard in reducing the incidence of these cases.

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4. Nonregulatory Alternatives

Introduction. The declared purpose of the Occupational Safety and Health (OSH) Act of 1970 is "* * * to assure so far as possible every working man and woman in the Nation safe and healthful working conditions and to preserve our human resources. * * *" Thus, the Act requires the Secretary of Labor, when promulgating occupational safety and health standards for toxic materials or harmful physical agents, to set the standard "* * * that most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity. * * *" It is on the basis of this congressional directive that OSHA has initiated regulatory actions to reduce the adverse health effects associated with occupational exposure to hazardous substances in the construction industry.

Market imperfections. Economic theory suggests that the need for government regulation is greatly reduced where private markets work efficiently and effectively to allocate health and safety resources. The theory typically assumes perfectly competitive labor markets where workers, having perfect knowledge of job risks and being perfectly mobile among jobs, command wage premiums that fully compensate for any risk of future harm. Thus, theoretically, the costs of occupational injury and illness are borne initially by the firms responsible for the hazardous workplace conditions and ultimately by the consumers who pay for the final goods and services produced by these firms. With all costs internalized, private employers have an incentive to reduce hazards wherever the cost of hazard abatement is less than the total cost to the firm, the workforce, and society of the expected injury or illness. The resultant level of safety and health is considered efficient in the sense that it minimizes the sum of the costs (and foregone benefits) of hazard prevention and of injury or illness.

The conditions of perfect competition do not need to be completely satisfied in order for the forces of the market to approximate an efficient outcome. However, some market imperfections can produce sub-optimal results that can be improved upon with regulatory action. In the case of this rulemaking, employees face a significant risk of material impairment of health which is not adequately addressed by current nonregulatory alternatives. OSHA therefore believes that it must take appropriate actions to provide greater health protection for construction workers exposed to toxic substances.

Evidence indicates that market forces in the construction industry have not been effective in reducing excessive occupational exposure to hazardous substances, thereby contributing to the consequent development of occupational diseases. In spite of the danger associated with the inhalation or other exposure to hazardous substances, the social costs of production have not been internalized, in part because of market imperfections and the existence of externalities. Consequently, the amount of protection that the private market will offer to construction workers differs from the socially desired level.

First, evidence on occupational health hazards in general suggests that in the absence of immediate or clear-cut danger, employees and employers have little incentive to seek or provide information on the potential long-term effects of exposure. Employers faced with potentially high compensatory payments may in fact have a disincentive to provide information to employees. When relevant information is provided, however, employers and employees might still find informed decisionmaking a difficult task, especially where long latency periods precede the development of chronic disabling disease. Moreover, if signs and symptoms are nonspecific—that is, if an illness could be job-related or could have other causes—employees and employers may not link disease with such occupational exposure.

Second, even if workers were fully informed of the health risks associated with exposure to hazardous substances, many face limited employment options. Nontransferability of occupational skills and high national unemployment rates sharply reduce a worker's expectation of obtaining alternative employment quickly or easily.

In many regions of the country, the practical choice for workers is not between a safe job and a better paying but more hazardous position, but simply between employment and unemployment at the prevailing rates of pay and risk. In addition to the fear of substantial income loss from prolonged periods of unemployment, the high costs of relocation, the reluctance to break family and community ties, and the growth of institutional factors such as pension plans and seniority rights serve to elevate the cost of job transfer. Thus, especially where wages are more responsive to the demands of more mobile workers who tend to be younger and perhaps less aware of job risks, hazard premiums for the average worker will not fully compensate. Where this is the case, labor market negotiations are unlikely to reflect accurately the value that workers place on health.

In addition to these market imperfections, externalities occur if employers and employees settle for an inefficiently low level of protection from hazardous substances. For the competitive market to function efficiently, only workers and their employers should be affected by the level of safety and health provided in market transactions. In the case of occupational safety and health, however, society shares part of the financial burden of occupationally induced diseases, including the costs of premature death, chronic illness, and disability. Those individuals who suffer from occupationally related illnesses are cared for and compensated by society through taxpayer support of social programs, including welfare, Social Security, and Medicare. These combined factors of labor market imperfections and the existence of externalities contribute to the failure of the market to supply sufficiently healthful working conditions where hazardous substances exist in the construction industry.

Tort Liability. The use of liability under tort law is one nonregulatory alternative that has been increasingly used in litigation concerning occupationally related illnesses. Prosser [1] describes a tort, in part, as a "civil wrong, other than a breach of contract, for which the court will provide a remedy in the form of an action for damages," although he says that "a really satisfactory definition has yet to be found."

If the tort system applies, it would allow a worker whose health has been adversely affected by occupational exposure to a hazardous substance to sue and recover damages from the employer. Thus, if the tort system is effectively applied, it might shift the liability of direct costs of occupational disease from the worker to the firm under certain specific circumstances.

With very limited exceptions, however, the tort system is not a viable alternative in dealings between employees and employers. All states have legislation providing that Workers' Compensation is either the exclusive or principal remedy available to employees against their employers. Thus, under tort law, workers with an occupational disease caused by exposure to a hazardous substance can only file a product liability suit against a third party manufacturer, processor, distributor, sales firm, or contractor. It is often difficult, however, to demonstrate a direct link between an exposure to a hazardous substance and the illness.

In order to pursue litigation successfully, there must be specific knowledge of the magnitude and duration of a worker's exposure to a hazardous substance, as well as the causal link between the disease and the occupational exposure. Usually, it is extremely difficult to isolate the role of occupational exposures in causing the disease, especially if workers are exposed to many toxic substances. This difficulty is further compounded by the long latency periods that are frequently involved. In addition, the liable party must be identifiable, but workers may have several employers over a working lifetime. The burden of proof that an occupational exposure to a hazardous substance occurred, that a specific employer is the liable party, and that the exposure level was significant may prohibit the individual from initiating the suit.

The costs associated with producing information and with litigation itself may be quite substantial. First, information is a public good, which means that once it is produced, it is available for all. As a result, public goods are typically underproduced relative to what is considered economically efficient. This general undersupply of information adversely affects workers' awareness of the cause of their illnesses and thus reduces the likelihood that they will pursue tort liability suits.

Second, information is not a costless commodity. The time and money required to obtain, research, and distribute information can be substantial. Since the outcomes of liability cases are frequently uncertain, additional information is almost always preferable. The complexity of the subject matter, for health standards in particular, requires a large amount of information as a starting point for a lawsuit. These costs would have to be borne by potential plaintiffs before the lawsuit is initiated.

Third, legal proceedings impose costs on both plaintiffs and defendants. In

deciding whether to sue, the tort victim must be sure that the size of the claim will be large enough to cover legal expenses. In effect, the plaintiff is likely to face substantial transaction costs in the form of a contingency fee, commonly 33 percent, plus additional legal expenses. The accused firm must also pay for its defense.

The majority of occupational disease tort activities has involved workers exposed to asbestos. To date, over 100,000 individual plaintiffs have filed asbestos lawsuits in the country. These employees avoided the exclusive remedy of Workers' Compensation by suing suppliers of asbestos instead of employers. A report prepared by the Research Triangle Institute [2] contains some data pertaining to legal costs and the size of awards associated with tort law. One investigator, for example, found that an average ratio of legal costs to proceeds was 37 percent for a sample of cases. The high costs and uncertainties associated with tort law make it an inefficient mechanism for ensuring adequate protection of workers' health.

Insurance and liability costs are not borne in full by the specific employer responsible for the risk involved. For firms that are insured, the premium determination process is such that premiums only partially reflect changes in risk associated with changes in exposure to hazardous substances. This lack of complete adjustment is the so-called "moral hazard" problem, which is the risk that arises from the possible imprudence of the insured. As the insured firm has paid an insurance company to assume some of the risks, that firm has less reason to exercise the diligence necessary to avoid losses. Transfer of risk is a fundamental source of imperfection in markets.

Workers' Compensation. The Workers' Compensation system is a result of the perceived inadequacies in liability or insurance systems to compel employers to prevent occupational disease or compensate workers fully for their losses. The system was designed to internalize some of the social costs of production, but in reality it has fallen short of compensating workers adequately for occupationally related disease. Thus, society shares the burden of occupationally related health effects, premature mortality, excess morbidity, and disability through taxpayer support of social programs such as welfare, Social Security disability payments, and Medicare.

Compensation tends to be inadequate especially in permanent disability cases, in view of the expiration of benefit entitlements and the failure to adjust benefits for changes in a worker's expected earnings over time. As of January 1987, eight states restricted permanent disability benefits either by specifying a maximum number of weeks for which benefits could be paid or by imposing a ceiling on dollar payments [3].

At present, time and dollar restrictions on benefit payments are even more prevalent in the area of survivor benefits. The duration of survivor benefits is often restricted to 10 years, and dollar maximums on survivor payments range from $7,000 to $60,000. In addition, it should be noted that if the employee dies quickly from the occupational illness and has no dependents, the employer need pay only nominal damages under Workers' Compensation (i.e., a $1,000 death benefit).

Finally, in spite of current statutory protection, disability from occupational diseases represents a continuing, complex problem for Workers' Compensation programs. Occupational diseases may take years to develop, and more than one causal agent may be involved in their onset. Consequently, disabilities resulting from occupationally induced illness often are less clearly defined than those from occupationally induced injury. As a result, Workers' Compensation is often a weak remedy in the case of occupational disease. For example, as recently as April 1983, the U.S. Supreme Court refused to hear an occupational disease case (Richard D. Bunker v. National Gypsum Co.) involving a worker who was diagnosed as having asbestosis 23 years after the expiration of the 3 year time limit allowed by Indiana law for filing a compensation claim [4]. Indeed, there is some evidence indicating that the great majority of occupationally induced illnesses are never reported or compensated [5].

The insurance premiums paid by a firm under the Workers' Compensation system are generally not experience rated; that is, they do not reflect the individual firm's job safety and health record. About 80 percent of all firms are ineligible for experience rating because of their small size. Such firms are class rated, and rate reductions are granted only if the experience of the entire class improves. Even when firms have an experience rating, the premiums paid may not accurately reflect the true economic losses. Segregation of loss experience into classes is somewhat arbitrary, and an individual firm may be classified with other firms that have substantially different normal accident rates. An experience rating is generally based on the benefits paid to workers, not on the firm's safety record. Thus, employers may have a greater incentive to reduce premiums by contesting claims than by initiating safety measures.

In summary, the Workers' Compensation system suffers from several shortcomings that seriously reduce its effectiveness in providing incentives for firms to create safe and healthful workplaces. The scheduled benefits are significantly less than the actual losses to the injured workers, and recovery is often very difficult in the case of occupational diseases. Thus, the existence of a Workers' Compensation system limits an employer's liability significantly below the actual costs of the injury. In addition, premiums for individual firms are unlikely to be specifically related to that firm's risk environment. The firm, therefore, does not receive the proper economic signals and consequently fails to invest sufficient resources in reducing workplace injuries and illnesses. The economic costs not borne by the employer are borne by the employee or, as is often the case, by society through public insurance and welfare programs; in addition, the numbers of injuries and illnesses and the associated costs are greater than they could be.

Conclusion. OSHA believes that there are no nonregulatory alternatives that adequately protect construction workers from the adverse health effects associated with exposure to the chemicals regulated in this rulemaking. Tort liability laws and Workers' Compensation provide some protection, but due to market imperfections they have not been sufficient. Some employers have not complied voluntarily with standards recommended by professional organizations. The deleterious health effects resulting from continued high levels of exposure to hazardous substances require a regulatory solution.

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5. Technological Feasibility

Introduction. This section presents supporting data on the technological feasibility of complying with proposed permissible exposure limits (PELs) for the construction industry. Activities with potential airborne or dermal exposure to the substances to be regulated were identified across all categories of construction establishments and for different types of construction projects. Almost one hundred specific activities were evaluated for potential exposures and the appropriate means of protection. The activities and applicable control methods are described below.

Consistent with OSHA's methods of compliance hierarchy, engineering controls are recommended whenever they are feasible. In construction these include portable exhaust hoods, fans, fresh air blowers, enclosure of the worker or operation, equipment-mounted dust catchers, vacuum cleaning, and water spraying for dust suppression. For most activities, the proposed PELs can be met through a combination of engineering controls and appropriate work practices. Where engineering controls and work practices are infeasible or insufficient, respiratory protection may be used.

Many activities occur outdoors with natural ventilation. This helps to keep exposures low but does not ensure that exposures will be below the proposed PELs. For example, high exposures to lead result from the outdoor grinding, blasting, burning, or cutting of surfaces coated with lead-based paints.

Activities with Exposures—Abrasive Blasting. Abrasive blasting involves the removal of paint, dirt and other substances from surfaces that require cleaning prior to the application of a protective coating. Workers are exposed to the particulate matter generated by the blasting agent and from the surface material.

Four different types of abrasive blasting activities were identified based on the type of blasting agent. Abrasive blasting can be done with sand, aluminum oxide, steel shot, or slag, depending on the application.

In abrasive blasting operations using sand the operator would be exposed to silica (crystalline quartz and respirable dust) as well as concentrations of nuisance particulates. Abrasive blasting with aluminum oxide can cause exposures to aluminum metal dust in addition to silica and inert dust exposures. Abrasive blasting with steel shot or slag may involve exposures to iron oxide dust.

The use of blasting agents will generate additional exposures to substances contained in the surface material and in surface coatings, such as paint, that may be present. These may include lead, iron oxide dust, chromium metal, wood dust, and other particulates.

In the open-air type blasting which occurs commonly in construction, contamination of the general work area can occur if the blasting work is not contained. The blasting crew, including the operator and cleanup personnel, may be exposed to high dust concentrations depending on existing wind conditions. Open-air work makes it difficult to control worker exposures to airborne particulates, especially to the blasting operator. In order to control the exposures, vacublast units which incorporate local exhaust ventilation at the point of operation can be added as an integral part of the sandblast equipment, and a sandblast hood with corresponding supplied air system can be used. In addition, screens and partitions and the use of dust suppressants during dry sweeping of used blasting agents are work practices that reduce the operators' exposure and limit exposure to workers in adjacent areas of operations.

A NIOSH Health Hazard Evaluation reported the workday exposure results obtained by a private industrial hygiene consulting firm during the sandblasting of the Golden Gate Bridge in San Francisco, California in 1981 [1]. Two air samples for lead and one air sample for crystalline silica were collected. The lead concentrations of the two samples collected outside the worker's supplied-air hood were 5.54 mg/m^3 and 8.42 mg/m^3. The exposure to crystalline silica inside the hood was 0.218 mg/m^3. The total dust obtained from the sample inside the hood was 2.74 mg/m^3.

The exposure to the worker occurred because the facepiece seal became ineffective and the lens of the helmet had to be replaced. NIOSH stated that "in analyzing the data, it seemed evident that excess exposures to lead and silica could occur, but that proper work practices which included the use of the two lens hood should take care of the potential health hazard."

CONSAD evaluated worker exposures to lead and silica quartz dust during the sandblasting of interior metal walls made of corrugated sheet metal at a coal preparation plant near Waynesburg, Pennsylvania in July, 1990 [2; Firm 32]. A crew of ten people was involved in the work and seven of these did sand blasting.

The results of the personal samples for lead indicated that the concentrations inside the hood were less than 18 μg/m^3 for each worker. The 8-hour TWA exposures were less than 8 μg/m^3.

The sampling results for the exposures to respirable silica quartz dust indicated airborne concentrations outside the hoods that ranged from 0.09 mg/m^3 to 1.2 mg/m^3. The resultant 8-hour TWA exposures for the workers ranged from 0.08 mg/m^3 to 0.44 mg/m^3. The controls in use provided workers with sufficient protection from the levels of airborne respirable silica quartz dust present.

All seven of the sand blasters wore supplied-air helmets and capes. The air supply was provided from an on-site compressor. All employees were wearing safety glasses, hard hats, and hearing protection. No other exposure control measures were utilized. It can be concluded that all of these workers were within the proposed permissible exposure limits and that exposures during abrasive blasting with sand can be adequately controlled with available technologies.

The use of aluminum oxide as an abrasive agent during compressed air blasting provides certain surface preparation characteristics that cannot be achieved with other materials. Workers using aluminum oxide must be protected to the same degree as in other blasting operations with a focus on the airborne exposure to the metal dust generated and to the contaminants contained on the work surface. The exposure potentials and capabilities for control are similar to those for sandblasting. Controls include enclosure of the activity, equipment with built-in exhaust ventilation, dust suppressants, and supplied-air hoods. These methods of protection can ensure that exposures are kept below the proposed PELs.

The use of steel shot blasting is common for the cleaning of concrete surfaces. The shot blasting activity is enclosed within the area of the blasting machine. Machines are commercially available that operate continuously with recycling of the shot pellets. Due to the dusty nature of the operation, workers may be exposed to silica and other

substances present in the surface to be blasted. In addition to dust-controlling machinery and temporary enclosures, exposures can be controlled through the use of dust suppressants and supplied-air hoods.

Worker exposure to total silica dust was evaluated by CONSAD during the preparation of a concrete floor prior to coating. This activity was observed during a site visit near Harrisburg, Pennsylvania in July, 1990 [2; Firm 29]. The assessment consisted of sampling of workers while operating a Blastrac portable shotblast system to clean a concrete surface of approximately 1,600 square feet. The shotblast unit contained a vacuum system for collecting generated debris.

Samples were collected on the shotblast operator and two other workers in the area and were analyzed for quartz. It was reported that the samples contained an average of 30 percent quartz. The total dust samples for the shotblast operator resulted in 8-hour time weighted averages of 10.0 mg/m^3 and a 12.0 mg/m^3 exposure during sampling. On the basis of the average percentage of silica quartz determined from the samples, exposures to the shotblast operator exceeded the proposed 8-hour TWA standard for total silica quartz dust. The shotblast operator wore personal protective equipment that included a disposable dust respirator, safety glasses/goggles, and safety shoes. The disposable dust respirator was adequate for protection from nuisance dust but was inappropriate for this particular application. General ventilation was provided from a small wall fan that was in constant operation. No other control measures were observed. Adequate protection from the silica quartz dust could have been achieved through the use of appropriate respiratory protection.

Abrasive blasting activity may also involve use of a slag (also commonly known under the trade name "Black Beauty") as the abrasive material. The slag consists of iron oxide and silica (quartz). Workers performing this activity have the potential for exposure to the surface coating, surface components and the abrasive. The engineering and work practice controls discussed above are also applicable to this activity and have been successful in keeping worker exposures below the relevant proposed PELs.

Worker exposures to lead dust and other particulates were evaluated by CONSAD during the abrasive blasting of a bridge near Scranton, Pennsylvania in April, 1990 [2; Firm 15]. The operation involved compressed air blasting of a bridge containing a lead-based paint. The abrasive agent used was "Black Beauty."

Two abrasive blasting workers were evaluated for their exposure to lead dust during the actual blasting activity. Three individuals characterized as blaster, flagman, and clean-up laborer were also evaluated for nuisance particulates.

The airborne lead dust exposures of the blasters were evaluated as 0.040 mg/m^3 for a 346 minute sample and 0.250 mg/m^3 for a 328 minute sample. The determination of the exposure to nuisance particulates revealed that one of the blasters and the flagman were exposed to airborne total dust concentrations of 2.53 mg/m^3 and 1.89 mg/m^3 on an 8-hour TWA basis, respectively. The clean-up worker was exposed to an 8-hour TWA total dust concentration of 42.75 mg/m^3.

The abrasive blasters used personal protective equipment that included air-supplied helmets, full body covering, gloves, and safety shoes. The flagman and clean-up laborer wore a dust mask and a half-mask cartridge respirator, hard hats, and safety shoes. In this project, use of appropriate protective equipment kept worker exposures below the PELs.

Worker exposure to lead dust and particulates not otherwise regulated (PNORS) were evaluated by CONSAD during a bridge renovation project in Pittsburgh, Pennsylvania in June, 1990 [2; Firm 31]. A work crew of six people was engaged in the bridge work. Of the six workers, two of the workers were engaged specifically in the abrasive blasting aspect of the work. Again, the abrasive agent "Black Beauty" was used.

The two blasters were assessed during a two-day period for exposures. Samples were collected from both inside and outside the blasters' protective hoods. The results of the samples indicated that inside the hood, airborne lead concentrations ranged from less than 0.011 mg/m^3 to under 0.015 mg/m^3. The translation of sampling period exposures to 8-hour TWAs resulted in full shift concentrations of less than 0.008 mg/m^3. The samples taken outside of the hood indicated potential lead exposures that ranged from 0.036 mg/m^3 to 1.10 mg/m^3. The equivalent 8-hour TWA concentrations were evaluated as being up to 0.264 mg/m^3. On the basis of the determination derived from inside the hood measurements, both of the abrasive blasters were within the proposed standard for lead.

These same two workers were also evaluated for their exposure to total dust. The analytical results indicated concentrations inside the hood of 0.70 mg/m^3 and 4.49 mg/m^3 for each of the blasters on an 8-hour TWA basis. The proposed PEL for PNORS is 15 mg/m^3. It can be concluded that the blasters were within the proposed total and respirable dust standards for PNORS.

Specific measures were taken to minimize exposure to the dust generated during the blasting operation. Prior to the commencement of work, a dust curtain was built around the blasting operational area. In addition, personal protective equipment was used by the work crew that included: respiratory equipment (supplied-air helmets with capes or disposable dust respirators), work gloves, hard hats, and hearing protection. The air supplied for the blaster helmets was provided by a compressor on site.

Roofing and waterproofing operations. Several specific activities involved in these operations were evaluated individually based on the types of substances present and the potential for exposures. These activities include: application of an asphalt roofing system, application of single ply membrane systems, application of polyurethane roofing systems, application of modified bitumen roofing systems, application of asphalt waterproofing, and tear-off operations.

An asphalt built-up roof system involves the use of hot asphalt as the cement and sealer for the roofing system. In a built-up system, roofing paper is rolled out over the roof's surface in sufficient layers to meet the specifications of the contract (e.g., 4, 5, or 6 ply roof). Hot asphalt is pumped from the asphalt heating kettle and mopped on the adjacent seams to cement the layers. This is followed with the mopping of asphalt over the entire roof surface. Exposures to asphalt (and its benzene soluble components) are likely during the mopping process and also to any workers at the kettle during the heating of the asphalt.

Worker exposures are not likely to exceed the proposed PEL of 5 mg/m^3 at the mopman and kettleman operations. Engineering controls to reduce the mopman's exposure do not appear to be practical and therefore a respirator would be required in potential overexposure situations. A half-mask respirator equipped with organic vapor cartridges would provide adequate protection. Insulating the pipes from the kettle or tank truck to the roof or reducing kettle temperatures will likely reduce the kettleman's exposure. Keeping the temperature of the kettle as low as possible can help keep worker exposures below the PEL.

A study sponsored by the National Roofing Contractors Association and the Roofing Contractors Association of Southern California evaluated both hot asphalt and hot coal tar pitch exposures to roofing workers in 1981 [4].

The study consisted of the collection of 211 personal samples (98 percent from hot emissions) from 16 work sites. Analyses of the pitch and asphalt samples indicated that the majority for both types of materials was below a total particulate concentration of 5 mg/m³. Other findings from the sample analyses regarding benzene solubles were that (1) 74 percent of the kettlemen samples exceeded 0.2 mg/m³, (2) 49 percent of the total pitch samples and 37 percent of the asphalt samples were below 0.2 mg/m³, and (3) 20 percent of the samples exceeded 1.0 mg/m³.

It was concluded based on observations during the sampling that the roofing industry could take steps to insure that 80 percent of the workers would be below a benzene soluble limit of 0.2 mg/m³. The recommendations to accomplish this included: (1) maintaining the prescribed hot material temperature, (2) selecting appropriately sized equipment and repairing broken equipment, and (3) training workers to incorporate work practices to minimize exposure potential (e.g., working upwind).

The study also concluded that reducing all exposures to below 0.2 mg/m³ without the use of respirators would be a difficult task. The factors cited for this were: (1) uncontrollable site ventilation—some workers could not avoid exposure because of wind direction and work pattern, and (2) some tasks involve a high exposure potential. The data indicated that exposures for virtually all workers would be below the proposed PEL of 5 mg/m³. A remedial action sufficient to control overexposures is the use of a filter mask for workers known to be exposed to excessive hot emissions.

NIOSH conducted an environmental survey during the application of a petroleum asphalt roof at a site in Houston, Texas in 1983 [3]. The survey was conducted during a two day period. Petroleum asphalt plugs were melted in a heated metal container. The liquified asphalt was transferred to a hot application vessel and mopped on to the prepared roofing surface.

Eleven samples were obtained during the two day period and evaluated for benzene solubles. The airborne concentrations ranged from 0.110 mg/m³ to 1.440 mg/m³. The highest airborne concentration reported was incurred by the kettleman.

A number of measures recommended by NIOSH to minimize contact with pitch would apply to asphalt as well. These included: (1) roofers should stay upwind of the pitch fume as much as possible, (2) showers should be taken after work each day and workers should have a change of clothes after showering, (3) workers should wear clean clothes each day, (4) workers should use waterless cleansers, and (5) personal protective equipment should consist of long-sleeved shirts that are tight-fitted at the wrists, full-length pants that extend to the shoes, goggles, gloves, and respirators. Respirators should be approved for protection from organic vapors. The application of these control methods would provide adequate protection to workers and enable compliance with the proposed PEL.

NIOSH conducted an environmental survey for the U.S. Corps of Engineers, Kansas City Supply Depot, in 1983, to evaluate working conditions during a re-roofing job [5]. ASTM Type III asphalt was used as the roof cement material. The survey was conducted over a three day period. The asphalt was received in metal containers and placed in a 300 gallon propane-fired kettle for melting. The material was heated to about 450 degrees Fahrenheit and mechanically pumped to the roof. The hot asphalt material was stored in a hot lugger until needed. The lugger was wheeled to the point of application where the asphalt was dispensed into mopping buckets. The asphalt was mopped onto the plies of roofing felt that were previously laid over the roof deck structure. A final flood coat of asphalt was then mopped over the entire surface. This was followed by the application of a layer of gravel that completed the finishing of the roofing surface.

The full shift average exposures to asphalt fume for the kettleman and the crew were 0.83 mg/m³ and 0.31 mg/m³, respectively. Recommendations to reduce exposures included: the scheduling of work to reduce the number of employees exposed, locating the kettle and work areas to reduce exposure to fumes, and keeping the temperature of the kettle at an absolute minimum necessary to meet material specifications.

CONSAD conducted a site visit to assess the exposure to asphalt fume during the heating and application of petroleum asphalt for a 4-ply built-up roof system in Washington, D.C. in January, 1990 [2; Firm 01]. Four workers were monitored during the heating and hot asphalt application. Specifically, these workers can be characterized as kettleman, material handler, paper setter, and mop man. The sampling results showed worker exposures ranging from 0.06 mg/m³ to 0.85 mg/m³ on an 8-hour TWA basis. None of the four workers had exposures which exceeded the proposed PEL of 5 mg/m³.

The installation of a single ply membrane rubber-based roofing system involves the use of sprayed, rolled, and brushed-on adhesives. These adhesives are applied following the mounting of the insulation to the roof decking and the layout of the rubber roof membrane. The adhesive is sprayed on the back of the membrane and rolled by workers that follow the sprayer. The seams of the membrane are brush coated with a lap seam adhesive and hand rolled. The workers are exposed to the solvents contained in the adhesives, including acetone, n-heptane, n-hexane, toluene, and xylene.

With the natural ventilation present when working outdoors, the likelihood of overexposure to any of the described organic solvents is considered to be minimal. Engineering controls on an open roofing application project do not appear to be practical or feasible and therefore controls would be limited to respiratory protective equipment if necessary. For exposures that are relatively high, disposable organic vapor masks would be appropriate.

NIOSH conducted a field survey to evaluate the potential of worker exposures in the application of a single ply rubber roofing system in Dayton, Ohio in 1983 [6]. Thirteen personal breathing zone samples were taken on two to six workers performing the application as described above over a four day period. The samples were analyzed for the solvents contained in the roofing adhesives: xylene, toluene, acetone, ethyl benzene, and hexane.

The analytical results for the thirteen personal samples were compared to OSHA's proposed standards. It was determined that the combined exposure to each worker was below that allowed by the proposed PELs. This indicates that the exposures to the workers were within OSHA's proposed limits for each chemical and below the relevant mixed exposure limit, and that compliance with the proposed PELs is feasible for this activity.

Other studies by NIOSH as well as data gathered by CONSAD during site visits confirm that exposures to airborne contaminants during the application of single-ply membrane systems are generally well below the proposed PELs [7; pp. 3.18–3.24]. If necessary, workers can be protected from exposures with respirators.

The application of a modified bitumen single ply roof involves possible exposures to solvents, asphalt, and other airborne substances. The modified bitumen roofing membrane is comprised of several layers; a rubberized or plasticized asphalt sheet, a glass fiber or plastic support and a protective film. These membranes are applied by torch fusing, by setting in hot asphalt or by cold setting on a primed surface.

The likelihood of overexposure to airborne contaminants such as asphalt fumes, nuisance particulates, or organic solvents is considered remote in this activity due to the method of application and the outdoor nature of the work. Control options such as local exhaust ventilation on an open roofing application project are not believed to be practical or feasible and therefore controls would be limited to improved work practices and respiratory protective equipment. For moderate exposures, disposable organic vapor masks would be appropriate with potential upgrade to half or full-face organic vapor respirators for elevated exposures.

CONSAD observed the installation of a modified bitumen roof system during a re-roofing project in Washington, D.C. in March, 1990 [2; Firm 08]. The installation procedure was conducted on two consecutive days. During the first day, workers applied a cold-set adhesive to the roof deck and then covered the deck with a layer of roofing paper. This was followed by the setting of a layer of perlite insulation on top of the roofing paper. On the second day of work, the modified bitumen roof membrane was laid out over the insulation and heat set into place. To heat set the roofing membrane, workers used a propane torch heater.

Five workers were sampled for their exposure to the solvents (stoddard solvent, styrene and toluene) used during the application of the cold-set adhesive during the first day of work. On the following day, three workers were sampled for their exposure to asphalt fumes during the installation of the roofing membrane. Sampling duration associated with the tasks of each day's work was approximately four hours.

For the workers engaged in the application of the adhesive, the airborne concentrations of the solvents contained in the adhesive were non-detectable. The three workers engaged in the heat setting of the roofing membrane incurred 8-hour TWA exposures to asphalt fume that ranged from 0.04 to 0.26 mg/m^3 on a total particulate basis. It is unlikely that worker exposures during the two days of this operation exceeded any of the proposed PELs.

The application of polyurethane roofing is similar to that of polyurethane foam insulation applications. Polyurethane systems are generally comprised of two part mixtures that are stored separately in 55 gallon drums. One component may consist of polymeric and monomeric forms of an isocyanate or diisocyanate and the other component consists of a mixture containing the curing agent, catalyst, solvent, and foaming agent. The mixtures are pumped from the drums to the mixing chamber of the spray gun and atomized onto the roof surface to a thickness of several inches.

The spray operator and helper(s) engaged in the foam application are exposed to airborne concentrations of the components of the specific product used. The important components may include: methylene bisphenyl isocyanate (MDI), toluene diisocyanate (TDI), alpha-methyl styrene, and fluorotrichloromethane. CONSAD found no evidence indicating that significant exposures occur during this application.

Roof tear off operations involve the removal of the old roofing materials that may contain petroleum asphalt, fiber board insulation, and asbestos flashing. The removal of the old roof is a manual operation. However, power saws are used to cut the roof into manageable pieces for removal.

Based on the data available for roof tear-off operations, exposure to airborne particulates generated during this activity can be controlled below the PEL if the roof is thoroughly wetted. Dust exposures can be controlled in most situations with the use of water, water spray, and wet sweeping work practices. If exposures exceed the PEL for particulates other than asbestos, then the use of a NIOSH approved disposable particulate respirator would be necessary. These respirators should provide adequate protection during this activity. If the roof consists of asbestos-containing materials then half-mask respirators with HEPA filters should be worn.

NIOSH conducted a survey to evaluate worker exposures to total dust and benzene solubles during a roof tear-off in Hamilton, Ohio in 1983 [8]. The tear-off work commenced with the breaking up of the old roof down to the insulation. This was done using a power cutter. This was followed by the separation and removal of the old roofing material. In this step, workers pry the old roofing material from the support structure while using shovels and other hand held prying tools. The material was then carted to the edge of the roof where it was discarded through a plastic chute.

Six breathing samples were collected during the tear-off operation from a crew of thirteen workers. The sampling time ranged from about 3½ hours to about five hours. The total particulate concentrations that were reported ranged from 2.1 mg/m^3 to 13.1 mg/m^3. The analytical results also showed concentrations of benzene solubles ranging from 0.6 mg/m^3 to 5.3 mg/m^3. All six of the workers were exposed to total particulate concentrations less than the proposed limit of 15 mg/m^3, and one of the workers exceeded the proposed OSHA standard of 5 mg/m^3 for benzene solubles during the tear-off operation. Although the roofing material contained coal tar pitch, the exposure levels are indicative of potential exposures to the benzene-soluble fraction of asphalt fume during similar activities.

NIOSH recommended a number of improvements that could be instituted to alleviate worker exposure to dust during the removal of roofing. It was advised that water should be used to thoroughly wet the surface of the roof prior to and during tear-off. The use of a vacuum system was recommended in comparison to power brooms and power blowers to remove small debris. Roofers should also stay upwind of the dust during tear-off if possible. OSHA believes that these work practice controls would be sufficient to keep exposures during this activity below the proposed PELs. For highly exposed workers, respiratory protection should be worn.

CONSAD conducted a site visit to assess exposures to fibers during roof tear-off operations in Washington, D.C. in January, 1990 [2; Firm 01]. Two employees were engaged in the tear-off operation for the entire shift; both employees were sampled. The roof tear-off activity consisted of using a power cutter to break up the roof into manageable pieces and then prying the material from the deck using hand tools. The waste material was loaded onto a wheelbarrow and transported to the edge of the roof where it was removed from the roof by crane lift.

The sampling was conducted to assess the worker's exposure to fibrous glass dust. The fibrous glass dust was generated from the loosening of the insulation material that was in place below the roofing.

The results of the personal sampling showed that both workers were exposed to total dust at less 0.1 mg/m^3 as an 8-hour TWA. Because the roofing was

thoroughly wetted by heavy rain that had fallen the night prior to the sampling, it is assumed that neither of the workers would have exceeded the proposed PEL of 1 f/cc.

Worker exposure to particulates was evaluated during the tear-off operation of an asphalt roof observed by CONSAD [2; Firm 14]. The roof removal included the use of pry bars and a plow-like device to lift off the old roofing and then the transportation of the waste material to a hoist bucket for disposal. The removal of the old roofing was completed in less than four hours.

Personal samples were collected from two tear-off laborers and evaluated for total dust. The sample analyses indicated airborne concentrations of 1.95 mg/m^3 and 1.77 mg/m^3 for each sampling period of 215 minutes. These individuals were well within the existing and proposed total dust standards.

The application of asphalt waterproofing involves the use of asphalt-based waterproofing products. Workers engaged in this operation are in contact with the waterproofing material and in close proximity to the area of application. The exposure concerns associated with this activity are the asphalt fumes which may become significant in poorly ventilated areas. Exposures may also occur due to the presence of solvents contained in the waterproofing product. These may include: Benzene, ethyl benzene, petroleum distillates (mineral spirits), toluene, and xylene.

Since the worker is in close proximity to the area of application, there is a potential for overexposure to asphalt fume. Engineering controls to reduce worker exposures do not appear practical and therefore a disposable mask with NIOSH approval for organic vapors should be used when overexposures are a possibility. Maintaining the temperature of the waterproofing agent to the minimum required will help to decrease the airborne concentration of fumes produced. Exposures during this activity can be kept below the proposed PEL through the use of appropriate work practices and, if necessary, respiratory protection.

Insulation Installation and Removal. Applying and installing insulation can cause potential exposures to airborne concentrations of the material involved. Typical insulation materials include: Fiberglass, mineral wool, and foam. Insulating foams may be some type of polyurethane or a urea-formaldehyde product. The manner of applying or installing the insulation can be either a manual or mechanically assisted type operation. In the manual operation, the worker installs the insulation by hand (e.g., the installation of fiberglass batts, the wrapping of pipe, etc.). One mechanically assisted process involves fiberglass or mineral wool blown into place by a pneumatic blower. In another process, an insulating foam is produced and sprayed into place.

Exposures to the solvents, fiberglass, minerals and other particulate matter in installing insulation will be highly variable dependent upon the nature of the job site. Exposures will vary depending on whether or not workers are in a relatively confined area, such as an attic, or in a well ventilated area, such as an open bay structure. When work is conducted in a relatively confined area, a fresh air supply would be likely to eliminate any overexposure to the solvent portions of the various application methods. In confined areas where insulation is being blown in place, it is possible that respiratory protective devices will be required to control the particulate and/or fiber exposures.

There is a greater potential for significant worker exposure to fiberglass and mineral wool dust generated by the blown-in process than in the manual process. There may also be exposures to the substances contained in the mechanically assisted spraying application. There may be additional exposures encountered in the manual process if any adhesives are used to fasten the insulation to equipment, pipes or ducts. These exposures generally consist of the solvents contained in the adhesive.

A study prepared for NIOSH was conducted on polyurethane foam application activities [9]. Personal samples were collected from two workers to determine their exposure to MDI (methylene bisphenyl isocyanate), fluorotrichloro-methane, alpha-methyl styrene, dimethylcyclohexylamine, and total particulates during the foam application. The application of the foam was made to the interior walls of a refrigeration room of a food warehouse that was under construction. A personal sample was also taken on both workers to determine their exposure to 2-ethoxyethanol. The 2-ethoxyethanol is reported as not being extensively used during application but was used during a repair period.

The results of this study showed that both of the workers exceeded OSHA's proposed MDI ceiling level of 0.02 ppm during foam application. None of the other chemical substances exceeded OSHA's proposed exposure limit. NIOSH concluded that engineering controls to reduce MDI levels during indoor foam application would be desirable but because of the highly mobile nature of the spraying operation, engineering controls could not be effectively applied [7; p. 3.27]. NIOSH also stated that the use of conventional fans and ducting would be impractical because of the tendency of foam particles to adhere to surfaces. As a result of these difficulties, NIOSH recommended the use of a Type C supplied air respirator with full facepiece, operated in the positive pressure mode during the indoor foam spraying of MDI.

Another report that was prepared for NIOSH discusses the exposures associated with the application of mineral wool blankets to an effluent combustion gas duct [10]. Two workers were monitored for mineral wool fibers, total suspended particulate matter, and respirable particulate matter. The average airborne concentrations during the work shift for all personal samples were 0.076 f/cc for fiber concentrations, 2.271 mg/m^3 for total particulates, and 0.871 mg/m^3 for respirable particulates. The exposure concentrations to mineral wool fiber were within OSHA's proposed PEL of 1 f/cc. Approved disposable respirators were supplied to the workers and their continued availability was recommended.

This report also discusses the exposures associated with "blown-in" mineral wool insulation [10]. In the report, two workers were monitored for their exposures to mineral wool fibers and total airborne particulate matter.

The installers used their truck that contained a gasoline-powered blower into which the bagged insulation was emptied. The hopper was tended by one of the workers during the blowing process. The insulation was loosened within the hopper and blown by a centrifugal fan through a hose to a house attic where it was applied. The second worker was stationed in the attic and directed the depth of the insulation applied.

The personal sampling results of the worker in the truck showed the following exposures: Mineral wool fibers, 0.087–0.238 f/cc; particulate matter, 0.910 mg/m^3. The monitored results of the worker stationed in the attic were 0.858–1.359 f/cc for mineral wool and 7.76 mg/m^3 for total particulate matter. It can be concluded that without the use of disposable respirators the workers' exposure to mineral wool in this study would have exceeded OSHA's proposed fiber standard of 1 f/cc (fiber count based on aspect ratio of 3 to 1). The workers' exposures to the metals contained in the

dust were within the respective proposed limits.

In another report prepared for NIOSH on the same insulation application process [10], the sampling results were reported as 0.035 f/cc mineral wool and 0.01 mg/m³ total particulate for the truck worker, and as 0.552 f/cc mineral wool and 41 mg/m³ total particulates for the applicator. The use of appropriate respiratory protection would enable worker exposures to be kept below the proposed PELs in such conditions.

CONSAD conducted air sampling of a worker involved in the installation of fiberglass batts during the renovation of an institutional facility in Aspen, Colorado in June, 1990 [2; Firm 24B]. The insulation worker placed the batts between the studs and up against one side of a dry wall panel. The fiberglass installer was sampled for 285 minutes.

The results of the breathing zone sampling showed that the insulator had been exposed to an 8-hour TWA concentration of 7.21 mg/m³ total particulate. The airborne concentration during the sampling period was 12.14 mg/m³. This worker's exposure to total dust would be considered in compliance with the proposed PELs. A fiber count exposure estimate was attempted but was not determined because most of the particulates were not fibrous. The fiberglass installer wore a disposable dust mask, hard hat, and safety shoes during the application. This equipment was considered sufficient for protection from exposures above the proposed PELs.

CONSAD conducted personal air sampling during the installation of fiberglass wool insulation in two residential structures in Panama City, Florida in June, 1990 [2; Firm 26A]. Two workers were involved. One of the workers was stationed in a truck and fed the fiberglass insulation into a Volumatic II pneumatic blower. The other worker controlled the application of the insulation within the attic spaces of the residences.

The workers were evaluated for exposure to airborne fiber concentrations at one of the residences and total particulate exposures at the other. The length of the sampling period was 90 minutes for fiber determinations and 106 minutes for total particulates.

Under the NIOSH "B" counting rules, it was determined that the sprayer operator was exposed to airborne concentrations of 1.7 f/cc and 0.3 f/cc during two individual sampling periods. Based on these airborne concentrations, the equivalent 8-hour TWA was determined to be 0.6 f/cc. It was further determined that this worker was exposed to a total particulate concentration of 11.5 mg/m³ at the second residence. The equivalent of an 8-hour TWA of 10.1 mg/m³ was estimated based on the spraying of four attics per day of equivalent size to the one observed.

Under NIOSH "B" and OSHA counting rules, it was determined that the insulation feeder operator was exposed to 0.4 f/cc and 0.2 f/cc in the two sampling periods. These values were also determined as being equivalent to 8-hour TWA exposure concentrations. The airborne exposure to total particulates was determined to be 0.85 mg/m³ which resulted in an equivalent 8-hour TWA of 0.74 mg/m³. Exposures for both of the workers were below proposed PELs.

The application of fireproofing material is similar to that of insulation. Fireproofing material is applied to steel structural members and the metal deckings of buildings. Fireproofing material that is similar to a mortar or plaster is prepared by filling a mixer with the bagged dry material and adding water. The material is then pumped to the point of application and is sprayed on using compressed air. Workers who are engaged in the mixing process are exposed to particulates such as gypsum, plaster of Paris, and calcium carbonate during the dumping of the bags into the mixer. Fireproofing applicators are potentially exposed to particulates from the spray and dispersion of the fireproofing material.

Mixer areas can be enclosed in wood or plastic on wood studding and placed under negative pressure using a portable exhaust system. By doing so, dust generated during the dumping of dry bagged ingredients would be contained within the enclosure and safely exhausted to areas remote from the operations. If any bulk raw materials are used they should be kept wet prior to adding them to the mixer. Water sprays or wet sweeping should be used to clean up any spilled materials to reduce ambient concentrations of dust. Where these controls are not effective to reduce exposures below the PEL, respiratory protection in the form of disposable dust mask would be required.

While selective solvents may be added to the batches as curing agents, the quantities of such materials are small in proportion to the overall mix. In conjunction with the wet method of application, the likelihood of worker exposures in excess of corresponding PELs is remote.

CONSAD conducted a site visit to evaluate exposures to workers engaged in the application of fireproofing material in Indianapolis, Indiana in March, 1990 [2; Firm 07]. At this particular site, a fireproofing material consisting of gypsum, cellulose, fibrous glass, and less than 3 percent quartz was sprayed on to structural members and exposed steel floor decking. Three crews consisting of four workers were involved in the activity. Each crew consisted of one mixer operator, one spray applicator, and two workers who transported the spray applicator on a portable scaffold. Two mixer operators and two sprayers were sampled for exposures to total silica dust.

The sample results showed that the percentage silica in the dust was less than one percent. The personal samples of the mixer operators were 30.7 mg/m³ and 87.2 mg/m³ on an 8-hour TWA basis. These exposures exceeded both the existing and proposed total dust standards. However, the sample results of the spray applicators were evaluated at 8-hour TWAs of 1.8 mg/m³ and 3.34 mg/m³.

Mixer operators doing this activity have a greater potential for overexposure than do the spray applicators. Mixer operators handle and dump dry material from bags; this activity is the primary source of airborne exposure. However, once the material is wetted in the mixing process, the potential for overexposure is reduced significantly. As discussed above, standard controls and work practices for handling dusty materials should be employed. The use of appropriate engineering controls, work practices, and if necessary, respiratory protection, can reduce worker exposures during this activity to levels below the proposed PELs.

Insulation removal activities can be divided into those involving asbestos and those where asbestos is not present. In addition to asbestos exposure, workers may experience exposures to plaster based materials such as gypsum, calcium sulfate, calcium carbonate and particulates not otherwise regulated. Further, the use of organic solvents and strippers to facilitate removal may involve potential exposures.

Specific regulations exist concerning the removal of asbestos-containing insulation. These regulations outline strict controls when handling asbestos-containing material. The controls include: total enclosure with exhaust (negative pressure) of the work area, wetting of the insulation prior to removal, and use of respiratory protection to help prevent inhalation of fibers. The control of asbestos fibers should ensure compliance with the proposed PELs for other substances.

In the removal of insulation not containing asbestos, the relevant materials involved are generally fiberglass or mineral wool compositions. The manual removal of such materials will generate particulate matter comprised of these materials. Workers engaged in this activity will have the potential for exposure to these substances and other substances that may have to be removed so that the insulation can be accessed, such as gypsum, calcium sulfate and calcium carbonate. Further exposures may be incurred if the insulation is secured by adhesives and organic solvents or strippers are needed to facilitate the removal.

Exposure to airborne particulates during this activity can most likely be controlled through a combination of engineering and good work practice controls. The engineering controls consist of constructing a total enclosure of the work area. Air should be removed from the enclosure by attaching an exhaust unit. Prior to removing the insulation, it should be thoroughly wetted with a water spray to suppress dust generated during the activity. Debris on the floor should be cleaned by wet sweeping work practices. If these or similar controls can be implemented, the use of respirators would most likely not be required.

Painting and Other Exposures to Surface Coatings. Different activities causing exposures to paints were evaluated separately based on differences in work conditions, control potentials, and substances present. The activities identified include exterior painting of masonry finishes, exterior painting of wood finishes, exterior painting on steel, grinding/drilling/cutting of painted surfaces, interior application of floor coatings, interior painting of metal substrates, interior painting of walls and ceilings, and surface cleaning and stripping.

The painting of exterior masonry can be performed by the usual methods of application: brush, roller, and spray. The paint is the source of exposure to workers engaged in this activity and the type employed will determine the specific chemical exposure. Alkyd and epoxy type coatings are generally used for this application. The solvents contained in these coatings constitute the primary exposure hazard but unreacted epoxy monomer, e.g., epichlorohydrin, may also be present.

Exterior painting applications (spray, roller, brush) result in at least some exposure to solvent vapors. The degree to which painters may be exposed to solvent vapors is dependent upon several factors: The method of application, nature of the work area (enclosed space or open area), and employee work practices. In most situations with adequate ventilation, exposures are likely to be below the relevant proposed PELs. When conditions are such that exposures become concentrated, reducing employee exposures will most likely involve the use of respiratory protection because engineering controls are often difficult to implement. For solvent vapors, a half-mask organic vapor cartridge respirator would be sufficient to protect workers from overexposure. Some paints contain chemical substances (epichlorohydrin, methyl alcohol) for which these types of respirators will not provide adequate protection. In this situation, an air-supplied respirator may be necessary.

Some paints also contain substances for which OSHA is proposing a "skin" designation. Two such substances include methyl alcohol and mercury (aryl and inorganic mercury compounds). Natural rubber or neoprene gloves provide adequate skin protection against these substances in most instances.

The exterior painting of wood is assumed to be applied either by brush or roller methods. The coatings used for wood finishes consist of stains, varnishes, lacquers, paints, and primers. The hazardous exposure potential from coating material is likely to be the solvent(s) contained in the product.

In most applications, exposures are expected to remain below the proposed PELs. As discussed above, implementation of engineering controls are often difficult with exterior painting operations. In the event of concentrated exposures, respirators would offer protection from potential overexposures.

The exterior painting of steel can be performed either by brush or spray methods. The type of paint used will be dependent upon the corrosiveness of the environment. Workers engaged in the activity are likely to experience some exposure to the solvent(s) of the product that is applied. The use of epoxy and urethane coatings may also present additional exposure risks to unreacted monomer residues, e.g., epichlorohydrin, methylene bisphenyl isocyanate (MDI), and toluene diisocyanate (TDI).

In construction, most painting of steel is done with conventional air spray equipment. Particulate and solvent vapor exposures in excess of allowable limits are possible due to overspray and rebound if adequate controls are not used. As with other exterior painting activities, implementation of engineering control measures is difficult. Most exterior painting on steel is conducted with employees wearing respiratory protection. For protection against inhalation of solvent vapors, a half-mask organic vapor cartridge respirator would be required. In the application of two component urethane and epoxy paint systems, operators may be required to wear air-supplied respirators.

Dermatitis and other skin conditions resulting from contact with solvents or thinners and sensitization from epoxy mixtures are potential health problems if skin contact is not minimized. Substances commonly found in paint for which OSHA is proposing a "skin" designation include: mercury, aryl and inorganic mercury compounds, n-butyl alcohol, and methyl alcohol. Natural rubber or neoprene gloves will provide adequate skin protection against these substances in most instances.

CONSAD conducted air sampling to evaluate the exposures with the use of a two-part epoxy-polyamide paint during a bridge renovation project near Scranton, Pennsylvania in April, 1990 [2; Firm 25]. Three painters were sampled for petroleum distillates (mineral spirits) and xylene while spray painting the structural members of the bridge.

Exposures to petroleum distillates of 0.83 mg/m^3 TWA8 were observed for the three painters. The 8-hour TWA exposures to xylene ranged from 0.313 ppm to 1.875 ppm. The maximum level of exposure obtained during sampling was 9 ppm. Worker exposure to these substances was considered to be adequately controlled because airborne concentrations were less than 10 percent of the proposed PELs. The painters utilized personal protective equipment that included air-supplied helmets, coveralls, gloves, and safety shoes.

CONSAD also observed the exterior painting of a steel bridge during new construction of a commuter rail transit line in Chicago, Illinois in May, 1990 [2; Firm 20]. The work consisted of the application of a base coat and top coat by the conventional compressed air spraying method. The painting operation was surveyed over a two day period. The base coat was a two-part epoxy that contained aluminum metal pigment and solvents (xylene and toluene). The top coat product, a two-part polyurethane, contained xylene, n-butyl acetate, cyclohexanone, hexone (MIBK), toluene, and hexamethylene diisocyanate.

One personal air sample was obtained for each of the three components during the base application. The analytical determination for the airborne presence of aluminum indicated an exposure of 0.42 mg/m^3 as an 8-hour TWA. The

proposed PEL for aluminum metal dust is 15 mg/m³ on a TWA8 basis.

The exposure to xylene during the base coat application was calculated at 45 ppm TWA8. The airborne concentration of toluene indicated a breathing zone exposure 5.6 ppm TWA8. It was concluded that no exposures above the proposed PELs occurred during this activity.

During the application of the top coat, two of the painters were sampled for each of the substances identified above except for hexamethylene diisocyanate (HDI), for which there is no proposed PEL. The sampling results in parts per million as eight hour time weighted averages are as follows:

Substance	Painter #1	Painter #2	Proposed PEL
N-butyl acetate	5.0	5.3	150
Cyclohexanone	0.1	0.1	25
Hexone (MIBK)	8.0	7.5	50
Toluene	0.7	0.6	100
Xylene	20	18	100

It can be concluded that all of the painters sampled during the base and top coat applications were within the proposed individual limits. It can also be concluded that the combined equivalent exposure based on the proposed standards was less than unity and therefore acceptable.

All of the painters under consideration employed respiratory devices. The base coat applicator wore a supplied-air helmet, while the top coat painters used half-mask organic vapor cartridges. The base coater also wore a cape, work gloves, and safety glasses. Both of the top coat painters used work gloves and wore long-sleeved shirts. Only one of the top coat painters utilized eye protection.

Three painters and a flagman were evaluated by CONSAD on April 24, 1990 for their exposure to zinc dust during a bridge renovation project near Scranton, Pennsylvania in which a zinc-based primer was applied [2; Firm 15]. The primer was applied by the conventional compressed air spray method.

The three painters were exposed to airborne concentrations of zinc dust ranging from 3.64 mg/m³ to 11.20 mg/m³. However, since the work was only performed for about an hour, the equivalent 8-hour TWA exposures ranged from 0.33 to 1.17 mg/m³. The flagman was exposed to an airborne concentration of zinc dust of 0.38 mg/m³ during the work period. The proposed PEL for zinc oxide total dust is 10 mg/m³. Although none of the sampled workers had exposures which exceeded the proposed PEL on an 8-hour TWA basis, if painting operations last longer the potential for overexposure may be possible.

Personal protective equipment needed to protect the painters includes respirators, full body covering, gloves, and safety shoes. This equipment is considered sufficient for protection from overexposures.

Workers that grind, drill or cut painted surfaces are susceptible to exposures generated by the action of the tools on these surfaces. Workers engaged in this activity will be exposed to the dispersed particulate matter of the surface material (e.g., iron oxide, chromium, zinc dust, etc.) and constituents of the paint, (e.g., lead, chromium, and zinc).

The principal dust control technique for grinding operations is ventilation. Portable grinding tools can be effectively exhausted by equipment that is retrofitted directly onto the tool. One such technique that has been successful in controlling dust from grinding is a low-volume high velocity exhaust system. In addition to ventilation, the use of water on grinding, drilling, or cutting operations can also help reduce the airborne particulate generated during these operations. If engineering controls cannot adequately reduce airborne dust concentrations, then disposable dust mask respirators can be used to reduce exposure levels to below those required by the proposed rule.

Investigations of grinding, drilling, and cutting activities by NIOSH indicate that the potential for overexposure to several substances exists. One study of exposures during the power brushing of painted metal surfaces at a Connecticut nuclear power station in 1981 shows that 8-hour TWA exposures exceeded the proposed standards for lead, iron, and total particulates [11].

A 1983 technical evaluation conducted by NIOSH and the Massachusetts Division of Occupational Hygiene assessed lead exposures to workers on the Tobin-Mystic River Bridge project near Charlestown, Massachusetts [12]. Workers were evaluated while scraping lead-based paint with pneumatic and hand tools; airborne concentrations of lead at levels far above the proposed PEL of 50 μg/m³ were common. The use of appropriate respiratory protection, including positive-pressure air-supplied respirators, was recommended to adequately control worker exposures.

In addition to engineering and work practice controls, appropriate respiratory protection should be utilized to ensure that worker exposures do not exceed the proposed PELs for any of the regulated substances that may be present. The potential for airborne levels of lead to reach hazardous levels is often underestimated when dealing with painted surfaces.

During the interior application of floor coatings workers use a number of different coating materials specific to a given application. Flooring coatings include ordinary paint (such as an alkyd), and polyurethane coatings. Whichever coating is selected, worker exposures arise primarily from the epoxy and isocyanate monomers and the organic solvents contained in the coating products.

Floor coating operations that occur indoors may result in excessive organic vapor exposures in worker breathing zones when provisions are not made to supply adequate ventilation. Fresh air should be blown into the work area to dilute solvent vapors. For most of the common solvents found in floor coatings, half-mask organic vapor cartridge respirators provide sufficient protection. Although overexposure to any of the monomeric isocyanates or diisocyanates is unlikely, a supplied-air respirator would be the appropriate form of protection due to poor warning properties (odor threshold) associated with these substances.

Personal air samples were collected by CONSAD from a three man work crew over a three shift, two day period during the applications of epoxy and polyurethane coatings to a concrete floor near Harrisburg, Pennsylvania in July, 1990 [2; Firm 29]. Three individual applications that consisted of one epoxy primer coat, one epoxy levelling coat, and a finish coat of polyurethane were made. Each coating material was a two-part mixture that was prepared in five-gallon containers at the site. The application process requires that each coat of material be allowed to dry for approximately 6–8 hours before the next coat is applied. The workers used long-handled rollers and brushes (for trim work at the baseboards) during application.

During the applications of the epoxy coatings, the workers were evaluated for their exposure to xylene, hexone (MIBK), epichlorohydrin, and propylene glycol monomethyl ether. The airborne concentrations of these contaminants were within the proposed concentration limits during the sampling periods and well under the proposed TWA8 PELs.

This same work crew was also evaluated for their exposure to xylene, n-butyl acetate, and methyl n-amyl ketone during the application of the polyurethane finish coat. The sampling results showed that all of the solvent vapors evaluated were within the

proposed PELs during the sampling period. Xylene was the major contaminant and an airborne concentration of 22 percent of the proposed PEL was measured. Two of the workers were also evaluated for exposure to hexamethylene diisocyanate (HDI), an unregulated substance with no proposed PEL. The airborne exposure to this substance was below the level of detection of 0.00054 ppm.

During the application of the coating system, the workers used personal protective equipment that included respirators (MSA half-mask or full face with NIOSH approved cartridges) and rubber gloves. General ventilation was provided by a wall fan that was operated during the entire application time.

The interior painting of metal substrates can involve a number of different interior paints. The normal means of coating are applicable to this activity (e.g., brush, roller, and spray). The selection of the particular type of paint and application method will depend on the environmental conditions to which the metal will be subjected. Workers involved in this activity will be exposed to a number of organic solvents.

Interior painting of metal substrates is usually conducted with conventional air spray equipment. As data from two NIOSH studies indicates [11, 13], exposures to paint mist or total particulates can potentially exceed the relevant proposed permissible exposure limits. Ventilation techniques such as fresh air supply systems or blowers to direct airborne contaminants away from workers can be applied to reduce worker exposures. The use of respiratory protection may also be applied by some painting crew members. For protection against inhalation of the particulate portion of the paint (fillers, binders, pigments, etc.), a disposable respirator will provide adequate protection in most situations. For overexposures to the solvent portion of these coatings, a half-mask organic vapor cartridge respirator would be required. Painters often use a combination half-mask organic vapor cartridge respirator with a mist removing pre-filter. Some paints contain chemical substances (2-Nitropropane, methyl chloroform, trichloroethylene) for which these types of respirators will not provide adequate protection. In these instances, a supplied-air respirator would be necessary.

Painters that are engaged in the use of compressed air spray painting as an application method have a greater exposure potential to the product solvents and other substances than when using either a brush or roller. Data from NIOSH industrial hygiene surveys [11, 13] and findings by CONSAD from site visits [2; Firms 15, 20, 22, 23, 25, 30] indicate that worker exposures during each of these activities can be controlled to levels below the proposed PELs. The combined equivalent exposures to the organic substances identified were also evaluated as capable of being controlled within the proposed limits.

Workers involved in interior painting of walls and ceilings generally use either a brush or a roller technique, although spray painting is also possible. A house paint commonly used is water based latex, although oil based (alkyd) and epoxy paints may also be employed. There is the potential for workers to be exposed to a number of organic substances and solvents contained in these paint products.

If there is an overexposure problem when conducting this activity, it most likely would occur during spray application methods. Inhalation hazards are increased due to overspray and rebound. In work situations involving considerable spraying in areas with poor natural ventilation, fresh air should be circulated into the workroom area to dilute solvent vapors. If adequate ventilation cannot be provided, workers may need to wear respiratory protection. A disposable particulate respirator would be sufficient for protection against the paint mist. If there are organic solvents in the coating, then a half-mask organic vapor cartridge respirator would be necessary.

Some paints also contain substances for which OSHA is proposing a skin designation. Three such substances include methyl alcohol, n-butyl alcohol, and mercury (aryl and inorganic mercury compounds). Natural rubber or neoprene gloves will provide adequate skin protection against these substances. CONSAD assessed the exposures with the use of an epoxy type paint from a site visit at the construction of an industrial facility in Indianapolis, Indiana in March, 1990 [2; Firm 12]. Two painters were sampled for petroleum distillates while roller-applying an epoxy ester-silica block filler to an interior concrete block wall. Three of the painters were also sampled for exposures to propylene glycol monomethyl ether and xylene while roller applying an epoxy finish coat to the same interior concrete block walls.

The 8-hour TWA results obtained from the sampling for petroleum distillates were 53 mg/m^3 and 26 mg/m^3. The proposed PEL for this substance is 1600 mg/m^3. The 8-hour TWA sampling results for exposure to propylene glycol monomethyl ether ranged from 0.42 ppm to 2.36 ppm. The proposed PEL for this substance is 100 ppm. The 8-hour TWA exposures to xylene ranged from 1.0 ppm to 7.3 ppm. The proposed PEL for xylene is 100 ppm.

Worker exposures to solvent vapors were evaluated by CONSAD in May, 1990 during the interior spray painting of structural steel and masonry associated with the new construction of an industrial facility in Indianapolis, Indiana [2; Firm 30]. Eight painters were involved in the activity and three were sampled for Propylene Glycol Monomethyl Ether (PGME) and xylene that were contained in the epoxy-polyamide product. Both compressed air and airless spray painting methods were employed.

The 8-hour TWA exposures to PGME ranged from 7 ppm to 8 ppm. The proposed PEL for PGME is 100 ppm. The breathing zone exposures to xylene resulted in 8-hour TWAs that ranged from 20 ppm to 30 ppm. The maximum average airborne exposure received by any of the painters during a sampling period was 41 ppm over a three hour period. The proposed PEL for Xylene is 100 ppm. It can be concluded that worker exposures to these substances were within the proposed standards.

All of the painters wore negative pressure half-mask respirators. The respirators contained pre-filters and organic vapor cartridges. Painters were also equipped with rubber gloves, protective clothing, and safety glasses. General ventilation was provided through the use of large fans which supplied fresh air to the rooms being painted. These fans were placed in the entrance and directed air toward the ceiling.

CONSAD obtained personal samples from five painters of two independent contractors during the painting of the inner surface of a large open air atrium in Milwaukee, Wisconsin in March, 1990 [2; Firms 13A and 13B]. Three of the workers applied a base and/or a finish coat. The other two painters were responsible for the final trim work and decorative painting in which only a finish enamel was used. Brush and rollers were the primary paint applicators, however, one of the painters used a paint sprayer but for less than one hour of application time.

Workers were monitored for airborne exposure to petroleum distillates. The TWA 8-hour equivalent exposures for the three workers involved in the base and/or finish coat application were all below 60 mg/m^3. The 8-hour TWAs of the remaining two workers solely

involved in the finish coat application were below 50 mg/m³. The proposed PEL for petroleum distillates is 1600 mg/m³. The painters used protective clothing (overalls, gloves) to avoid direct skin contact with the materials used.

Breathing zone samples were obtained by CONSAD from two painters while engaged in the coating of interior masonry walls of the shower and locker room areas of a building reconstruction project in South Park, Pennsylvania in April, 1990 [2; Firm 16]. The painters used a long-handled roller and a brush to apply an epoxy paint to the masonry block walls. The painting was conducted in relatively confined spaces with very little natural ventilation. Worker exposures to xylene, ethyl benzene, 2-butoxyethanol and petroleum distillates (naphtha) were evaluated.

The results of the laboratory analysis showed that the exposure levels for both painters exceeded the proposed PEL for xylene of 100 ppm on an 8-hour TWA basis. The xylene exposure for one of the painters was as high as 200 ppm during a two hour sampling period. The airborne concentrations and TWA exposures for the other substances sampled were 50 percent or less of the respective proposed PELs. Each of the painters wore a NIOSH approved half-mask organic vapor cartridge respirator which should have provided adequate inhalation protection.

CONSAD also conducted an evaluation of exposures to workers in contact with interior wood coating materials in a small town near Indianapolis, Indiana in May, 1990 [2; Firm 23]. The painting work under consideration was part of a renovation project of an institutional building. Two painters were observed: one applying a lacquer with an airless sprayer and the other applying the same material with a brush. Only the sprayer was sampled.

The breathing zone samples obtained were analyzed for 2-butoxyethanol, the primary solvent in the lacquer. The airborne concentrations surrounding the sprayer during a one hour sampling period ranged from 2.0 ppm to 7.8 ppm. The painting occurred during one-half of the shift and the 8-hour TWA concentration was estimated to be 2.0 ppm. The proposed PEL for butoxyethanol is 25 ppm and the sampled painter was clearly within the proposed limits. The painter wore a half-mask negative pressure respirator with pre-mist filter and organic vapor cartridge.

On another site visit in May, 1990, CONSAD evaluated airborne exposures associated with the interior painting of drywall during the construction of a multi-use commercial building in Chicago, Illinois [2; Firm 22]. Samples were obtained from two painters while applying latex flat wall paint by the conventional compressed air spray method. The work was conducted in a large basement area with little natural ventilation. Worker exposures to titanium dioxide, silica quartz and total dust were evaluated.

Both of the painters were sampled for titanium dioxide for a period of approximately 155 minutes. The analysis of the samples indicated breathing zone concentrations of 0.047 mg/m³ and 0.057 mg/m³. The proposed PEL for titanium dioxide is 10 mg/m³ on a TWA8 basis.

The analysis of the total dust samples determined that they contained 1.2 percent and 2.4 percent silica quartz. The determination of the airborne concentration of total dust indicated that the painters were exposed to dust levels of 12.0 mg/m³ and 23.4 mg/m³. The 8-hour TWA concentrations were calculated as 10.5 mg/m³ and 20.5 mg/m³. Exposures to both of these workers were considered to have exceeded the existing and proposed standards for total silica quartz dust and total dust.

One of the painters employed the use of a half-mask cartridge respirator and the other wore a disposable respirator that was not NIOSH approved for spray paint mists. The painters also wore work gloves and safety shoes. The use of appropriate respiratory protection can ensure that workers' exposures remain below the proposed PELs.

The application of a solvent or organic compounds to concrete, metal, or wood surfaces is a preliminary cleaning step for many other activities. The application of solvents or organic compounds will generally be some form of direct contact method to allow the solvent to clean or work on the surface. The solvent or organic compounds may be used for general cleaning and specific applications.

Control of organic vapors and gases which may be generated from this type of work can be achieved with fresh air supply blowers. Blowers should be located outside the immediate work room area to supply fresh air for dilution of contaminated air. If dilution becomes inadequate to control vapor concentrations at or above the PEL, then respiratory protection should be used. Supplied-air or self-contained breathing units are required for exposure to chlorinated solvents because these solvents have poor warning properties (odor threshold) and organic vapor cartridges have a short service life. Use of disposable half mask organic vapor cartridge respirators would be sufficient for exposures in excess of the PEL for other common organic solvents which may be encountered (VM&P Naphtha, xylene, toluene).

CONSAD conducted a site visit in January, 1990 to evaluate worker exposure to 1,1,1 trichloroethane during the cleaning of electrical transformer vault (concrete) pads in Arlington, Virginia [2; Firm 02]. Three workers were directly involved with the cleaning, handling and vacuum pick-up of the solvent during the shift. Two alternating washes of solvent and water were performed on each of the three vault pads worked on during the shift.

The activity consisted of pouring the solvent from a five gallon container onto the floor and broom brushing the surface. Two of the three workers were engaged in this task. This was followed by the vacuum pick-up of the solvent which was accomplished by the third worker. Water was then sprayed on to the floor surface from a hand pump sprayer and also broom brushed. This again was followed by vacuum pick-up. Two solvent and water washes were performed on each pad.

The total time that the workers were actually involved with the cleaning activity was 280 minutes. The 8-hour TWA exposures to 1,1,1 trichloroethane obtained from the personal sampling ranged from 63 to 80 ppm. These exposures include the total time of the work shift and zero exposures for the time that the workers were not involved in the cleaning. The average exposures for the time that the workers were involved in the cleaning ranged from 107 to 138 ppm. In any event, the exposures to workers were within the proposed PEL of 350 ppm.

The exposures associated with the use of a paint stripper were surveyed by CONSAD during the painting of a large open-air atrium in Milwaukee, Wisconsin in March, 1990 [2; Firms 13A and 13B]. Two of the painters were involved periodically with the process of paint stripping. These two painters were sampled for their exposure to petroleum distillates from the paint product and were sampled for exposure to methylene chloride (not included in this rulemaking). The 8-hour TWA exposures to methylene chloride were 4.75 ppm and 6.28 ppm. The maximum average level of airborne exposure found was 9.0 ppm during a five hour sampling period. It is believed that similar exposure results would have been obtained if other chlorinated solvents such as methyl chloroform, trichloroethane, or trichloroethylene had been used. If this assumption is correct the equivalent exposure levels would be below proposed PELs for these solvents.

Welding and metal fumes. Many activities in construction produce welding and metal fumes; OSHA has evaluated each type of activity to determine the nature of exposures and the feasibility of controls. The activities include welding or cutting of aluminum, carbon steel, galvanized steel, and stainless steel; burning, cutting or welding on materials with painted surfaces; soldering and brazing; and lead caulking.

Aluminum is typically used for architectural applications but also has industrial applications. The welding of aluminum is generally achieved using an electric arc and shielded with an inert gas such as helium. The cutting of aluminum stock can be accomplished with an oxy-acetylene torch.

During aluminum welding, the contaminants reaching the breathing zone of the welder may include ozone, nitrogen dioxide, aluminum metal fume plus other metallic oxides (zinc, magnesium, iron, copper). Of these contaminants, one of primary concern is ozone [7; p. 3.115]. Ozone is produced by ultraviolet light from the welding arc. Ventilation and work practices should be used to control exposure to welding fumes and gases. Good work practices involve using a properly fitted welding helmet and keeping the welding plume away from the breathing zone whenever possible. The most effective means of controlling worker exposure to airborne contaminants is through the use of portable exhaust ventilation. A properly designed exhaust system and hood will capture welding contaminants at the source before the fumes enter the worker's breathing zone. If exposures to metal fumes and ozone cannot be controlled through the use of ventilation, then respiratory protection may be needed. The minimum level of respiratory protection is a half-mask air purifying unit approved for metal fumes and ozone.

Carbon steel is a common construction material that has many applications. Structural members, masonry and stone supporting brackets, process tanks, and piping are its most common uses. The joining of carbon steel components is achieved using an electric arc with a shielding gas such as carbon dioxide or nitrogen. The application of an oxy-acetylene torch to carbon steel will achieve any cutting demands.

The major component of carbon steel is iron. Therefore, the primary airborne contaminant to which welders are exposed when welding on carbon steel is iron oxide fume. Gases are also produced in this welding process. Ozone, nitrogen dioxide, and carbon monoxide are the most common gases formed. These gases usually do not create an overexposure problem unless the work is conducted in a confined area. As with welding on aluminum, ventilation and work practices should be used to control exposures to welding fumes and gases. The most effective means of control is the use of portable exhaust ventilation. Some manufacturers also produce MIG welding units with an exhaust system directly built into the unit. In this unit, the exhaust hose is mounted close to the electrode. If engineering controls (portable exhaust ventilation or tip-exhausted welding gun units) do not provide sufficient protection, then respiratory protection should be used to supplement them. The minimum level of respiratory protection would be a half-mask air purifying unit for metal fumes and ozone.

In December 1981, Maryland Occupational Safety and Health (MOSH) conducted an investigation of construction workers that were exposed to lead [14]. In one case, employees were involved in the removal of blast furnace stoves from a steel mill. After the stoves were removed with the use of explosives, the workers used oxy-acetylene torches to cut each stove's outer steel jacket into pieces that could be transported from the site.

Air sampling of these workers showed that their exposure to airborne concentrations of lead ranged from approximately 200 $\mu g/m^3$ to 500 $\mu g/m^3$. In this process lead was a by-product in the blast furnace operation and only existed as a surface contaminant. When cutting, the workers used torches four feet long that placed them farther away from the point of fume generation.

CONSAD conducted site visits to evaluate worker exposure to welding fume and metals. The observations at one firm in Indianapolis, Indiana in March, 1990 consisted of the welding (fabricating) of limestone wall support brackets for an office building. Only one welder was observed and sampled while joining (stick welding) short lengths of carbon steel angles [2; Firm 06].

At another site in March, 1990, six crews each consisting of one welder and one grinder were involved in the welding of carbon steel piping for an industrial facility in Indianapolis, Indiana [2; Firm 11]. Shielded metal arc (stick welding) was employed by the workers observed. Two welders were sampled to assess their exposure to iron oxide fume.

The results of the samples obtained at the first site showed that exposure to iron oxide was 1.2 mg/m^3 TWA8 and well within the proposed PEL of 10 mg/m^3. The samples obtained at the second site indicated 8-hour TWA iron oxide exposures of 0.14 mg/m^3 and 0.28 mg/m^3. These results also were within the proposed PEL of 10 mg/m^3. Further, the workers' exposures were considered to be in compliance with the new PEL for welding fume of 5 mg/m^3 and with requirements for additive exposures.

Galvanized steel has applications in situations that demand corrosion protection. The welding and cutting of this type of material would be the same as that of carbon steel (i.e., arc welding, and oxy-acetylene torch).

The primary metal oxide of concern when cutting or welding on galvanized steel is zinc oxide fume. The same gases (ozone, nitrogen dioxide and carbon monoxide) discussed in previous welding activities are also produced when welding on galvanized steel. The primary method of controlling worker exposures to these airborne contaminants is local exhaust ventilation. This is most commonly achieved with portable exhaust units which capture contaminants at the source. Where adequate ventilation and good work practices are not sufficient to control employee exposures below the PEL, respiratory protection can be used to keep worker exposures below the proposed PELs.

Stainless steel is most often used in applications that demand high resistance and protection to corrosive and contaminating substances (e.g., food and drug processing equipment and piping, nuclear applications, etc.). The welding of the material is accomplished with an arc welder and an inert shielding gas such as argon or helium.

Chromium and nickel are important constituents of stainless steel. The welding and cutting processes typically associated with work on stainless steel do not appear to generate significant amounts of airborne contaminants. However, whether a visible plume is observed or not, significant concentrations of nickel and chromium metal fume may be produced. Therefore, local exhaust ventilation as discussed above should be utilized.

NIOSH conducted an industrial hygiene survey of airborne exposures generated during the welding, grinding and arc gouging of stainless steel at a nuclear power plant in Perry, Ohio [13]. Three personal breathing zone samples were obtained from three workers and analyzed for zinc, iron, lead, nickel, and chromium. The sampling durations were approximately five hours in length. In addition, a consultant of the construction contractor also conducted

a survey of the same processes which NIOSH reviewed. The consultant's survey consisted of obtaining eleven breathing zone samples and measuring for iron oxide, zinc, nickel and chromium. Six personal samples were collected for lead and twenty-two personal samples were also obtained specifically for the determination of the presence of chromium VI.

The reported concentrations of all of the personal samples were as follows: Zinc, 0.02 mg/m³–0.57 mg/m³; iron, 0.04 mg/m³–9.9 mg/m³; lead, not detected–0.017 mg/m³; nickel, 0.005 mg/m³–0.85 mg/m³; and chromium, 0.005 mg/m³–1.1 mg/m³. The results indicate that although a potential for overexposure exists, adequate ventilation and good work practices can be effective in keeping exposures below the proposed PELs. NIOSH cited vacuuming and housekeeping as important means of reducing dust levels. If necessary, respiratory protection can be used to keep worker exposures below the proposed PELs.

CONSAD conducted a site visit to assess the exposures to chromium and nickel metal fume during the heli-arc welding of stainless steel piping during the construction of an industrial facility in Indianapolis, Indiana in March, 1990 [2; Firm 11]. Three welders were sampled.

The 8-hour TWA exposure results obtained revealed that exposures to chromium ranged from 0.0015 to 0.0052 mg/m³ and that exposures to nickel ranged from 0.0011 to 0.0036 mg/m³. The average exposure for these workers was relatively low because the inspection requirements of the project reduced the actual arc time. The proposed PELs for chromium and nickel are the same at 1.0 mg/m³.

Burning, cutting, or welding on steel with painted surfaces can potentially produce many hazardous airborne substances. Exposures can be generated in the process of heating a coated steel by using an oxy-acetylene torch or an arc welder. The exposures that are produced consist of the base metal components (e.g., iron oxide, chromium, etc.), the volatized components of the coating (e.g., lead, zinc, chromium, etc.), and the resultant gases that are generated (e.g., ozone and carbon monoxide).

Workers can be adequately protected from lead exposures by engineering controls and respirators that are approved for that purpose. Lead is one of the primary health hazards associated with burning, welding, and cutting activities and is often present in relatively large quantities. The lack of adequate protection for workers is often due to a lack of awareness of the presence of lead or the erroneous assumption that the amount of lead fumes generated would not create a hazard.

Control of worker exposures to the metal fume and gaseous compounds created during the burning processes can be accomplished by equipment-mounted exhausts on the torches and by portable exhausts used as local ventilation at the point of operation. Screens or partitions between burning operations help reduce exposures to any workers in adjacent areas. Where engineering controls can not adequately reduce exposures below PELs, supplementary respiratory protective equipment can be used. This respiratory protection could range from half-mask respirators to air-supplied systems depending upon the nature of the contaminant and the level of exposure.

Maryland Occupational Safety and Health (MOSH) conducted an investigation after bridge workers were hospitalized for acute lead poisoning [14]. The workers were engaged in the resurfacing of a four-lane highway bridge that was located over a major river. In the course of the normal workday, torch cutting, arc welding, and grinding were performed. Lead overexposures were not expected since the work was performed outdoors and it was believed that normal air currents would disperse the contaminants.

Personal air sampling was conducted on employees that continued to work on the bridge. Airborne exposures of up to 4900 μg/m³ were observed. Precautionary measures to protect the workers from the potential of this health hazard were absent.

An article in Welding Journal illustrated several cases which demonstrated the potential hazards of lead in these types of activities [15]. In one case, workers were engaged in the cutting and welding of I-beams in a conversion project of an old power generating station. The work was being done indoors in a wide open area that was thought to be adequately ventilated.

The state of Maryland was notified that the workers involved had been hospitalized for lead poisoning. State health investigators arrived shortly after notification and conducted personal air sampling on workers who had replaced the hospitalized workers. Most of the samples reflected airborne concentrations of lead in excess of 1,000 μg/m³, 20 times the proposed permissible level (50 μg/m³), with one sample exceeding 1,400 μg/m³. There was no indication that any measures were taken to protect the workers from lead exposure.

The Occupational Health Service (OHS) of the New Jersey Health Department initiated industrial hygiene follow-up evaluations subsequent to elevated blood lead levels received from workers involved in construction activities. OHS conducted one such industrial hygiene evaluation at a bridge construction site [7; p. 3.42]. The project involved the rehabilitation of a span bridge, which was fabricated from steel coated with lead chromate paint.

A crew of five ironworkers was engaged in the process of removing and replacing the rivets in the bridge structure. The ironworkers would torch cut the old rivet heads and punch out the remaining part with a pneumatic hammer. The replacement nut and bolt was installed with an impact wrench. Within two weeks of work, all five workers became ill. Two of the workers were hospitalized and treated for lead poisoning. The OHS determined that the source of the lead exposure was the paint coating of the steel and not the rivets which were suspected initially. OHS conducted short-term breathing zone sampling of a burner and a hammer operator. The exposures to lead were 5.956 μg/m³ and 0.363 mg/m³ for the burner and hammer operator, respectively. The hammer operator's exposure to lead originated from dust generated from the hammering out of the rivets. Several recommendations were made as a result of the evaluation. These included: a lead awareness program for all workers with lead exposure, the use of powered air purifying respirators with HEPA filters, and medical surveillance.

In March, 1988 five of nine workers employed to demolish a bridge in western Massachusetts were diagnosed for lead poisoning [16]. These workers used oxy-acetylene torches to cut apart sections of the bridge. OSHA investigators determined that the bridge coating contained 30 percent lead by weight and that the available respirators were not always equipped with the proper cartridges to protect the workers from lead fume.

CONSAD conducted a site visit in Chicago, Illinois in March, 1990 to evaluate worker exposures to coating materials and fumes generated during the burning/cutting of painted steel [2; Firm 09]. The project consisted of the installation of a new HVAC and sprinkler system as part of the renovation of an office building. The primary interest in this project was the torch cutting enlargement of girder holes and the emissions generated from the paint coating on the girders. Following

the hole enlargement, support plates were stick-welded around the holes.

Two employees were involved in the process. One was responsible for the torch cutting and the other for the welding. Both workers were sampled for iron oxide and lead fume. The 8-hour TWA exposures to iron oxide fume were determined to be 0.16 mg/m³ and 0.30 mg/m³ for the torch cutter and the welder, respectively. The proposed PEL for iron oxide fume is 10 mg/m³. Both of the workers experienced overexposures to lead, based on a proposed PEL of 0.05 mg/m³. The TWA 8-hour determinations were 2.13 mg/m³ and 4.98 mg/m³ for the torch cutter and the welder, respectively. Personal protective equipment consisted of cotton gloves, long sleeved shirts, safety shoes, and welding helmets and shields. The torch cutter had a 12" portable fan situated behind him to blow fumes away from his breathing zone. There also were two air filtration/exhaust units located in the work area.

Soldering and brazing are techniques that are used to join metal pieces or parts. These techniques use heat in the form of a propane, MAPP gas or oxy-acetylene flame and a filler metal (tin/lead compositions, rosin core, and brazing rods) to join materials. Fluxes are used in soldering to remove metal oxides and surface contaminants from the metal surfaces before the filler metal is applied. Potential exposure sources are the heated metal (e.g., copper, iron oxide), the filler metal (e.g., lead, tin oxide and rosin core pyrolysis products) and the flux (e.g., zinc chloride, ammonium chloride, methyl alcohol and ethyl alcohol).

Brazing operations present potentially greater exposure hazards because of heat intensity. Most soldering operations occur at temperatures that are less than 800 °F. The melting point of the filler metals is relatively low (less than 600 °F) and the activity usually does not generate significant concentrations of metal fume. Brazing operations usually occur at temperatures in excess of 800 °F. The temperature of the operation is of major importance since it determines the vapor pressure of the metals that are heated and therefore the potential concentration of metal fumes to which the employee may be exposed. Because most field soldering and brazing work is conducted with a torch, it is difficult to regulate operating temperatures to within recommended limits to reduce the amount of metal fumes generated. In confined areas, portable exhaust ventilation is recommended to remove metal fumes and gases associated with this type of work. However, worker time weighted average exposures to metal fumes are usually not exceeded due to the limited durations of exposure encountered with soldering/brazing work.

Fluxes are also used to clean the metal surfaces to be bonded. The flux, which may be a solid, liquid, paste, or gas, is designed to remove surface contamination and keep the area clean until the solder metals can be applied. The flux is commonly applied by brushing or rolling directly onto the metals to be bonded. Most of the fluxing compounds do not present a significant airborne hazard but are corrosive to the skin. Therefore, skin contact should be minimized by specific work practices and good housekeeping. Volatile compounds such as alcohols (isopropyl, ethyl or methyl alcohol) are added to fluxes for viscosity purposes. Should a potential overexposure to methyl alcohol exist, an airline or supplied-air respirator is recommended as well as the use of special protective gloves. Methyl alcohol is one substance for which OSHA is proposing a skin designation. A natural rubber or neoprene glove would provide adequate skin protection.

CONSAD evaluated worker exposures to copper, silver, tin oxide and zinc chloride during the soldering of copper pipe joints in a water heater, kitchen sink and bathroom area of a single family house in Panama City, Florida in June, 1990 [2; Firm 26C]. A plumber completed 40 joints in approximately three hours. Tin-silver-copper solder wire (lead free) was used during this application. The soldering flux consisted of zinc chloride. The airborne concentrations of the contaminants obtained in the sample were all well below the proposed limits.

CONSAD conducted another site visit to evaluate exposures to plumbers during the renovation/remodeling of a school building in Bethel Park, Pennsylvania in July, 1990 [2; Firm 27]. One plumber was sampled for exposure to aluminum, copper, lead, silver, and tin while soldering copper pipe joints. When the plumber was not soldering, he was drilling holes for the piping installation. All of the exposures were below the proposed PELs for the individual substances. These exposure determinations were considered to be representative of the soldering activity for an average day.

The use of lead pots for lead caulking activities produces potential exposures to lead fumes. Lead caulking is used in some commercial construction building applications including the sealing of iron waste pipe joints. The use of lead for this purpose requires that it be liquified. The process of heating the lead and its application as a liquid presents possible exposure to lead oxide fume.

Since a lead pot remains stationary during heating, a portable exhaust system mounted near the pot can control lead fume. Appropriate thermostatic control devices can also be installed on the pot to prevent overheating, thus reducing the amount of metal fume. Also, the handling of lead requires that employees follow good personal hygiene and proper work practices to reduce the exposure potential. If respiratory protection is required, a half-mask respirator with NIOSH approved filters would be adequate [7; p. 3.136].

Concrete work. Workers are potentially exposed to a variety of substances when doing concrete, brick, and masonry work. Work activities include patching and resurfacing, sealing and coating, grinding/drilling/cutting, handling mortar or cement, handling and pouring concrete, preparing floors with a chemical stripper, installing concrete forms, and batch mixing of concrete, mortar, and cement.

Concrete patching and resurfacing work can range from the patching of small holes and cracks to a complete resurfacing. Various patching products are available depending on the application, e.g., epoxy resin patching compounds and plastic mortars.

Many patching and resurfacing products contain organic solvents. Epoxy compounds, used extensively in this activity, contain amine compounds (hardening agents) and organic ether compounds which can cause both skin sensitization/irritation and respiratory tract irritation. For this reason, adequate ventilation and good work practice controls need to be implemented to prevent worker overexposures. Blowers, located outside the immediate work room area to supply fresh air, dilute organic vapors and help to reduce exposure levels. There is no indication from the available data that exposures above the proposed PELs occur during this activity. If dilution ventilation should prove inadequate to control vapor concentrations, then respiratory protection would be necessary.

Sealing and coating operations involve the application of products that impart a durable finish to concrete or that seal it from water penetration. The products used for these purposes can be applied by brush, roller, and spray. Workers engaged in the use of these products may be potentially exposed to a range of organic solvents and active

reagents that these products may contain.

Sealing and coating operations that occur indoors may result in excessive organic vapor exposures when provisions are not made to supply adequate ventilation. Fresh air should be blown into the work room area to dilute solvent vapors. If adequate ventilation cannot be provided to the work area, respiratory protection may be necessary. For most of the solvents found in floor sealants, half-mask organic vapor cartridge respirators provide sufficient protection.

NIOSH conducted a health hazard evaluation to assess the health effects of isocyanates during floor waterproofing operations at a Philadelphia hospital in 1984 [17]. The floor waterproofing activities consisted of the following: (1) The caulking of the risers, baseboards, uprights, floors, and walls with a moisture-cured polyurethane sealant, (2) the filling of cracks in the flooring—the aliphatic urethane material used was troweled into place, (3) the application of the deck waterproofing material—one part liquid polyurethane was poured onto the floor, squeegied, and rolled out with a long-handled paint roller, and (4) application of the finish coat—two part liquid polyurethane materials were mixed for about six minutes and applied to the flooring with a long-handled paint roller and/or brush. The waterproofing contained one or more of the isocyanates: methylene bisphenyl (MDI), polymethylene polyphenyl (PAPI), and toluene diisocyanate (TDI).

Five personal samples were taken. No detectable concentrations of the monomeric isocyanates and ethylene glycol were found in any of the samples. NIOSH recommends the use of supplied-air full-face respirators for protection from overexposures to diisocyanates.

CONSAD observed the application of a waterproofing membrane to a concrete floor during the construction of a new building in Pittsburgh, Pennsylvania in February, 1990 [2; Firm 03]. Two employees were involved in the process of troweling the waterproofing membrane onto the floor. Xylene was a primary component and therefore exposures to the substance were evaluated. The analytical determinations for the exposures to xylene were 7.5 ppm and 3.8 ppm on an 8-hour TWA basis. The proposed PEL for xylene is 100 ppm as an 8-hour TWA. The two employees involved in the application process wore rubber gloves to prevent skin contact.

Workers that engage in grinding, drilling, chipping, or cutting on concrete, brick, or masonry can use a variety of tools to perform these tasks (e.g., sawing equipment, hammer drills, chippers, core drills, and jack hammers). Exposures result from the generation of particulate matter and dust. Workers can be exposed to some form of calcium silicate, silica (quartz), and other particulate respirable dusts.

The principal control techniques to reduce worker exposures to dust are ventilation and enclosure. However, airborne dust concentrations can be quite high during this activity; the use of disposable dust masks is very common. In most situations, this type of mask should provide adequate protection.

Worker exposure to silica quartz was evaluated by CONSAD during the construction of an industrial facility in Indianapolis, Indiana in March, 1990 [2; Firm 10]. The activity involved a worker engaged in the cutting of concrete blocks with a 14-inch diamond blade cut-off saw. The saw cutter was exposed to airborne dust containing 8 percent quartz at concentrations of 12.5 mg/m^3 and 57.1 mg/m^3 during the sampling periods. On an 8-hour TWA basis, this worker's exposure was estimated to be 37.8 mg/m^3. Thus, the worker was overexposed to total particulates and exposure to silica quartz respirable dust exceeded the proposed standard.

Personal protective equipment including safety glasses, goggles, cotton work gloves, and respirators should be provided to the employees doing this work. The proper use of this equipment would provide adequate protection from potential overexposures.

Worker exposure to total silica dust was evaluated during the preparation of a concrete floor prior to coating near Harrisburg, Pennsylvania in July, 1990 [2; Firm 29]. Two workers were sampled while using scarifying tools to clean the concrete floor surface in areas that could not be shotblasted. Samples were collected from two workers and from a shotblast operator. It was determined that the average quartz concentration of the dust samples was 30 percent. The two scarifying workers were exposed to airborne dust concentrations of 73.6 mg/m^3 and 20.1 mg/m^3 during the sampling periods. Their exposures were 62.3 mg/m^3 and 5.0 mg/m^3 respectively, on an 8-hour TWA basis reflecting minimal exposure while not engaged in the activity. On the basis of the percentage silica determined from the samples, the airborne concentrations exceeded the proposed standard for total silica quartz dust.

The workers wore personal protective equipment including disposable dust respirators and safety glasses. The disposable respirators were considered adequate for nuisance dust and silica. A small wall fan provided general ventilation to remove contaminants during the work period.

Mortar and cement are common products in the work of bricklayers and masons. Workers apply mortar when working with concrete block, brick, and stone. The primary components of mortar consist of portland cement and sand. The potential for overexposure to dust is present at mixing work stations. The potential for dust exposures in excess of corresponding PELs is unlikely after mixing with water. If curing compounds are added to the mix, workers may be exposed to organic solvents. However, the potential for overexposure to solvent vapors during this activity is remote.

Handling or pouring concrete and troweling and leveling concrete do not create significant airborne exposures. Concrete is normally delivered to the pouring site pre-mixed. As a result, workers are not exposed during the pouring, leveling, and finishing of concrete. Workers that are engaged in the application of curing compounds to the poured concrete may experience exposures to organic solvents contained in such products. The potential for overexposure to solvent vapors during this activity is remote.

Worker exposure to solvent vapors were evaluated by CONSAD during the spray application of a concrete curing compound in Chicago, Illinois in May, 1990 [2; Firm 19]. Air samples were collected from one laborer during the application of a concrete curing product with a hand-held sprayer. The primary solvent was mineral spirits. The exposure determination from the personal sampling resulted in an 8-hour TWA of 10.6 mg/m^3. Air concentrations of the mineral spirits for three time intervals of less than two hours each ranged from 31 mg/m^3 to 45 mg/m^3. The proposed PEL for petroleum distillates (mineral spirits) is 1,600 mg/m^3 TWA8. Personal protective equipment worn by the employee included a half-mask organic vapor cartridge respirator, rubber gloves, and eye protection.

At the construction of a hotel in Denver, Colorado in June, 1990, CONSAD was present during the pouring of concrete and the application of a curing material to a concrete surface [2; Firm 24A]. As the pouring progressed and the concrete started to set and harden, it was worked with power floats. A short time before the pouring was completed, one worker used a hand pump sprayer to coat the concrete with a curing agent. Air samples were collected and analyzed for n-hexane. Two workers were sampled. One worker was the curing

material applicator and the other was a floater working in the area of the applied material. The airborne concentrations of n-hexane obtained during sampling periods of one to three hours (representing the highest potential exposures) were less than 2 mg/m³ for each of the workers. The proposed 8-hour TWA PEL for n-hexane is 180 mg/m³.

Preparation work on concrete floors using chemical strippers is generally done to remove surface impurities or prior coatings. Application of the chemical strippers is done manually in a number of ways, e.g., direct pouring of stripper onto the surface followed by brushing or rolling. Concrete cleaners and strippers may contain mineral acids, organic acids or organic solvents.

Airborne contaminants generated from most activities involving preparation work on concrete floors can be controlled by implementing engineering controls. To prevent exposures to organic vapors and acid mists associated with this work, the use of fans to supply fresh air and to blow contaminants away from workers appears practical. Where exposures cannot be controlled within allowable limits by engineering controls, respiratory protection must be provided. For potential overexposures to the most common stripping agents (phosphoric acid, MEK, toluene, MIBK) the use of a disposable half-mask organic vapor cartridge/acid gas respirator is necessary.

The preparation and installation of concrete forms can cause exposures to several substances. Concrete forms are casings that hold and shape poured concrete. These forms are generally made of wood but can be metal as well. To prevent the concrete from adhering to the forms and to facilitate removal, the forms may be coated with oils. These oils generally consist of petroleum distillates, naphtha, xylene or some combination of organic solvents. Workers that engage in this activity are potentially exposed to these substances.

Since the majority of this work occurs outdoors and is of limited duration, exposures can be controlled by implementing good work practices (working upwind and not overspraying). If exposures still exceed the PEL, a half-mask organic vapor cartridge respirator should be used.

Batch mixing of concrete is normally accomplished by adding the ingredients (portland cement, sand and gravel) to water in a mixer. Significant airborne dust concentrations can be produced during this activity. The primary source of exposure occurs during the handling of bagged material. Most batch mixing operations require the operator to manually dump dry portland cement into the mixer after opening bags with a utility knife. Sand is usually stored in bulk form near the mixing area and is added to the batch by shovel. It is not uncommon to find the mixer operator covered with dust from the manual handling of raw materials. Mixer operators may be required to wear a single use or disposable dust mask to ensure that exposures are kept below the proposed PELs. Airborne dust concentrations can be reduced with isolation, by using a water spray on the bulk sand pile, and through the implementation of wet sweeping practices in the mixing area.

CONSAD observed the batch preparation of concrete and mortar during the construction of an industrial facility in Indianapolis, Indiana in March, 1990 [2; Firm 10]. Two workers were sampled for their exposures to silica and total dust while engaged in the mixing process. The prime activity of these workers was adding portland cement, sand, gravel and water to the two- and four-bag capacity mixers. The portland cement was contained in bags while the sand and gravel were piled on the ground near the mixers.

The sample determinations revealed that the workers were exposed to airborne concentrations of 12.4 mg/m³ and 13.2 mg/m³ of total dust containing less than one percent silica quartz on an 8-hour TWA basis. The primary component of the dust generated was portland cement and the proposed PEL for this substance is 10 mg/m³.

The employees were provided with personal protective equipment that included glasses/goggles, cotton work gloves, and respirators. This equipment provided adequate employee protection.

CONSAD observed the installation procedure of a terrazzo marble floor during the construction of a large multi-use commercial building in Chicago, Illinois in May, 1990 [2; Firm 21]. Primary tasks included the preparation of the flooring material and the grout. The components of the flooring material consisted of marble chips, white portland cement and sand. The components of the grout consisted of portland cement and color pigment. These materials and water were added to a two-bag mixer. CONSAD evaluated the exposures to silica quartz and to total dust for the individual responsible for the mixing operation.

During the sampling period of approximately four hours, the mixer operator was exposed to a total dust concentrations of 5.25 mg/m³. Exposures to total dust and silica were within the proposed limits. CONSAD observed that the bulk storage piles of marble chips and sand were periodically wetted to suppress the dispersion of dust in the work area.

The preparation of mortar is analogous to that of concrete. The preparer fills a mixer with portland cement and sand. The exposures occur from filling the mixer with cement from bags. There is also the potential of exposure to silica (quartz) during the sand addition. The control measures discussed above also apply to batch mixing of mortar and cement. These controls include enclosure of the mixing area, ventilation, use of a water spray on bulk sand piles, and implementation of wet sweeping work practices. Where these controls fail to reduce worker exposures to within acceptable limits, an air-purifying dust respirator would provide adequate protection.

Carpentry and Glazing. Activities during carpentry and glazing operations with potential exposures to airborne contaminants include the application of caulking compounds and sealants, bonding and gluing activities, cutting wood, applying wall and floor materials, and sanding.

Caulking involves the use of various caulks and sealants to fill cracks or spaces left by joints from general construction activities such as the installation of windows, or caused by the movement of material, such as asphalt pavement cracks. Generally, the caulking or sealant is applied manually with a spring-loaded caulking gun.

Workers using caulks and sealants are potentially exposed to solvents contained in these joint-filling materials but it is unlikely that overexposures to solvents will occur during normal use. Several commercially available products contain tin and mercury compounds. These substances may be present in small concentrations, and workers should protect skin areas where contact may occur.

While caulking compounds contain many solvents as ingredients, carriers, or drying agents, it is unlikely that workers would be overexposed to corresponding PELs during the normal application of these materials. The method of application is slow by nature and therefore a large volume of materials can not be used over a normal work day. In addition, the composition of the solvent portion of the caulking compound is such that it does not represent a significant source of exposure.

Bonding and gluing activities are common in the carpentry trade. There is a potential for exposure primarily to the solvent(s) contained in the adhesive.

Typical applications would include: counter top lamination, panelling installation or the joining of work pieces.

As with the application of sealants and caulking, the likelihood of worker exposure in excess of corresponding PELs for the organic solvent components of glues and adhesives is believed to be remote. The relatively small percentage of the solvents in the adhesives, the normal mechanical methods of application, and the small total volume of materials utilized in a given work day would normally maintain worker exposures below proposed limits. However, in situations where single applicators spend the majority of their day applying adhesives, especially in relatively confined areas, exposures in excess of PELs may occur. In these instances, fresh air supply blowers can be used to dilute the airborne concentrations to within acceptable limits. In circumstances where this can not be achieved, organic vapor respirators would be required. In addition, some of the materials which might be present in a given adhesive, such as cyclohexanone, would require special gloves such as neoprene, to reduce the skin absorption hazard.

The potential for exposure to soft and hard wood dusts exists during the cutting of wood. The exposure may become significant when workers using power saws are dedicated to this activity for an extended period of time. This is not believed to be a situation commonly found in construction work.

When equipment is dedicated to full time operation local exhaust ventilation should be supplied in order to control the operator's exposure. Work practices such as using dust suppressant compounds prior to dry sweeping will reduce exposures during clean-up. In rare instances where these practices do not substantially control exposures, respiratory protective equipment such as a disposable dust mask would be required.

CONSAD conducted personal air sampling to evaluate the wood dust exposures associated with the framing of a 1,200 square foot single family residence in Panama City, Florida in June, 1990 [2; Firm 26B]. Two carpenters sampled used a hand-held radial saw to cut pine boards and planking throughout the day.

Airborne concentrations of 0.93 mg/m^3 and 0.30 mg/m^3 were determined for each of the samples obtained. The equivalent 8-hour TWA concentrations were slightly lower at 0.67 mg/m^3 and 0.28 mg/m^3, respectively. These workers were exposed to less than 20 percent of the permissible exposure level allowed by the proposed standard.

Applying floor and wall coverings is a manual activity in which the worker is directly in contact with the floor/wall materials and any primers, adhesives and grouts used. Flooring materials may be ceramic, hardwood, linoleum, or carpeting. To help prevent worker exposures to organic solvent vapors, ventilation is the method of control believed to be most practical for this activity. Blowers can be located outside the immediate work room area to supply fresh air for dilution of contaminated air. If dilution becomes inadequate to control vapor concentrations within the PEL, then half-mask air-purifying (organic vapor) respirators are recommended.

Some adhesives contain organic solvents for which OSHA is proposing a skin designation. One substance commonly encountered with this designation is cyclohexanone. A butyl rubber glove can provide good protection when handling this substance.

In the laying of a ceramic floor, an adhesive is mixed and troweled on to the floor surface. The tiles are placed and the adhesive is allowed to set. This is followed by the filling of the tile joints with grout. Linoleum flooring is generally sold with a self-adhesive backing that is protected with a thin film of plastic or cellophane. The removal of the protective film exposes the adhesive and allows the direct application of the flooring.

However, the installation of ceramic tile still requires the manual application of an adhesive. The primary exposures to workers in these activities arise from the contact and use of flooring adhesives and any specialty grouts. The products used for these applications generally contain one or more organic solvents.

CONSAD conducted a site visit in May, 1990 to observe a commercial carpeting installation near Chicago, Illinois and to perform personal air sampling of the workers [2; Firm 17]. The installation involved the application of an adhesive to the flooring prior to laying the carpet. VM&P naphtha was the primary solvent component of the product. Two workers were sampled during the application. Neither of the employees was equipped with personal protective equipment and no engineering or administrative controls were observed.

The 8-hour TWA equivalent exposure determinations were the same for both employees at 3.3 ppm. The airborne concentrations were also approximately the same for the individual sample times. The maximum exposure level determined was 4.0 ppm. The proposed PEL for VM&P Naphtha is 300 ppm.

CONSAD also conducted air sampling during the installation of ceramic wall tile to evaluate the associated exposures in Pittsburgh, Pennsylvania in February, 1990 [2; Firm 03]. On this project, a tile setter troweled on an adhesive to a concrete block wall prior to the setting of the ceramic tile. Two finishers worked after the tile was set and applied the grout. All three of the workers were sampled for their exposure to VM&P Naphtha. The 8-hour TWA exposures for each were less than 1.0 ppm.

Worker exposure to carpeting adhesive solvents was evaluated by CONSAD during carpeting installation at a college dormitory in Pittsburgh, Pennsylvania in February, 1990 [2; Firm 04]. The primary solvent component of the adhesive was VM&P naphtha. One worker was sampled during an application period of approximately two hours. The airborne concentration of VM&P naphtha during the period was determined to be 3.7 ppm.

Sanding of wood surfaces may be accomplished either by hand or by the use of electrically driven reciprocal and belt sanding equipment. The potential exposure to wood dust exists with either method but may be more severe when power equipment is used and the activity duration is long.

When using electrically driven or pneumatic sanding equipment, worker exposures to dust can be controlled by exhausting air at the point of operation. Mechanical sanding equipment is commercially available with built-in exhausts in the design of the unit. This captures dust before it enters the worker's breathing zone. To clean the work area of material which has reached the floor, either dust suppressant compounds or wet sweeping practices should be implemented.

Hand sanding operations may be quite dusty but are usually of limited duration throughout a work day. Time weighted average exposures would most likely be below the proposed PELs for the corresponding dusts generated. If the potential for an overexposure exists, the use of a NIOSH approved disposable mask would provide adequate protection.

Sanding of drywall surfaces is generally done by hand. Workers that engage in drywall installation are likely to be exposed to the plaster material (gypsum-calcium sulfate) when sanding the dried joint compound.

A significant amount of dust can be generated during this activity; the degree of worker exposure depends on several factors. If the installation process is done by one worker then the time spent sanding may be intermittent and represent only part of the work shift; the resulting TWA exposure is likely to be within proposed limits. When several workers are sanding in close proximity to one another in an area without adequate ventilation and the sanding is done continuously for most of the work shift, then the potential for overexposure is increased.

Power sanding tools can be equipped with dust collection devices to reduce worker exposures. During power sanding and hand sanding, ventilation can reduce airborne concentrations through general dilution. Appropriate work practices can play a critical role in preventing excess exposures. The work area should be kept clean; the use of dust suppressants and vacuuming are effective means for keeping particulates out of the air. Exposures during clean-up operations may exceed those during actual sanding work if proper housekeeping measures are not taken. If engineering controls and work practices are not able control exposures adequately then respiratory protection would be required.

Worker exposure to hardwood dust was evaluated by CONSAD during the sanding of a maple gymnasium floor in Bethel Park, Pennsylvania in July, 1990 [2; Firm 27]. The floor sanding was a one-man operation and was conducted for a full 8-hour day. The sanding machine was equipped with a dust collector to capture dust which appeared to be effective. The sander used personal protective equipment that included a dust mask (NIOSH approved), ear plugs, and safety shoes. It was observed that the main source of exposure occurred while emptying the capture bags.

The sample collected from the worker's breathing zone (outside the worker's dust mask) was evaluated at an exposure level of 5.38 mg/m^3 of wood dust on an 8-hour TWA basis. The proposed PEL for wood dust (hard and soft wood) is 5 mg/m^3 TWA8. This worker was potentially overexposed to wood dust concentrations above those specified by the proposed standard, but the dust mask provided protection from the inhalation of the wood dust and was considered sufficient for compliance with the proposed standard.

Roadwork and Pipelines. Construction workers face many potential exposures during activities associated with roadwork and pipelines. These include asphalt laying, blasting and excavation, pipe wrapping, rock crushing, water well drilling, traffic line painting, and cable laying.

Asphalt laying involves the application of asphalt mixtures to traffic bearing surfaces such as roads and parking lots. The asphalt is normally dispensed from pavers at temperatures ranging from 200°F to 350°F. Some paver models can lay pavement that is more than 20 feet wide. The number of workers may vary from four to seven per paver. After the asphalt is placed, it is smoothed by a rolling machine. If the edges along curbs cannot be rolled, then they must be tamped manually. Associated with the laying of asphalt is the application of the tack coat. This asphalt emulsion must be sprayed on all the edges where an asphalt joint occurs. On those jobs where only a new layer of asphalt will be applied, an asphalt tank truck can be used to apply the tack coat. For joints and curbs, a worker can apply the tack coat from an oil pot equipped with a sprayer. Workers engaged in all of the functions associated with asphalt traffic surface construction are likely to incur exposure to asphalt fume.

Operators of paving equipment have the potential to be exposed to asphalt fumes. Enclosure in a plexiglass cab with a supplied air system that filters out asphalt fumes would control the operator's exposure. Workers immediately adjacent to the paving operation would have the greatest likelihood of exposure after the paving machine operator. If exposures are above the proposed PELs, these workers would be required to use a NIOSH approved air purifying respirator to control exposures.

CONSAD evaluated worker exposure to asphalt fumes during the paving of an airport runway near Pittsburgh, Pennsylvania in August, 1990 [2; Firm 33]. The paving operation crew consisted of nine people. An asphalt paver and vibrating roller were used to settle and roll the asphalt surface. The asphalt was delivered to the site by trucks and fed into the paver. Inside the paver, the asphalt is heated to about 300°F. The asphalt was dispensed from the paving machine onto the work area. The paver was operated by one worker, while two others made leveling adjustments to the machine. The remainder of the crew followed behind the paver to level, roll, and finish the paved material. The paving work was done in a large open area, and a variable breeze was present throughout the shift.

Four workers were evaluated for their exposure to asphalt fume (benzene soluble portions) during the sample period of six hours. These four workers consisted of the paver operator, the two paver adjusters, and a follow-up worker. The lab results of the samples obtained showed that all of the airborne concentrations of asphalt fume (benzene soluble) for the sampling period were less than 0.09 mg/m^3.

Workers engaged in blasting and the excavation of tunnels are assumed to have a potential for exposure to particulate matter (total dust) following the blast and also to the off-gases generated by the explosives. Excavation done by earth movers will also be a source of exposure to total dust for the workers in the area. Additionally, the exhaust from excavation equipment in tunnel areas may expose workers to elevated levels of carbon monoxide, carbon dioxide, and nitrogen dioxide.

Exposures to total dust above the proposed PEL can be controlled with disposable dust masks. While the off-gases generated by explosives and the particulate matter generated during excavation or blasting processes may result in worker exposures, it is likely that these exposures are within corresponding PEL's due to the distance that workers would be located away from the immediate point of operation. However, the use of earth moving equipment in confined areas could result in overexposures to carbon monoxide. Portable exhaust and fresh air supply systems should be used to provide fresh air into the work zone. In addition, constant monitoring for carbon monoxide should be conducted anytime gas or diesel powered heavy equipment is used in a confined area.

Workers engaged in the handling and preparation of charges for blasting may be exposed to the components of the explosive. However, it is believed that these exposures are not significant due to their short duration.

Workers engaged in earthmoving and trench excavation operate large-scale earthmoving equipment. The sources of exposure to these workers include particulate matter during the movement of dirt and natural gas containing hydrogen sulfide that may be present beneath and around trenches. Water spraying can be used to reduce exposures in some applications, but workers will generally need to rely on respiratory protection if potential exposures exceed the proposed PELs.

Wrapping pipes with asphalt wrapping material is done in underground piping installation. Asphalt is used as a pipe wrapping joint sealant and adhesive. Exposures to asphalt fume arise in both the heating process that liquifies the material and in the

application of the material to the pipe wrapping.

Because this work activity may occur over vast areas (several miles of pipeline), exhaust ventilation may not be feasible to prevent exposure to asphalt fume. Therefore, respirators may be relied upon to prevent inhalation of airborne contaminants when concentrations exceed the PEL. A disposable respirator with NIOSH approval for organic vapors would provide adequate protection in most work situations involving this activity.

Rock drilling and water well drilling activities present potential exposures to airborne substances. A NIOSH Health Hazard Evaluation of water well drillers [18] was conducted to assess exposures to PVC cement used in the installation of well casing and water well pipe. (There was no indication in the report that there were any potential exposure problems from the drilling activity itself.) All of the firms involved in the study used a cement which contained tetrahydrofuran, cyclohexanone, and PVC resin.

The well drillers' exposure to tetrahydrofuran and cyclohexanone was determined from four breathing zone samples. The tetrahydrofuran exposure from one 20 minute sample was at a level of 20 ppm. Given that there was no other exposure during the rest of the shift, this was considered as a full day's exposure of less than an 8-hour TWA of 1 ppm. Tetrahydrofuran was not detected in any of the remaining samples. Cyclohexanone was not detected in any of the samples.

Workers may be exposed to various dusts during drilling activities. The nature of the dust is dependent upon the geologic structure where the work is conducted. The airborne concentration of dust is affected by the moisture content of the soil, weather conditions, and work practices. Where potentially high airborne dust concentrations exist, worker exposures can be reduced through the use of a water spray. The water spray can act to suppress dust in worker breathing zones. Where the exposure cannot be adequately controlled through the use of a water spray, a single use or disposable particulate respirator could provide adequate protection to prevent inhalation of dust.

Exposures may be a concern during the spray painting of traffic lines for both large and small projects. For large projects, such as roadways, a tank truck is generally employed with the sprayer mounted at the rear. For smaller projects, such as parking lots, a hand held compressed air sprayer or a manual compressed air liner may be used.

On roadway projects, workers who follow the spray truck placing barriers to protect the freshly painted lines may be exposed to paint solvents, lead, or chromates. For manual operations, exposures may take place at any time during the application process.

The potential for overexposure to solvent vapors and metal pigments in the paint is unlikely. Most of this work occurs outdoors with natural ventilation. If an overexposure potential exists, it would be controllable through the use of an approved half-mask air-purifying respirator.

The installation or service of water pumps or pipelines and the handling and manipulation of water service piping does not present any apparent chemical exposures. However, there may be a potential for exposure to chlorine during the preparation and treatment of the water prior to service activation.

If an exposure to chlorine occurs, it would most likely be of short duration; OSHA is proposing a short-term exposure limit for such situations. To prevent overexposure to brief high concentrations of chlorine, a full-face respirator would be required. This is necessary because chlorine is a potent irritant of the eyes and respiratory tract. Implementation of engineering controls does not seem practical due to the unpredictability of the frequency of exposure, duration of exposure, or airborne concentration which may be encountered.

Cable plowing involves the formation of a trench for the laying of cable broadcast and telephone cable lines. The trench is created by mechanical means and the cable is unrolled into it from a spool. Workers associated with the process of digging the trench have the potential for exposure to particulates generated.

The likelihood of overexposure to dust generated during formation of cable trenches is considered remote. Engineering controls do not appear practical and therefore controls would be limited to respiratory protective equipment. For moderate exposures, disposable dust masks would be appropriate with potential up-grade to half-mask respirators should exposures become severely elevated.

Asphalt or concrete road milling is done during road reconstruction projects. During the process, mechanical equipment is used to remove several inches of road surface while maintaining the road base. The mechanical action of the road milling machine on the road surface generates and disperses particulate matter. The machine operator and nearby workers are exposed to the dust produced.

The machine operator, facing the highest exposure, can be enclosed in a ventilated cab and isolated from the dust. A continuous water spray and wetting operation can also be maintained. If the wet spray does not achieve adequate suppression, then the use of disposable dust respirators would provide additional protection for members of the work crew.

Rock crushing activities are typically set-up as stationary units in close proximity to the area that the rock removal is taking place. The rocky material is collected and unloaded into the crusher. The rock crushing operation is an inherently dust-generating operation that affects equipment operators and ground workers. Exposures to respirable silica quartz and other respirable dusts occur.

Water sprays that are applied at the point of dust generation provide an inexpensive means of reducing employee exposure to airborne dust. Engineering controls consisting of an air ventilation system can be used to protect equipment operators; plexiglass enclosures with supplied-air ventilation are recommended. In addition to wetting operations, appropriate disposable dust respirators are recommended to protect ground workers.

The practice of spraying oil on gravel road surfaces is conducted to minimize dust generation. The operators of these spray trucks have the potential for airborne exposure to oil mist (mineral oil) that is generated during the atomization process. The likelihood of an overexposure to oil mist is considered remote due to the fact that the area with the highest elevated concentration of oil mist is near the rear of the vehicle, and the operator is typically driving away from the point of application. Furthermore, the vehicles typically have enclosed cabs which effectively isolate the operators.

Spreading gravel for roadbeds and other purposes may also cause some exposures. Road building requires the use of a substantial amount of gravel. The gravel is used as a base for the road surface. As a consequence of the amount of material involved, the gravel is normally delivered to the work site in 20 ton dump trucks. In the process of unloading the gravel, workers that are in close proximity to the operation are potentially affected by the dust generated.

Keeping workers a reasonable distance away from the dumping operation is likely to control most worker exposures during this activity.

However, gravel with a high small-particle content could be sprayed with water prior to dumping and spreading to control dust generation. Since most of the delivery vehicles (dump trucks) are equipped with enclosed cabs, the drivers' exposures are likely to be within acceptable limits. Enclosure of the equipment operators spreading the gravel, in plexiglass-enclosed cabs with supplied-air ventilation systems which filter out particulate matter, could be used where water spray techniques fail to adequately control extremely high dust levels.

Miscellaneous Activities. Workers that are engaged in the fueling of construction service vehicles and equipment that operate on gasoline, diesel fuel or propane are likely to be exposed to these fuels. There may also be additional exposure to the combustion products associated with these fuels. It is believed, however, that these exposures will generally be insignificant.

The primary hazard posed by fuels is fire and explosion. Because of this potentially catastrophic danger, fuels should be handled with care. Proper storage, handling and transport of fuel is extremely important. If workers follow proper fuel handling practices, only minor exposures to fuel vapors and gases should occur. Also, this activity typically occurs outdoors where natural dilution ventilation occurs.

Joining pipes with joint compounds is a manual activity that occurs with the use of plastic pipe. The activity is normally carried out by applying the joint sealing compound with a fibrous applicator or brush to the joints to be mated. Worker exposure occurs from a number of organic solvents that may be contained in the sealant.

This activity usually occurs in situations in which it is difficult to implement engineering control measures. The activity is usually intermittent and involves crews of one or two employees who are mobile throughout the day. If a potential overexposure to a solvent vapor or metal fume would occur, the use of a respirator would be the required protection measure.

Some joint compounds contain organic solvents for which OSHA is proposing a skin designation. Two such substances include cyclohexanone and methyl alcohol. A natural rubber glove would provide adequate protection against skin contact from both substances while conducting this activity.

A field survey of exposure to organic solvents during the installation of plastic pipe was conducted by NIOSH in the San Francisco, California area in 1983 [19]. Six of the breathing zone samples taken were at least five hours in duration. These samples were tested for methyl ethyl ketone (MEK), tetrahydrofuran (THF), dimethylformamide (DMF) and cyclohexanone solvents of plastic pipe adhesives and cleaners. The work performed by the plumbers and pipefitters included installing plating line pipe, laying 3", 4" and 6" PVC pipe in a ditch, installing PVC pipe in a sewage treatment building, and setting bathroom, kitchen and bar sink traps in a residence. All exposures were below the corresponding proposed PELs.

NIOSH also conducted a health hazard evaluation of plumbers in the Boston area in 1981 to assess the health effects from working with PVC pipe cements and cleaners [20]. Personal samples were taken from one plumber at one housing development and from seven plumbers at another housing project. The samples were analyzed for MEK (methyl ethyl ketone), THF (tetrahydrofuran) and Cyclohexanone. The results reported showed TWA exposures below proposed limits. Combined equivalent exposure for one of the eight plumbers exceeded unity. NIOSH recommended that air concentrations of PVC cements and cleaners be reduced by increasing natural room ventilation in open spaces and by providing local exhaust ventilation in closed or semi-confined spaces. NIOSH also stated that wearing a well fitted, well maintained respirator should provide a sufficient factor of protection.

The joining of plastic electrical conduits is a manual activity similar to the joining of plastic pipes. The ends of the conduit to be joined are coated with an adhesive by brush or lid applicator and mated. The organic solvents contained in the adhesive create the exposure hazard. For the solvents encountered during this activity, organic vapor cartridge half-mask respirators would provide adequate protection. Two commonly used solvents in this activity also appear on the list of substances for which OSHA is proposing a skin designation. These substances are n-butyl alcohol and cyclohexanone. A natural rubber glove would provide adequate protection when handling compounds containing these substances.

The demolition of plaster walls is usually performed by applying a sledge hammer or similar tool to the wall with force. The break-up of the wall will generate particulate matter that will be dispersed into the immediate environment. Exposures encountered will consist of plaster of paris (calcium sulfate) and total dust (particulates not otherwise regulated).

Demolition work is inherently a very dusty operation. Because the structure is being removed, exhaust units may not be practical. In most situations, a portion of the work crew would be expected to wear respiratory protection. A single use or disposable dust mask would generally be adequate. To reduce the total number of workers requiring respiratory protection, partitions or screens could be set-up to isolate the demolition area. Also, a water spray and wet sweeping practices would help to suppress dust in worker breathing zones.

CONSAD surveyed the environmental exposures associated with a kitchen demolition project in Pittsburgh, Pennsylvania in June, 1990 [2; Firm 28]. Two workers were involved in a tear-down of the wall under observation. These men used a crowbar and a reciprocating saw to remove bricks, ceramic tile, plaster, tar paper and wood materials. The workers were monitored for their exposure to total dust. The TWA exposures to total dust during the sampling period of about five hours were determined to be 1.87 mg/m^3 and 3.89 mg/m^3. Exposures during the remainder of the shift were minimal. Personal protective equipment was employed that included cartridge respirators, safety glasses, steel-toed shoes and cotton gloves.

Torquing galvanized bolts is done by ironworkers during the erection of structural steel. The use of impact wrenches to torque galvanized bolts causes zinc oxide dust to be generated and dispersed into the worker's breathing zone. The likelihood of exposures above the proposed PEL is considered remote. This activity usually occurs outdoors, involving small mobile crews in situations where it is difficult to implement engineering control measures. If an overexposure problem to zinc oxide dust exists, a disposable particulate respirator with appropriate NIOSH approval would provide adequate protection.

Airborne exposures to carbon monoxide, carbon dioxide and nitrogen dioxide fumes generated by gasoline and diesel fuel powered machinery may become problematical when the equipment is operated indoors. Mobile cranes (cherry pickers), man lifts, and portable cutting tools are examples of the kinds of powered equipment that could be used for indoor construction work. Therefore, precautions should be taken to assure that fresh air is provided and that the entire area is ventilated.

To control the build-up of carbon monoxide, carbon dioxide and nitrogen dioxide generated by portable machinery operated indoors, blowers should be used to provide fresh air to the work area to dilute the combustion gas by-products. In most situations, worker exposures can be controlled with fresh air controls. Where space heaters are used in work areas, attempts should be made to vent the emissions directly outside. This will prevent the release of gaseous by-products in the work area. If this cannot be done, then provisions should be established to supply fresh air.

The heat fusion of plastic pipes and conduits is a technique used for joining purposes. In this method, a device similar to a curling iron is used to heat the plastic material to 180–200 degrees fahrenheit. At these temperatures, the pipe joint can be fused. This method is believed to only be used in polyethylene pipe installations and it is reported that no measurable quantities of gases or vapors are emitted during the process.

Repair and cleaning activities in sewer systems may involve the physical removal of material from occluded sewer lines and also the possible repair of damaged pipes. Material removal and/or repair may require that employees enter the sewer system. Employees engaged in this work may have the potential for exposure to ammonia, carbon dioxide, methane, and hydrogen sulfide.

Worker exposures to the chemicals used in cleaning sewer systems are often exacerbated by the fact that the work is being done in a confined space. Additional hazards of confined spaces include oxygen deficient atmospheres and/or atmospheres with hazardous levels of methane or hydrogen sulfide. Exposure monitoring should be conducted prior to and during all work activities involving a confined space. Fresh air should be supplied to the confined area at all times to reduce the health and explosion hazards, and workers should wear supplied-air respirators if exposures are potentially in excess of allowable limits.

Conclusion. OSHA has preliminarily concluded that compliance with the proposed PELs is feasible in the construction industry. Respirator use may be necessary in some high-exposure situations, but the overall need for respiratory protection in the industry should remain relatively low. Standard engineering controls and work practices designed for dust suppression and the dilution of fumes can be applied effectively to many activities. An important factor in providing workers with adequate health protection is the awareness of the presence of potentially hazardous substances.

Notes

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6. Costs of Compliance

The total annual cost of compliance with the proposed regulation of airborne contaminants in the construction industry (with a PEL of 5 mg/m^3 for asphalt fume) is estimated to be approximately $94 million for all firms affected by the rule. With alternative PELs for asphalt fume of 0.5 mg/m^3 or 0.2 mg/m^3, the annual cost would increase by an estimated $25 million or $51 million, respectively.

Methodology. The costs of compliance were estimated for individual activities in each SIC group. For each activity, the potential for hazard exposure was estimated based on data from a variety of sources. CONSAD Research Corporation conducted a survey of the construction industry for this purpose. Survey responses by construction establishments described the circumstances in which the activities took place, how the work was done, and the nature and extent of existing hazard abatement controls in place. Establishments identified hazardous substances used and the products where they were found. The chemical components of specific products were identified by referencing material safety data sheets (MSDSs). The survey responses also provided information on the numbers of employees involved in activities, the frequency and length of exposures during the activities, and the number of days the activities took place In addition, the survey supplied data on the use of specific engineering controls, work practices, and personal protective equipment by work activity, currently in

place across a representative sample survey of all job sites nationwide.

Survey data was supplemented with detailed descriptions of activities in NIOSH studies. Further information on construction activities was collected by CONSAD through a series of site visits. Exposure monitoring data were collected during site visits, from NIOSH studies, from investigations by state health departments, and from OSHA's IMIS database. Monitoring results from the IMIS database were linked to specific circumstances and activities by reviewing information from the inspection reports (form 91a) in OSHA area offices.

The activity-based approach used to determine exposures and the need for additional controls allowed for explicit consideration of mixed exposures. Exposures to multiple substances were evaluated according to the formula in the proposed regulation; combined exposures exceeding unity were considered to be exposures above the proposed PELs. As a result, the costing methodology included allowances for meeting the mixed exposure limit (MEL) as well as individual PELs.

Once the nature and extent of potential substance exposure was determined for a specific activity and SIC group, the costs of the controls necessary for compliance with the proposed PELs was assessed. The control hierarchy prescribed by OSHA requires that engineering and work practice controls be implemented when feasible. Engineering controls are widely available and effective for controlling exposures during many construction activities.

The most common type of engineering control involves increased ventilation. Depending on the application, this can be accomplished with a fan, a fresh air supply blower, or a portable exhaust hood. Unit costs for this equipment range from under $300 for a fan to over $1,500 for a portable exhaust ventilation unit.

Physical isolation of workers from the exposure source is another method of engineering control. Screens and partitions could cost an estimated $240 on affected projects; total enclosure of a worker in a plexiglass cab would have a cost of up to $4,000.

Equipment mounted dust catchers, vacuum cleaning, and water spraying devices are relatively inexpensive control methods that are applicable in many situations. The insulation of pipes (to keep asphalt kettles at lower temperatures) and the use of vapor control nozzles are additional examples of adapting engineering controls to construction activities. A description of the applicable controls for each activity is presented in the section on technological feasibility.

The annualized cost of engineering controls was derived by spreading the unit cost over the average useful life of the equipment and including an annual cost of capital of 10 percent. Annual and recurring costs associated with the use of controls were added to the annualized cost of controls to obtain the total annual cost to establishments of providing the controls.

Substances for which OSHA is adding a skin designation to the PEL should be controlled for dermal exposure. Different types of gloves are available to adequately prevent skin contact with different types of substances. Costs for gloves range from $1.80 per pair for cotton gloves to over $4.00 per pair for butyl rubber gloves. Barrier skin cream is also effective in some applications and is estimated to cost less than $10 per worker per project.

In the event that feasible engineering controls and work practices are insufficient for reducing exposures below the proposed levels, respiratory protection is necessary. Disposable dust masks are available for about $1.60 each while a powered air purifying respirator may cost as much as $570. Various respirators with intermediate levels of protection range in cost from $8.00 for a disposable metal fume mask to $165 for a full mask respirator. The replacement of cartridges for full-and half-mask respirators is estimated to cost $8.00.

The costs of compliance for specific activities in the affected SIC groups were aggregated to national estimates by using a computer model with standard statistical extrapolation techniques for representative sampling. The survey responses provided information on the value of work done, the value of the whole project, the duration of the project, the percent of time devoted to a particular activity, the total number of workers involved, and the total worker-hours of exposure. Within each survey cell, these numbers were compared to aggregate figures published by the Department of Commerce, such as total numbers of employees by size of establishment and SIC code, and total values for categories of projects and types of contruction work done. Sample data were then multiplied by appropriate weighting factors to project the sample up to nationwide "universe" estimates of the variables being measured. For a detailed explanation of this methodology, see CONSAD Report "Economic Analysis of Proposed Changes to Airborne Contaminant Standards for the Construction Industry" with Appendices, April 1, 1991 [1].

Breakdown of Costs. The total estimated annual costs of compliance, $93.9 million, are presented in three different ways. Table V–B10 presents the costs for construction establishments in each SIC group; Table V–B11 presents the costs of compliance for specific types of activities; and Table V–B12 presents the estimated costs by the type of control methods and protective equipment necessary for compliance.

TABLE V–B10.—ESTIMATED ANNUAL COSTS OF COMPLIANCE BY SIC CODE

SIC code	SIC description	Estimated annual costs ($ thousands)
1521/22/31	General Contractors—Residential Buildings	3,562
1541	General Contractors—Industrial Buildings	1,154
1542	General Contractors—Other Buildings	10,399
1611	Highway and Street Construction	5,104
1622	Bridges, Tunnels, and Elevated Highways	316
1623	Water, Sewer, Communication and Power Lines	1,594
1629	Heavy Construction, Not Elsewhere Classified	77
1711	Plumbing, Heating, and Air Conditioning	936
1721	Painting and Paper Hanging	21,166
1731	Electrical Work	399
1741	Masonry, Stone Setting, and Other Stone Work	5,776
1742	Plastering, Drywall, and Insulation Work	5,651
1743	Terrazzo, Tile, Marble, and Mosaic Work	2,409

TABLE V-B10.—ESTIMATED ANNUAL COSTS OF COMPLIANCE BY SIC CODE—Continued

SIC code	SIC description	Estimated annual costs ($ thousands)
1751	Carpentry Work	572
1752	Floor Laying and Other Floor Work	942
1761	Roofing, Siding, and Sheet Metal Work	2,224
1771	Concrete Work	3,746
1781	Water Well Drilling	124
1791	Structural Steel Erection	5,074
1793	Glass and Glazing Work	2,296
1794	Excavation Work	1,474
1795	Wrecking and Demolition Work	467
1796	Installation or Erection of Building Equipment	589
1799	Special Trades, Not Elsewhere Classified	17,615
Total		93,870

Source: Office of Regulatory Analysis, OSHA, Dept. of Labor; based on CONSAD Research [1].

TABLE V-B11.—ANNUAL COSTS OF COMPLIANCE BY ACTIVITY

[In thousands of dollars]

Activity	Costs of compliance
Abrasive Blasting-Sand	4,425
Application of Asphalt Roofing	852
Application of Fireproofing	3,662
Applying Caulking Compounds and Sealants	997
Applying/Installing Insulation	671
Asphalt Laying or Black Topping	158
Blasting/Excavation of Tunnels	8
Bonding (Gluing) Operations	2,826
Burning/Cutting/Welding Painted Metal	19,597
Cutting of Wood	569
Earthmoving Trench Excavation	3,333
Exterior Painting of Masonry	2,488
Exterior Painting of Wood	12,418
Exterior Painting of Steel	240
Patching and Resurfacing Concrete	556
Sealing and Coating (Concrete)	10,088
Grind/Drill/Chip on Painted Surfaces	3,712
Interior Application of Floor Coatings	1,760
Interior Painting of Metal Substrates	553
Interior Painting—Walls, Ceilings	4,156
Joining Pipes With Joint Compounds	1,001
Joining Plastic Electrical Conduits	400
Laying Floors	830
Prepare/Install Concrete Forms	74
Batch Mixing of Concrete	1,557
Batch Mixing of Mortar/Cement	8,272
Repair Work, Restoration of Brick	1
Soldering/Brazing	56
Surface Preparation—Sanding	2,580
Surface Cleaning (Stripping)	96
Welding/Cutting Steel	3,386
Abrasive Blasting—Black Beauty	217
Lath and Plaster Walls—Demolition	11
Application of Single Ply Roofing	85
Application of Waterproofing	1,362
Asphalt/Concrete Road Milling	742
Rock Crushing	75
Sealing/Joining Pipes With Mastics	2
Total	93,870

Source: Office of Regulatory Analysis, OSHA, U.S. Department of Labor; based on CONSAD Research [1].

TABLE V-B12.—ESTIMATED COSTS OF COMPLIANCE BY TYPE OF PROTECTION

[In thousands of dollars]

Type of protection	Cost
Engineering controls	29,534
Blowers to direct fumes or dusts away	2,999
Portable exhaust hoods and fans	6,135
Fresh air supply blowers	3,824
Total enclosure of the worker	75
Total enclosure to Confine the Emission	2,101
Equipment mounted dust catchers	12,376
Water spray (truck)	734
Equipment mounted water sprayers	594
Insulated Pipes	606
Work practices	3,808
Water spray (manual hosing)	2,500
Dry sweeping with dust suppressant	1,308
Personal protective equipment	6,403
Gloves (cotton)	3,415
Gloves (leather)	972
Gloves (of special material)	130
Welding helmet/mask	1,886
Respiratory Protection	64,000
Disposable Dust Masks	14,784
Disposable Metal fume or Chemical Mask	19,313
Half Mask canister and/or cartridge	4,314
Powered Air Purifying Respirator (PAPR)	404
Air Line (Full or Half Mask)	12,305
Sand blast hood with airline	2,880
Administrative controls	125
Exposure monitoring (personal constant)	125
Total	93,870

Source: Office of Regulatory Analysis, OSHA, Department of Labor; based on CONSAD Research [1].

Establishments in SIC 1721, Painting and Paper Hanging, would have estimated total annual compliance costs of $21.2 million. Over half of these costs are expected to be for exterior painting of wood and masonry finishes. These activities may involve potential exposures above the proposed PELs for petroleum distillates and silica. A variety of other chemicals would also be present, including ethylene glycol, propylene glycol, ethyl benzene, xylene, titanium dioxide, and others. Interior painting of walls, ceilings, and metal substrates would account for an estimated $4.2 million in compliance costs. The relatively low estimated compliance costs associated with these activities reflect the current widespread use of protective measures already provided for workers involved in these activities. Employees may encounter potential exposures to a long list of chemicals. In addition to those specified above, airborne contaminants may include ammonia, butoxyethanol, organic and inorganic mercury compounds, methyl alcohol, 2-butanone, lead, chromates, toluene, and others.

The estimated annual compliance costs for SIC 1799 are $17.6 million. This SIC group includes miscellaneous special trade contractors not elsewhere classified. Establishments in this group may be involved in antenna installation, caulking, preparing forms for poured concrete, ornamental metal work, waterproofing, epoxy application, grave excavation, lead burning, paint stripping, sandblasting, swimming pool construction, welding, fence construction, and other specialized services. Over half of the estimated compliance costs are attributable to activities that entail burning, cutting, or welding on painted surfaces resulting in potential exposures above the proposed PELs for lead, nickel, and zinc oxide fume. Additional protection for employees would also be necessary for grinding, drilling or cutting painted surfaces, application of waterproofing, batch mixing of concrete, earthmoving, abrasive blasting, and several other activities. Employees in SIC 1799 may be exposed to dozens of substances. Substances for which potential exposures above the proposed PELs are most prevalent include silica, portland cement, lead, welding fume, butoxyethanol, ethyl benzene, methyl

chloroform, ethylene glycol, petroleum distillates, titanium dioxide, and xylene.

SIC 1542 is comprised of general contractors for buildings other than residential or industrial buildings. Total estimated annual compliance costs for these establishments are $10.4 million. Employees of these general contractors are engaged in a wide variety of activities which may involve potential exposures above the proposed PELs. Protection of employees during sealing and coating operations accounts for an estimated $8.8 million of the total costs for this SIC group. The primary hazards identified during this activity are exposures to petroleum distillates, toluene, and xylene.

Establishments in SIC 1741, masonry and stone work, would have estimated annual compliance costs of $5.8 million. These costs are primarily attributable to providing additional protection to employees from exposure to portland cement and silica during batch mixing of mortar and cement.

Firms engaged in plastering, drywall, and insulation work (SIC 1742) would have estimated total annual compliance costs of $5.7 million. Activities for which additional protection would be necessary include application of fireproofing, insulation installation, burning, cutting and welding on painted surfaces, interior painting, and batch mixing of mortar and cement. Workers may experience potential overexposures to lead, mercury, and ethylene glycol.

Annual compliance costs for structural steel erection (SIC 1791) are estimated at $5.1 million. Activities of concern include welding, cutting, grinding and drilling, especially when conducted on painted surfaces. Substances for which overexposures may occur include lead and welding fume.

The estimated annual cost of compliance for SIC 1611, Highway and Street Construction, would be $5.1 million. Most of these costs are attributable to abrasive blasting activities. Employees would primarily be provided with additional protection from silica exposures as necessary.

The proposed PEL for asphalt fume of 5 mg/m³ is not expected to involve significant additional costs for establishments involved in highway and street construction. However, establishments in this SIC group would face significantly larger compliance costs under alternative PELs for asphalt fume of 0.5 mg/m³ or 0.2 mg/m³, and OSHA estimated the potential costs associated with the lower PELs for these establishments.

The primary activities of concern would be paving and black topping. About 240,000 employees are involved in this type of work, representing about 160,000 full-time equivalent (FTE) workers. This figure was adjusted downward by 40 percent to account for days or shifts of work without direct and prolonged exposure to hot asphalt. For example, workers involved in site preparation and breakdown, traffic control, truck driving, and other activities would not experience the levels of exposure typically attributed to paving work. Thus, about 144,000 employees or 96,000 FTE workers are considered to have exposure to asphalt fume during paving operations.

Exposure monitoring data for these activities was submitted to NIOSH in response to a request for information published in July, 1990. The National Asphalt Pavement Association (NAPA) found that full-shift time-weighted average (TWA8) exposures for paving workers ranged from 0.10 mg/m³ to 0.76 mg/m³ with a mean of 0.21 mg/m³ (benzene-soluble fraction) [2]. The Asphalt Institute cited data in their submission indicating that the mean TWA8 exposures for paving workers was 0.33 mg/m³, and also included summary data from dozens of studies published by NIOSH and other authors. For example, Virtamo et al (1984) reported to have found mean exposure levels of 1.2 mg/m³, with a range of 0.1 mg/m³ to 2.7 mg/m³, among one group of paving workers [3]. The Minnesota Department of Transportation reported that worker exposures during asphalt crack filling in 1990 averaged 0.89 mg/m³, with a range of 0.30 mg/m³ to 1.29 mg/m³ [4]. CONSAD reported that during a site visit to an asphalt runway paving operation worker exposures averaged less than 0.1 mg/m³ [1]. A report prepared for the Asphalt Institute by Radian Corporation found exposures during paving ranging from less than 0.1 mg/m³ to 5 mg/m³, with a geometric mean of 0.29 mg/m³ and an arithmetic mean of 0.57 mg/m³ [6]. All monitoring results represent the benzene-soluble fraction of asphalt fume.

Appropriate work practices and control of the temperature of the asphalt can help minimize employee exposures. Engineering controls should be used to reduce exposures before resorting to respiratory protection. Due to the nature of paving operations, the prospects for achieving significant reductions in exposures by these means appear to be limited. OSHA estimates that 50,000 FTE workers (about half of the exposed workforce) would be exposed above the proposed PEL of 0.2 mg/m³ and would be required to wear respiratory protection (such as a half-mask respirator with organic vapor cartridges). With a PEL of 0.5 mg/m³ this number would be about 10,000 FTE workers. Depending on the extent of rotation among jobs, these figures represent from 100,000 to 200,000 employees wearing respirators for some time each year.

For purposes of estimating the cost of complying with the proposed PEL of 0.2 mg/m³, it is assumed that 100,000 respirators would be purchased for $22 each. With an average life span of five years, the annualized cost would be about $580,000. In addition, the respirator cartridges would need to be replaced on average for 50,000 workers every four days at a cost of $8 per set. This would amount to an annual cost of $26 million (260 work days per year divided by 4, multiplied by $8 and by 50,000 workers). Providing protection from asphalt exposure with a PEL of 0.2 mg/m³ thus would account for about $26.6 million in compliance costs for establishments in SIC 1611.

With a PEL of 0.5 mg/m³, it is assumed that 50,000 respirators would be purchased, and that cartridges would be replaced on average for 10,000 workers every four days. Protection from asphalt exposure would thus account for $5.5 million in total compliance costs for SIC 1611.

Total annual compliance costs for establishments in SIC 1761, Roofing, Siding, and Sheet Metal Work, are estimated to be $2.2 million with a PEL for asphalt fume of 5 mg/m³. Most of these costs would be incurred for activities involving grinding, drilling, or cutting painted surfaces and for the application of roofing systems. Employees may potentially be exposed to lead, fibrous glass, and asphalt fumes.

Estimated compliance costs for establishments in SIC 1761 would be significantly higher under alternative PELs for asphalt fume. OSHA estimated the compliance costs that would be associated with PELs for asphalt fume of 0.5 mg/m³ and 0.2 mg/m³.

The Asphalt Institute cited data in their submission to NIOSH indicating that the mean TWA8 exposures for asphalt roofing workers was 0.80 mg/m³, and also included summary data from dozens of studies published by NIOSH and other authors. For example, Brandt et al (1985) is reported to have found mean exposure levels of 3.30 mg/m³, with a range of 1.40 mg/m³ to 7.80 mg/m³, among one group of roofers [3]. Exposure monitoring data from four NIOSH studies show mean exposure levels (and ranges of exposures) among roofers of 0.54 mg/m³ (0.11 mg/m³–1.89 mg/m³), 1.07 mg/m³ (0.90 mg/m³–1.20 mg/m³), 0.83 mg/m³ (0.35 mg/m³–2.10

mg/m³), and 0.71 mg/m³ (0.30 mg/m³–1.30 mg/m³) [3]. Radian Corporation found exposures during asphalt roofing ranging from less than 0.1 mg/m³ to 2.5 mg/m³, with a geometric mean of 0.28 mg/m³ and an arithmetic mean of 0.52 mg/m³ [6].

The Bureau of Labor Statistics reports that there are over 210,000 roofers in the United States [5]; these employees represent about 140,000 full-time equivalent workers. According to the 1990/1991 market survey conducted by the National Roofing Contractors Association, asphalt roofing comprises about 35 percent of roofing work, which would represent about 49,000 FTE workers. Some employees in these operations may not need respiratory protection; employers may have to make a determination of need before each shift.

For purposes of estimating the costs associated with this proposed rulemaking, the number of workers engaged in asphalt roofing was adjusted downward by 30 percent to account for days and shifts that do not involve significant exposure to asphalt fume. For example, roofing jobs typically include time for removing old roofs, transporting and setting up equipment, and other activities without direct exposure to asphalt fume. Thus, about 34,000 FTE workers are estimated to be working with hot asphalt in roofing jobs.

Asphalt exposures during roofing can be minimized through the use of appropriate work practices and by carefully controlling the temperature of the asphalt. The asphalt in the kettle can be kept at lower temperatures if the pipes transporting the asphalt to the point of use are insulated. Improvements in these areas and the use of other engineering controls should be made prior to reliance on respiratory protection and may help keep some exposures below the proposed PEL. However, the available monitoring data indicate that exposures for most roofers working with asphalt are likely to be above 0.2 mg/m³ even after engineering control and work practice modifications are made. OSHA estimates that 30,000 FTE workers, representing 50,000 to 80,000 employees, would be required to wear respirators with a PEL of 0.2 mg/m³.

With a PEL of 0.5 mg/m³ about 25,000 FTE workers would be exposed above the PEL during asphalt roofing, and about 20,000 FTE workers would be required to wear respirators.

Based on the results of the construction survey, approximately 1,000 establishments would need to insulate their asphalt-carrying pipes. The unit cost would be about $150 with a life span of one year. The total cost to the industry of installing additional insulation on pipes would be $150,000.

A half-mask respirator with organic vapor cartridges would provide adequate protection from asphalt fumes. The cost of each respirator would be about $22 and a set of replacement cartridges would cost about $8 [1]. Cartridges would need to be replaced every 4 days on average. With a life expectancy of five years, respirators purchased for 60,000 employees would have an annualized cost of about $350,000. The replacement of cartridges every four days for an average of 30,000 employees would have an annual cost of about $15.6 million. Providing protection from asphalt exposure during asphalt roofing with a PEL of 0.2 mg/m³ would thus account for an estimated $16.1 million in compliance costs for establishments in this SIC code.

With a PEL of 0.5 mg/m³, costs for insulating pipes and purchasing respirators would be about the same, and replacement of cartridges would have an annual cost of $10.4 million. Protection from asphalt exposure during asphalt roofing with a PEL of 0.5 mg/m³ would thus account for an estimated $10.9 million in compliance costs for establishments in SIC 1761.

Other SIC groups each have estimated compliance costs of less than $4 million each. These costs are spread over various activities for which the potential need for employee protection is already recognized to some degree. The estimated compliance costs for many activities may include costs that would have to be incurred to meet existing PELs.

Based on the survey results, activities with the highest total compliance costs across all SIC groups would be burning, cutting, or welding on painted surfaces ($19.6 million), exterior painting ($15.1 million), concrete sealing and coating ($10.1 million), and batch mixing of mortar and cement ($8.3 million). Other activities with significant compliance costs include abrasive blasting, trench excavation, grinding and drilling on painted surfaces, interior painting, sanding, and welding and cutting steel.

Almost $30 million of the total compliance costs would be used to provide engineering controls such as equipment-mounted dust catchers, portable exhaust hoods, fresh air supply blowers, enclosures, and water sprayers. The largest portion of costs, when examined by the type of control used, is for respiratory protection at an estimated annual cost of $54 million. Work practices (e.g. the use of dust suppressants), personal protective equipment such as gloves and welding masks, and administrative controls that include personal constant exposure monitoring, are additional measures that reduce potential risks to employees.

References

1. CONSAD Research Corporation. Economic Analysis of Proposed Changes to Airborne Contaminant Standards for the Construction Industry. Final Report and Appendices. Prepared for the Office of Regulatory Analysis, OSHA, U.S. Department of Labor. April 1, 1991.
2. Comments to the National Institute for Occupational Safety and Health, Submitted by National Asphalt Pavement Association. November 9, 1990.
3. Comments submitted to the National Institute for Occupational Safety and Health by the Asphalt Institute. December 21, 1990.
4. Comments submitted to the National Institute for Occupational Safety and Health by the Minnesota Department of Transportation. Industrial Hygiene Sampling Reports 1989–1990.
5. Bureau of Labor Statistics, Employment and Earnings, January 1991.
6. Radian Corporation, Asphalt Industry Cross Sectional Exposure Assessment Study, Final Report, July 12, 1991, bound with supplemental technical memorandum to Bernie McCarthy, Asphalt Institute, from Jeff Hicks, Radian Corporation, of July 31, 1991.

7. Economic Impacts

This section examines the economic impacts associated with OSHA's proposed revisions to the PELs in the construction industry. The overall impact of compliance with the proposed rule is not expected to be a substantial burden for the industry and is considered economically feasible for each sector.

The economic impacts were evaluated by comparing the cost estimates presented in the previous chapter with economic and financial statistics available for the construction industry. These data were combined with an economic analysis of the structure of the industry and the affected markets to determine the likely effects. The economic impacts were assessed for each 4-digit SIC group by type of construction project.

Table V–B13 shows the estimated costs of compliance as a percent of the net value of construction for each SIC group. For the industry as a whole, total costs of compliance are estimated to be less than one thirtieth of one percent of the total net value of construction. For each of the SIC groups, compliance costs would represent less than 0.3 percent of the respective value of construction.

Table V–B14 presents data for each of the SIC groups on the number of

employees, the number of establishments, the total annual payroll, the average annual payroll per establishment, the average cost of compliance per firm, and the average cost of compliance per employee. The average annual payroll per establishment in the construction industry is about $150,000. The average estimated cost of compliance per firm would be approximately $175, and the average cost of compliance per employee would be less than $24.

TABLE V-B13.—COMPLIANCE COSTS AS A PERCENT OF NET VALUE OF CONSTRUCTION

SIC code	SIC description	Net value of construction (millions)	Estimated costs of compliance (thousands)	Compliance costs as a percent of net value of construction
1521/22/31	General Contractors, Residential Buildings	$60,414	53,562	0.0059
1541	General Contractors, Industrial Buildings	11,095	1,154	0.0104
1542	General Contractors, Other Buildings	39,510	10,399	0.0263
1611	Highway and Street Construction	27,984	5,104	0.0182
1622	Bridges, Tunnels, and Elevated Highways	4,187	316	0.0075
1623	Water, Sewer, Communication and Power Lines	15,055	1,594	0.0106
1629	Heavy Construction, NEC	21,209	77	0.0004
1711	Plumbing, Heating, and Air Conditioning	44,518	936	0.0021
1721	Painting and Paper Hanging	7,446	21,166	0.2843
1731	Electrical Work	34,658	399	0.0012
1741	Masonry and Stone Work	8,269	5,776	0.0699
1742	Plastering, Drywall, and Insulation Work	15,137	5,651	0.0373
1743	Terrazzo, Tile, Marble, and Mosaic Work	2,182	2,409	0.1104
1751	Carpentry Work	10,039	572	0.0057
1752	Floor Laying and Other Floor Work	3,371	942	0.0279
1761	Roofing, Siding, and Sheet Metal Work	14,183	2,224	0.0157
1771	Concrete Work	13,854	3,746	0.0270
1781	Water Well Drilling	1,299	328	0.0253
1791	Structural Steel Erection	4,510	5,074	0.1125
1793	Glass and Glazing Work	3,142	2,298	0.0731
1794	Excavation Work	7,491	1,474	0.0197
1795	Wrecking and Demolition Work	845	467	0.0553
1796	Installation of Building Equipment	5,010	589	0.0118
1799	Special Trades, NEC	9,833	17,615	0.1791
Total		365,241	93,870	0.0257

Source: Office of Regulatory Analysis, OSHA, Dept. of Labor.

TABLE V-B14.—COSTS OF COMPLIANCE PER FIRM AND PER EMPLOYEE

SIC code	Number of establishments	Number of employees	Total annual payroll (millions)	Average annual payroll per establishment	Estimated average cost of compliance per firm	Estimated average cost of compliance per employee
1521/22/31	119,287	448,052	$6,952	58,276	530	$7.95
1541	7,014	110,785	2,497	355,993	165	10.42
1542	31,337	366,871	7,756	247,501	332	28.35
1611	10,986	239,111	5,397	491,276	465	21.35
1622	1,159	40,092	910	785,090	273	7.88
1623	9,919	165,879	3,428	345,571	161	9.61
1629	14,532	238,204	5,726	39,349	5	0.32
1711	69,566	470,793	10,311	148,214	13	1.99
1721	29,867	145,385	2,386	79,893	709	145.59
1731	49,436	405,961	9,622	194,635	8	0.98
1741	23,284	150,308	2,462	105,722	248	38.43
1742	17,809	217,392	4,348	244,142	317	25.99
1743	5,089	27,908	554	108,892	473	86.32
1751	36,009	164,191	2,610	72,480	16	3.48
1752	8,174	34,666	645	78,968	115	27.17
1761	25,673	186,916	3,111	121,178	97	11.90
1771	23,422	186,840	3,278	139,956	160	20.05
1781	3,414	13,628	249	72,791	96	24.07
1791	4,017	54,729	1,232	306,608	1,263	92.71
1793	4,636	28,730	607	130,855	495	76.92
1794	13,422	79,198	1,598	119,022	110	18.81
1795	1,240	11,686	195	157,609	377	39.96
1796	3,777	50,244	1,447	383,236	156	11.72
1799	23,198	141,615	2,269	97,799	759	124.30
Total	536,267	3,979,184	79,590	148,412	175	23.59

Source: Office of Regulatory Analysis, OSHA, Dept. of Labor, based on Department of Commerce 1987 Census of Construction.

The estimated costs of compliance are compared to the value of construction by the type of construction project in Table V–B15. For each type of construction project the compliance cost would represent less than one tenth of one percent of the total value of such construction. Compliance costs for both building and nonbuilding construction on average would represent less than 0.03 percent of the total value of such construction.

The SIC group with the highest ratio of estimated compliance costs to net value of construction is SIC 1721, Painting and Paper Hanging. The need for and use of respirators and other methods of exposure control is already relatively widespread in this industry sector. Establishments in this SIC group are regularly engaged in activities involving airborne and dermal exposures to the substances in this proposed rulemaking. Activities include interior and exterior painting (including bridge painting), abrasive blasting and other methods of surface preparation, and occasional soldering, brazing, grinding, and cutting. These activities expose workers to several hazardous substances, including ethylene glycol, toluene, xylene, petroleum distillates, mercury, and silica.

TABLE V–B15.—COMPLIANCE COSTS BY TYPE OF CONSTRUCTION

Type of construction	Value of construction (thousands)	Cost of compliance (thousands)	Cost as a percent of value
Building Construction	$368,029,969	$ 83,620	0.023
Single family houses	122,202,213	21,962	0.018
Apartment buildings	21,057,191	445	0.003
Other residential	14,302,766	1,383	0.010
Office buildings	60,570,245	12,975	0.021
Other commercial buildings	46,266,032	22,251	0.048
Industrial buildings	52,477,389	12,559	0.024
Religious buildings	4,535,939	751	0.017
Educational buildings	16,282,387	8,925	0.055
Hospital buildings	19,305,021	901	0.005
Farm buildings	1,560,486	36	0.002
Amusement, social buildings	5,046,890	2	0.000
Other buildings	3,946,021	944	0.024
Land development	2,475,609	486	0.020
Nonbuilding Construction	105,411,564	10,250	0.010
Highways, streets	33,692,504	525	0.002
Parking areas, fencing	4,665,769	73	0.002
Recreational facilities	3,387,728	1,745	0.052
Bridges, tunnels	7,261,700	5,069	0.070
Dam and reservoir	990,275	58	0.006
Marine construction	1,595,977	24	0.002
Harbor & port facilities	619,269	0	0.000
Conservation/development	1,520,673	13	0.001
Power & communication lines	6,232,816	775	0.012
Sewers, water mains, etc.	13,506,798	880	0.007
Pipe line construction	3,259,867	0	0.000
Mass transit	1,416,178	0	0.000
Blast furnaces	6,776,004	0	0.000
Power plants	6,915,723	696	0.010
Sewage treatment plants	6,057,721	291	0.005
Oilfields	506,020	0	0.000
Earthmoving, nonbuilding	2,677,903	12	0.000
Other nonbuilding nec	4,327,839	79	0.002
Total	494,346,548	93,870	0.019

Source: Office of Regulatory Analysis, OSHA; based on U.S. Department of Commerce, *1987 Census of Construction*, and CONSAD survey [1].

The compliance costs in this sector are primarily attributable to a minority of firms that do not implement the sound industrial hygiene practices followed by most firms. Many circumstances involving exposures above the proposed PEL also involve exposures above the existing PELs. The proposed rule codifies current consensus standards which have already been adopted by responsible employers.

The estimated average cost of compliance per establishment in SIC 1721 would be less than 1 percent of the average annual payroll per establishment or about $150 per employee annually. Given the extent of protection currently provided in this industry, the incremental costs necessary to achieve compliance with the proposed rule are relatively small. The rule would not require widespread changes in accepted practices; many firms with appropriate industrial hygiene practices would remain unaffected.

Estimated compliance costs for SIC 1799, Special Trade Contractors, Not Elsewhere Classified, are less than 0.18 percent of the value of construction for this sector. The group includes establishments engaged in paint removal, waterproofing, and miscellaneous construction work, resulting in worker exposures to lead and other chemicals. Many of these workers are not adequately protected from exposures to hazardous substances. A lack of awareness of the presence and nature of potential health hazards may contribute to the problem.

The cost of providing protection would not be a significant burden for employers; the requirements associated with the proposed PELs are equivalent to those for the existing PELs in many activities. Responsible employers already provide the necessary protection for their employees. The average cost of compliance per firm represents less than 0.8 percent of the average payroll per firm.

With a PEL of 5 ug/m^3 for asphalt fume, activities with potential asphalt

exposure would largely be unaffected, and the impact of the proposed rule on paving and roofing establishments would be negligible. With a PEL of 0.2 mg/m³ for asphalt fume, employees engaged in asphalt roofing and paving work would probably be required to wear respirators routinely, and the resulting costs of compliance for establishments in SIC 1761 (roofing) and SIC 1611 (paving) would be significantly higher. As outlined below, the impacts of a standard with a PEL of 0.2 mg/m³ for asphalt fume would be nonnegligible but economically feasible for each of these industries.

With a PEL of 0.2 mg/m³ for asphalt fume, the estimated compliance costs for firms in SIC 1761, Roofing, Siding, and Sheet Metal Work, would represent about 0.15 percent of revenues. Increases in the cost of construction for these establishments primarily would result from the need to protect employees from exposure to asphalt fume. Most employees working with hot asphalt would be required to wear a respirator.

The additional costs of providing employees with respiratory protection during asphalt roofing work would result in higher costs for completing this type of work. In addition to the costs of the respirators, some loss of productivity could be expected as employees wearing respirators are less able to perform strenuous work. The costs of supplying employees with respirators during a typical asphalt roofing job would represent about 1 percent of the revenues. The average productivity loss associated with wearing respirators may increase labor costs by 10 percent which would represent about 2 percent of revenues. The total estimated compliance costs to employers would thus be about 3 percent of revenues for asphalt roofing.

Routine use of respirators may potentially involve effects on productivity, safety, and health. In response to OSHA's recent Methods of Compliance proposal, several commenters expressed concern about such effects and the associated burdens for workers. Many statements generally supported the conclusion that "men often cannot sustain respiratory wear for the full shift." [Testimony of Morton Corn, May 30, 1990. See also statements by the United Steelworkers of America, July 10, 1990, by the AFL–CIO, October 3, 1989, and other comments in docket number H–160].

The impact of the increased costs would produce a combination of effects. Employers could attempt to raise prices by about 3 percent to offset the compliance costs. The ability of employers to raise prices sufficiently to cover all compliance costs would be limited, but a substantial part of the costs could be recouped. Employers engaged in asphalt roofing would probably experience a partial reduction in profits in conjunction with limited increases in prices to avoid losing business. Without any increase in prices, the estimated compliance costs would absorb about 60 percent of the average profits. Some marginal employers may decide to discontinue asphalt roofing operations.

The total demand for asphalt roofing work would not be expected to change drastically as a result of a proposed standard for a PEL of 0.2 mg/m³ for asphalt fume. Adequate substitutes may not be available, and the increase in costs to building owners would not be unbearable. All employers in the country would be subject to the same requirements, and work in foreign countries cannot be substituted for projects in the United States. The inherent inelasticity of demand for asphalt roofing would ensure continued demand for such work. Total revenues and hours worked should not decline; establishments unable to continue asphalt roofing profitably would be replaced by more efficient firms. The proposed standard would be economically feasible for the industry as a whole.

With a PEL of 0.2 mg/m³ for asphalt fume, establishments in SIC 1611, Highway and Street Construction, would have the highest estimated cost of compliance per firm. This would partly be due to the relatively large number of employees per firm (22), and would partly be due to the extensive nature of the impacts in this sector. Over 80 percent of the estimated compliance costs would be associated with providing respiratory protection for employees working with hot asphalt. Apart from other potential effects of wearing respirators, the costs of providing protection as required by the proposed rule should be economically feasible for employers. The total costs would represent about one tenth of one percent of the revenues for this sector; the costs per establishment would be about 0.06 percent of the average payroll per establishment.

Other sectors and activities in the construction industry would be less affected by the proposed rule and the potential alternatives for the PEL for asphalt fume. The proposed changes in the PELs generally would not require drastic or widespread alterations in the nature of the work. The protection required by the standard would basically involve circumstances in which potential health hazards are already recognized by many employers. To a significant extent the estimated costs of compliance reflect a lack of awareness by some employers or an insufficient application of all feasible controls in some activities. The incremental compliance cost for the proposed rule represents the increase in protection necessary to achieve more comprehensive protection for all employees. The feasibility of the proposed standard is evident since many employers conduct the relevant activities in compliance with the proposed requirements.

This proposed regulation would apply to all construction work done in the United States. While competition among domestic firms in the industry may be strong, the industry as a whole does not face significant competition from foreign contractors. Foreign contractors would also be subject to the requirements of the proposed rule for work done in this country. As a result, the costs of compliance are likely to be passed on in the form of higher prices for completed construction work. The expected increases for each type of construction project are small and are not expected to influence total demand for construction work. For example, the average costs of increased worker protection would add about $40 to the cost of a new $200,000 single-family house.

Reduced worker exposures to hazardous chemicals is likely to generally increase worker productivity and reduce the number of lost workdays due to illnesses. The lower costs associated with illnesses should result in an improvement in the overall efficiency of the construction industry.

Regulatory Flexibility Analysis. Pursuant to the Regulatory Flexibility Act of 1980 [Pub.L. 96–353, 94 Stat. 1164 (5 U.S.C. 60 et seq.)], OSHA has assessed the impact of the proposed standard on small businesses, defined as establishments with fewer than 20 employees. The impacts were evaluated for potential adverse impacts on small firms and their relative consequence compared with large firms.

In general, the costs of compliance for any firm will depend on the extent of worker exposures involved in the activities engaged in and on the extent to which workers are currently protected. The extent of worker exposure is directly related to the number of workers involved and the amount of work being done. For any given activity, work is likely to be done in a similar manner by both large and small firms, with costs proportional to

the scale of the project. Thus, it is estimated that the annual costs of compliance for any firm would also be proportional to the amount of work done over the course of a year.

Most of the compliance costs associated with this rulemaking would be incurred on a per employee basis. For example, costs for disposable dust masks can be estimated per employee for each project and purchased as needed. There is no evidence that small firms would be put at a competitive disadvantage as a result. Small firms compete effectively with large firms for all types of construction work and outnumber large firms by a factor of nine to one.

The size of the estimated compliance costs in relation to both payroll costs and revenues suggests that any potential negative impacts would be negligible for both large and small size firms. OSHA concludes that the proposed regulation will not have an adverse impact on a substantial number of small entities.

Table V–B16 shows the number of small establishments and the average cost for small establishments. The average cost for small firms is approximately half of the average cost for all firms. Approximately 90 percent of all firms are small, and about 44 percent of the total costs of compliance would be borne by these establishments. The proposed standard would not create any differential between small and large firms in the cost of completing work, and thus no competitive disadvantage would be imposed on small firms.

Environmental Impacts. This proposed rule and its major alternatives have been reviewed in accordance with the requirements of the National Environmental Policy Act (NEPA) of 1969 (42 U.S.C. 4321 et seq.), the Guidelines of the Council on Environmental Quality (40 CFR Parts 1500–1517), and OSHA's DOL NEPA Procedures (29 CFR Part 11). As a result of this review, OSHA has determined that the promulgation of this rule would have no significant environmental impact. Any changes that would result from compliance with this proposed rule would tend to reduce emissions of airborne contaminants from construction worksites.

TABLE V–B16.—ESTIMATED COSTS OF COMPLIANCE FOR SMALL AND LARGE FIRMS BY SIC CODE

SIC code	SIC description	Total number of establishments	Number of small establishments	Average cost for all establishments	Average cost for small establishments
1521/22/31	General Contractors, Residential Buildings	119,287	110,406	$30	$12
1541	General Contractors, Industrial-Buildings	7,014	6,492	165	130
1542	General Contractors, Other Buildings	31,337	29,004	332	276
1611	Highway and Street Construction	10,986	8,525	465	126
1622	Bridges, Tunnels, and Elevated Highways	1,159	886	273	57
1623	Water, Sewer, Communication and Power Lines	9,919	7,697	161	33
1629	Heavy Construction, NEC	14,532	11,277	5	2
1711	Plumbing, Heating and Air Conditioning	69,566	63,500	13	7
1721	Painting and Paper Hanging	29,867	27,263	709	447
1731	Electrical Work	49,436	45,125	8	5
1741	Masonry and Stone Work	23,284	21,254	248	148
1742	Plastering, Drywall, and Insulation Work	17,809	16,256	317	109
1743	Terrazzo, Tile, Marble, and Mosaic Work	5,089	4,645	473	282
1751	Carpentry Work	36,009	32,869	16	10
1752	Floor Laying and Other Floor Work	8,174	7,461	115	73
1761	Roofing, Siding, and Sheet Metal Work	25,673	23,434	87	46
1771	Concrete Work	23,422	21,380	160	79
1781	Water Well Drilling	3,414	3,116	96	73
1793	Glass and Glazing Work	4,636	4,232	495	204
1794	Excavation Work	13,422	12,252	110	56
1795	Wrecking and Demolition Work	1,240	1,132	377	152
1796	Installation of Building Equipment	3,777	3,448	156	57
1799	Special Trades, NEC	23,198	21,175	759	374
Total		536,267	489,508	175	85

Note: Small Establishments are Defined as Firms with 1 to 19 employees.
Source: Office of Regulatory Analysis, OSHA, U.S. Department of Labor.

C. Preliminary Regulatory Impact Analysis for Maritime

1. Introduction and Executive Summary

The proposed standard, extending the new Z-table PELs for general industry to the maritime industry, is examined in this report to estimate the costs, benefits, and economic impacts associated with the proposed rulemaking. In addition, a profile of the industry affected by the proposed standard is provided, along with a discussion of the regulatory and nonregulatory environment.

Industry Profile. An estimated 1,681 establishments will be affected by the proposed standard, 590 of which are in SIC 3731 (Shipbuilding and Repairing) and 1,091 of which are in SIC 4491 (Marine Cargo Handling). Of a total employment of about 183,537 workers, 68 percent are employed in shipyards and 32 percent are in marine cargo handling.

Within shipyards, the proposed standard is expected to primarily affect welders and painters. In marine cargo handling, stevedores involved in RO–RO and forklift operations, as well as workers involved in various dry bulk operations, are expected to be primarily affected.

Employee Exposures and Benefits. Based on data obtained from OSHA's Integrated Management Information System (IMIS), a survey conducted for the general industry air contaminants rule, shipyard, marine terminal and longshoring studies, U.S. waterborne commerce data, and expert opinions, OSHA estimated that about 40,530 full-time equivalent workers are potentially exposed to substances affected by the proposed rule. Of these 40,530 potentially exposed workers, approximately 3,615 are estimated to be overexposed.

The overexposures in the shipbuilding and repairing industry result from welding and painting. In marine cargo handling, overexposures are likely to occur during RO–RO and break bulk forklift operations, as well as during the

loading and unloading of dry bulk cement.

OSHA estimates that an average of 245 total illnesses, of which 77 are lost workday cases, result from overexposures to substances affected by the proposed regulation. Of these illnesses, 196 total are expected to be avoided as a result of the proposed rulemaking, 62 of which are lost workday cases.

The proposed rule is also expected to reduce fatalities. Since long latency periods associated with occupationally related illnesses cause difficulty in diagnosing illnesses as being work related, fatalities resulting from such illnesses are difficult to determine. However, some substances covered by the proposed rulemaking to which workers are exposed are known to cause fatalities at levels above the proposed PELs. OSHA therefore believes, that by reducing current PELs, an average of 1 to 2 fatalities per year will be avoided.

Nonregulatory Alternatives. OSHA believes that there are no nonregulatory alternatives that adequately protect most workers from the adverse health effects associated with exposure to the chemicals under consideration. OSHA believes that the tort liability laws and Workers' Compensation do not provide adequate worker protection due to market imperfections. Some employers have not complied with the standards recommended by professional organizations. The deleterious health effects resulting from continued high levels of exposure to hazardous substances require a regulatory solution, and the OSH Act requires the Agency to protect workers' health.

Technological Feasibility and Costs of Compliance. OSHA has determined that the standard is technologically feasible, as all of the provisions can be met by using currently available engineering controls and personal protective equipment.

The proposed rule will require firms to incur costs for engineering controls necessary to bring overexposed workers to levels below the proposed PELs. The total annualized cost for firms to come into compliance with the proposed rule is estimated to be $5,682,245. Of this amount, $5,416,098 will be incurred by shipbuilding and repairing facilities to reduce welding and painting exposures. An estimated $266,147 will be incurred annually by stevedoring establishments to reduce exposures to carbon monoxide and portland cement.

Economic Impacts and Regulatory Flexibility. The potential impact of compliance on the industry was evaluated using estimated revenues and profits for each SIC group. The analysis revealed that even if compliance costs were fully absorbed by the industry (i.e., no pass through of costs to the consumer), neither profitability nor revenues will be significantly reduced. Costs as a percentage of revenues would not exceed 0.07; costs as a percentage of profits would not exceed 1.13 for either industry subsector.

Pursuant to the Regulatory Flexibility Act of 1980 (Pub. L. 96–353, 94 STAT. 1164 [5 U.S.C. 60 et seq.]), OSHA has assessed the impact of the proposed rule on small entities and concludes that there will be no significant effects on a substantial number of small firms. This act requires that in proposing new rules, federal regulatory agencies must examine their potential for imposing disproportionately heavier burdens on small businesses. Nearly 50 percent of the firms covered by the standard employ fewer than 20 employees and are defined as small. For the vast majority of these firms, it is anticipated that the economic impact of the standard will not be significant given the small magnitude of per worker compliance costs.

Other Effects. The proposed rule is not expected to have any significant effect on international trade because of the small magnitude of the costs.

The proposed rule has been reviewed in accordance with the requirements of the National Environmental Policy Act (NEPA) of 1969 (42 U.S.C. 4321, *et seq.*), the regulations of the Council on Environmental Quality (CEQ) (40 CFR part 1500), and OSHA's DOL NEPA Procedures (29 CFR part 11). As a result of this review, OSHA has determined that the proposed standard will have no significant effect on air, water or soil quality, plant or animal life, use of land, or other aspects of the environment.

2. Industry Profile

This section will provide an overview of the industry subsectors covered under the proposed rule. The discussion will include a description of the basic duties performed, the number of employees and establishments, and the financial situations for each SIC group. The proposed standard will cover two four digit SICs: 3731 (Shipbuilding and Repairing) and 4491 (Marine Cargo Handling). SIC 3732 (Boat Building and Repairing) is a separate industry which was covered under the General Industry PEL update.

SIC 3731: Shipbuilding and repairing. The shipbuilding and repairing industry (SIC 3731) includes establishments which engage in the construction, repair, alteration, conversion, and maintenance of ships, barges and lighters, whether self-propelled or towed by other craft [3]. Ship repair work may range from one-time, emergency corrections of particular problems to overhauls involving scheduled maintenance projects lasting from six months to one year, during which ships are completely refurbished and retrofitted in order to upgrade their equipment to the current state of the art. Shipbreaking, which involves disassembling ships for scrap and components, is also included in SIC 3731, although this activity is seldom performed in the U.S. In addition, many shipyards have been actively soliciting non-marine industrial work involving the same operations typically performed in shipbuilding and repair work, such as steel fabrication, welding, boiler repairs and engine overhauls.

The establishments which comprise the U.S. shipbuilding and repair industry range from very large facilities, employing over 25,000 workers, to firms with as few as 2 workers. Of the shipbuilding and repair facilities in the U.S., the largest 3 percent employ over 70 percent of the total workforce. The Census of Manufactures reported that SIC 3731 contained 605 shipyards operated by 542 companies in 1977. This decreased to 580 shipyards operated by 547 companies in 1987. The Census of Manufactures data are collected every 5 years, and 1987 is the last year data are available from that source. Most major facilities are located in the Gulf/Mississippi Region, followed by the East Coast, Great Lakes, and West Coast regions [11].

The U.S. Active Shipbuilding Base (ASB) includes those shipyards which are "open and currently engaged in or seeking contracts for the construction of major oceangoing or Great Lakes ships 1,000 gross tons or over" [11]. During 1989 alone, the U.S. ASB decreased from 19 to 16 shipyards. U.S. ASB shipyards account for about 75 percent of total SIC 3731 employment.

Shipbuilding and repairing is a large scale heavy manufacturing activity which requires both skilled and unskilled labor. Table V–C1 shows the employment trend for SIC 3731 since 1987.

Potential exposures are most likely to result from activities such as welding, painting, abrasive blasting, tank cleaning and grinding. The breakdown of the shipbuilding and repairing industry's workforce by occupational group is shown in Table V–C2.

TABLE V-C1.—TREND IN U.S. SHIPYARD EMPLOYMENT 1980-1989

Year	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989
Average Annual Employment	177,000	184,600	182,400	152,600	151,900	141,000	136,300	126,500	127,100	125,625
Percent Change from Previous Year		4.3	−1.2	−16.3	−0.5	−7.2	−3.3	−7.2	−0.5	−1.2

Source: (1) The years 1980 through 1984 are based on CONSAD Research Group Corp., Data to Support a Regulatory Analysis of the Proposed Standard for Shipbuilding and Repairing, Final Report. Prepared for the U.S. Dept. of Labor, Occupational Safety and Health Administration, under Contract No. J-9-F-4-0024. Nov. 1985.

(2) U.S. Department of Labor, Bureau of Labor Statistics "Employment and Earnings", 1985-1989.

TABLE V-C2.—OCCUPATIONAL BREAKDOWN OF SIC 3731 WORKFORCE

Occupational group	Percent of workforce	Number of workers
Outfitters	[illegible]	[illegible]
Welders	14.7	18,467
Other Steel Trades	14.0	17,588
Painters/Blasters	6.5	8,166
Tank Cleaners	0.7	[illegible]
Maintenance Workers	2.2	2,764
Supervisors	5.7	7,161
Office and Administrative	23.4	29,396
Others	9.0	11,306
Total	100	125,625

Source: CONSAD Research Corp., Data to Support a Regulatory Analysis of the Proposed Standard for Shipbuilding and Repairing: Subpart B, Prepared for the U.S. Department of Labor, OSHA under Contract No. J-9-F-4-0024.

U.S. Department of Labor, Bureau of Labor Statistics, "Employment and Earnings", 1989.

The primary occupational groups affected by the proposed regulation are welders and painters. Welders fuse metal objects by means of an exyacetylene torch or arc-welding apparatus in the fabrication of metal shapes and in repairing broken or cracked metal objects aboard ships. In addition to performing hand welding or brazing operations, the welder may also lay out guide lines or marks on metal parts and may cut metal with a cutting torch. Depending on the task, welders may be actively engaged in welding for up to eight hours per day. Other groups of employees may work in either the same environment or in adjacent compartments. Among these groups are the outfitting trades, carpenters, electricians, outside machinists and others involved in erecting steel such as riggers and grinders.

Market structure. The domestic market for commercial shipbuilding has decreased dramatically over the past twelve years. Nearly all sales have been lost to foreign producers, especially Japan and South Korea, where most ships can be built for less than the U.S. price. Foreign competitors usually have lower labor costs and often receive generous subsidies from their governments, while labor and material costs are relatively high in the U.S. The U.S. ranked thirty-first in worldwide merchant shipbuilding with only 0.1 percent of the gross tonnage on order [9].

The workload of U.S. shipyards is dominated by U.S. Navy ship construction projects. As of October 1, 1989, 91 military combatant and auxiliary ships were under construction or on order by the Navy, and 2 medium endurance cutters were under construction for the U.S. Coast Guard. Among the 91 Navy ships ordered were 21 T-ships, i.e. auxiliary crane ships, hospital ships, fleet oilers, and ocean surveillance ships. However, Navy ships tend to require large-scale, highly technical production which has benefited the larger shipyards more than smaller shipyards. In 1988, the total value of the work completed by U.S. shipyards was $8.7 billion in current dollars, down 2 percent from 1987 when adjusted for inflation [9]. Total revenues in 1989 were $9.1 billion [3].

Despite a declining market for commercial ship construction, shipbuilding and repair facilities in the U.S. continue to invest millions of dollars to improve, expand and modernize their facilities. Over $195 million was invested in the 1989 fiscal year, and industries planned to invest at least $250 million in the year ending June 30, 1990 [9]. Such large investments have dramatically increased the capability of U.S. shipyards, although facilities remain substantially underused.

SIC 4491: Marine cargo handling. The Marine Cargo Handling industry includes those facilities which are responsible for the handling of marine cargo from the time of arrival at shipside, dock, pier, terminal, staging area, or in-transit area to the completion of cargo loading or unloading. SIC 4491 also includes establishments which engage in the transfer of cargo between ships and barges, trains, trucks, pipelines and wharves. The maintenance and operation of piers and docks, including related facilities, are also included in this SIC.

Marine terminals are defined as "wharves, bulkheads, quays, piers, docks and other berthing locations and adjacent storage or contiguous areas and structures associated with the primary movements of cargo or materials from vessel to shore or shore to vessel including structures which are devoted to receiving, handling, holding, consolidation and loading or delivery of waterborne shipments and passengers, including areas devoted to the maintenance of the terminal or equipment. The term does not include production or manufacturing areas having their own docking facilities and located at a marine terminal nor does the term include storage facilities directly associated with those production or manufacturing areas."

For several years, there has been a trend toward public ownership of marine terminals, though most are still privately owned. Because they tend to be operated by or for the exclusive use of a single firm, most privately-owned terminals are directly competitive neither with one another nor with publicly-owned terminals. Privately owned terminals traditionally specialize in bulk cargoes, since large investments are often required in bulk terminal facilities.

Many terminals are publicly owned and leased by private tenants. Stevedoring establishments are firms which contract with a ship's owner, agent, charter operator, or the owner of the cargo to load or unload vessels in ports. The stevedoring firm typically hires longshoremen, usually on a daily basis, from a hiring hall or labor pool of union or nonunion members. The stevedoring labor force size varies directly with the amount of work the stevedoring establishment contracts to perform, which is a function of port activity. At any given time, a stevedoring firm may be working several vessels or none at all. Thus, longshoremen are hired as needed by the stevedoring establishment and discharged upon completion of an individual job. However, some longshoremen, such as equipment room managers or foremen, receive long-term employment commitments from an individual firm and work a standard work week plus required overtime.

There are approximately 57,912 total employees and 1,091 establishments in SIC 4491 [10]. The nature of marine cargo handling work is such that full-

time employees rarely work the typical 2,000 hour work-year. Full-time longshoremen work an average of 1,565 hours per year [5].

Vessels and commodities. Potential exposures to the substances affected by the proposed rulemaking occur during the loading and unloading of a wide variety of commodities. For example, the unloading of vehicles from RO–RO vessels creates a potential for exposure to substances such as carbon monoxide and carbon dioxide. Similarly, the loading of grain into a barge or bulk carrier creates potential exposures to grain dust as well as to any fumigants or pesticides, such as phosphine and methyl bromide, which may have been applied to the grain.

Table V–C3 shows the foreign and domestic waterborne commerce of the United States for 1988 [6]. Vessel types are relevant for addressing potential exposures associated with the shipment of these commodities.

Self-propelled vessels—Bulk Carriers are self-propelled vessels designed to carry bulk commodities such as heavy ores and minerals, pallets and wood chips, coal, and general dry bulk commodities such as grains. Loading a bulk commodity, such as grain, involves a system of conveyors and chutes or spouts. Grain is taken by conveyor from a grain silo to a chute positioned over the open hatch of a ship's hold into which the cargo is loaded. Once the cargo is loaded, one or two longshoremen may be called upon to level the grain in the hold. These vessels may also be used to carry bulky general freight such as logs or steel.

General tankers are vessels arranged for the carriage of liquic cargoes in tanks integral to the hull. The majority of these vessels carry crude oil, petroleum, and petroleum products. Chemical tankers are vessels structurally arranged for particular services or cargoes, such as asphalt, asphalt bitumen, phosphorus, solvents and various specialty chemicals.

Special tankers are tankers which were designed for a specific type of cargo. The characteristics of these vessels effectively limit their applicability to specialty products. Included in this category are molasses, liquid latex, sulphur, and wine tankers.

TABLE V–C3.—WATERBORNE COMMERCE OF THE UNITED STATES 1988

Code	Commodity	Imports	Exports	Total foreign	Domestic	Total
	Farm Products					
0101	Cotton, raw	1,240	1,484,722	1,485,962	2,684	1,488,646
0102	Barley and rye	81,677	2,383,295	2,464,972	704,101	3,169,073
0103	Corn	41,506	49,540,037	49,581,543	32,067,630	81,649,173
0104	Oats	510,626	5,988	516,614	375,979	892,593
0105	Rice	136,678	2,461,712	2,598,390	1,088,610	3,687,000
0106	Sorghum grains	0	5,264,659	5,264,659	2,209,648	8,474,307
0107	Wheat	107,244	44,562,575	44,669,819	14,445,004	59,114,823
0111	Soybeans	50	19,438,538	19,438,618	16,180,935	35,619,553
0112	Flaxseed	0	0	0	29	29
0119	Oilseeds, NEC	27,823	246,802	274,625	35,540	310,165
0121	Tobacco, leaf	177,745	267,259	445,004	1,708	446,712
0122	Hay and fodder	0	0	0	53,259	53,259
0129	Field crops, NEC	77,780	92,159	169,939	27,006	196,945
0131	Fresh fruits	151,035	1,434,284	1,585,319	333,979	1,919,298
0132	Bananas and plantains	3,465,265	9,090	3,474,355	16,233	3,490,588
0133	Coffee, green and roasted	990,126	103,886	1,094,012	31,477	1,125,489
0134	Cocoa beans	265,173	1,771	266,944	3,629	270,573
0141	Fresh and frozen vegetables	197,630	914,868	1,112,498	241,838	1,354,336
0151	Live animals (livestock)	679	13,566	14,245	79,069	93,314
0161	Animals and animal products, NEC	118,801	156,932	275,733	14,007	289,740
0191	Miscellaneous farm products	45,011	29,994	75,005	38,234	113,239
	Forest products					
0841	Crude rubber and allied gums	1,232,760	33,212	1,265,972	2,211	1,268,183
0861	Forest products, NEC	142,512	39,866	182,378	110,554	292,932
	Fresh Fish and Other Marine Products					
0911	Fresh fish, except shellfish	470,760	365,788	836,548	747,566	1,584,114
0912	Shellfish, except prepared	266,691	74,387	341,078	501,791	842,869
0921	Marine shells, unmanufactured	3,187	9,980	13,167	3,079,625	3,092,792
	Metallic Ores					
1011	Iron ore and concentrates	20,894,501	5,536,061	26,430,562	60,825,332	87,255,894
1021	Coppers ore and concentrates	2,173	734,359	736,532	73,479	810,011
1051	Aluminum ores, concentrates	16,559,007	1,062,127	17,621,134	433,593	18,054,727
1061	Manganese ores, concentrates	505,649	5,966	511,615	478,074	989,689
1091	Nonferrous ores, content, NEC	2,053,173	696,015	2,749,188	606,136	3,355,325
	Coal					
1121	Coal and lignite	826,000	94,631,696	95,457,696	196,781,253	292,238,949
	Crude Petroleum					
1311	Crude petroleum	252,524,052	160,558	252,684,610	199,233,326	451,917,936

TABLE V–C3.—WATERBORNE COMMERCE OF THE UNITED STATES 1988—Continued

Code	Commodity	Imports	Exports	Total foreign	Domestic	Total
	Nonmetallic Minerals, Except Fuel					
1411	Limestone	560,379	3,275,977	3,936,356	33,137,372	37,073,728
1412	Building stone, unworked	0	63,653	63,653	4,945	68,598
1442	Sand, gravel, crushed rock	4,640,047	1,458,051	6,298,098	82,595,854	88,893,952
1451	Clay	2,122,119	2,901,500	5,023,619	1,078,282	6,101,901
1471	Phosphate rock	743,913	9,721,686	10,465,605	5,735,058	16,200,863
1479	Natural fertilizer mats, NEC	39,356	0	39,356	4,295	43,651
1491	Salt	5,118,521	706,052	5,824,573	0	5,824,573
1492	Sulphur, dry	0	1,248,552	1,248,552	19,067	1,267,619
1493	Sulphur, liquid	0	0	0	6,960,050	6,960,050
1494	Gypsum, crude and plasters	9,235,781	140,557	9,376,338	1,319,630	10,695,968
1499	Nonmetallic minerals, NEC	2,105,447	631,806	2,737,253	5,335,735	8,072,988
	Ordnance and Accessories					
1911	Ordnance and accessories	22,387	5,769	28,156	569	28,725
	Food and Kindred Products					
2011	Meat, fresh, chilled, frozen	844,833	899,514	1,744,347	318,933	2,063,280
2012	Meat and products, NEC	298,846	32,835	329,681	69,295	398,976
2014	Tallow, animal fats and oils	0	1,334,619	1,334,619	155,044	1,489,663
2015	Animal by-products, NEC	11,654	687,555	699,209	15,180	714,389
2021	Dairy products, NEC	156,676	41,686	198,362	49,761	248,123
2022	Dried milk and cream	31,941	251,121	283,062	16,860	299,922
2031	Fish and shellfish, prepared	347,024	89,569	436,593	374,904	811,497
2034	Vegetables and pre, NEC	749,735	241,658	991,393	246,413	1,237,806
2039	Prep fruit and vegetable juice, NEC	2,149,234	522,755	2,671,989	434,253	3,106,242
2041	Wheat flour and semolina	0	1,215,496	1,215,496	30,807	1,246,303
2043	Animal feeds	369,756	11,988,869	12,358,625	10,118,955	22,477,580
2049	Grain mill products, NEC	295,385	6,632,376	6,921,761	4,631,607	11,553,368
2061	Sugar	1,310,656	213,117	1,523,773	1,939,313	3,463,086
2062	Molasses	1,142,803	284,055	1,426,858	722,762	2,149,620
2081	Alcoholic beverages	2,236,102	309,747	2,545,849	349,317	2,895,166
2091	Vegetable oils, margarine, shortening	1,519,000	1,759,359	3,278,359	1,842,905	5,121,264
2092	Animal oils and fats, NEC	12,528	73,912	86,440	21,977	108,417
2094	Groceries	0	0	0	488,388	488,388
2095	Ice	282,300	1,003	283,303	99,944	383,247
2099	Miscellaneous food products	1,004,158	1,416,398	2,420,556	1,573,968	3,994,524
	Tobacco Products					
2111	Tobacco manufactures	6,384	234,168	240,552	20,302	260,854
	Basic Textiles					
2211	Basic textile products	1,180,050	624,561	1,804,611	66,466	1,871,077
2212	Textile fibers, NEC	43,196	45,708	88,906	10,379	99,285
	Apparel and Other Finished Textile Products, Including Knit					
2311	Apparel	1,082,532	81,409	1,163,941	382,907	1,546,848
	Lumber and Wood Products, Excluding Including Knit					
2411	Logs	223,332	21,087,131	21,310,463	652,227	21,962,690
2412	Rafted logs	0	0	0	9,905,656	9,905,656
2413	Fuel wood, charcoal, wastes	274,024	29,476	303,500	193,902	497,402
2414	Timber, posts, poles, piling	6,821	30,095	36,916	42,039	78,955
2415	Pulpwood, log	86,417	504	86,921	3,945,378	4,032,299
2416	Wood chips, staves, moldings	749,463	6,917,238	7,666,701	3,932,850	11,599,551
2421	Lumber	2,176,776	3,868,877	6,045,653	1,258,148	7,303,801
2431	Veneer, plywood, worked wood	1,093,348	704,344	1,797,692	123,895	1,921,587
2491	Wood manufactures, NEC	352,921	439,571	792,492	230,635	1,023,127
	Furniture and Fixtures					
2511	Furniture and fixtures	900,772	80,631	981,403	201,084	1,182,487
	Pulp, Paper and Allied Products					
2611	Pulp	805,711	5,033,852	5,839,562	477,104	6,316,667
2621	Standard newsprint paper	2,029,093	568,990	2,598,083	128,748	2,726,831
2631	Paper and paperboard	2,032,319	3,684,438	5,716,757	532,108	6,248,865
2691	Pulp and paper products, NEC	176,262	194,398	370,660	1,411,671	1,782,331

TABLE V–C3.—WATERBORNE COMMERCE OF THE UNITED STATES 1988—Continued

Code	Commodity	Imports	Exports	Total foreign	Domestic	Total
	Printed Matter					
2711	Printed matter	199,123	87,082	286,205	21,065	307,270
	Chemicals and Allied Products					
2810	Sodium hydroxide	0	0	0	5,978,436	5,978,436
2811	Crude tar, oil, gas products	630,033	543,638	1,173,671	1,766,155	2,939,826
2812	Dyes, pigment, tanning mats	0	28,614	28,614	805	29,419
2813	Alcohols	2,524,744	914,059	3,438,803	5,923,416	9,362,219
2816	Radioactive materials, wastes	14,831	5,368	20,199	2,558	22,757
2817	Benzene and toluene	653,176	436,714	1,089,890	6,658,253	7,748,143
2818	Sulphuric acid	0	0	0	2,070,239	2,070,239
2819	Basic chemicals and prod, NEC	17,953,775	15,398,090	33,351,865	27,645,535	60,997,400
2821	Plastic materials	763,009	3,503,941	4,266,950	104,905	4,371,855
2822	Synthetic rubber	15,459	530,701	546,160	54,396	600,556
2823	Synthetic (man-made) fibers	78,787	300,547	379,334	2,544	381,878
2831	Drugs	113,033	80,387	193,420	55,340	248,760
2841	Soap	158,884	410,666	569,552	156,272	725,824
2851	Paints	458,424	60,262	533,686	32,490	566,176
2861	Gum and wood chemicals	27,397	212,277	239,674	608,446	848,120
2871	Nitrogenous chem fertilizers	1,928,020	2,719,895	4,647,915	5,794,600	10,442,515
2872	Potassic chem fertilizers	2,470,497	2,700,295	5,170,792	1,412,427	6,583,219
2873	Phosphatic chem fertilizers	116,468	1,356,882	1,473,350	694,587	2,167,937
2876	Insecticides, disinfectants	102,093	187,343	289,436	8,021	297,457
2879	Fertilizer and materials, NEC	121,999	7,107,591	7,229,590	6,675,067	13,904,657
2891	Miscellaneous chemical products	630,654	1,136,175	1,766,829	711,413	2,478,242
	Petroleum and Coal Products					
2911	Gasoline	22,247,104	1,087,194	23,334,298	85,012,138	108,346,436
2912	Jet fuel	3,736,588	1,451,404	5,187,992	13,013,893	18,201,885
2913	Kerosene	102,778	67,669	170,447	1,851,482	2,021,929
2914	Distillate fuel oil	17,882,631	3,870,283	21,752,914	56,442,391	78,195,305
2915	Residual fuel oil	47,966,059	12,661,453	60,627,512	91,696,700	152,524,212
2916	Lubricating oils and greases	185,513	2,152,159	2,337,672	5,885,169	8,222,661
2917	Naptha, petroleum solvents	5,626,425	293,866	5,920,291	9,216,056	15,136,347
2918	Asphalt, tar, and pitches	2,314,747	176,942	2,491,689	10,781,849	13,273,538
2920	Coke, petroleum coke	3,685,966	17,368,726	21,054,692	6,764,238	27,818,930
2921	Liquefied gases	3,483,044	1,343,780	4,826,824	1,772,065	6,598,889
2951	Asphalt building materials	0	29,182	29,182	23,783	52,965
2991	Petroleum and coal prod, NEC	63,298	107,100	170,398	5,281,045	5,451,443
	Rubber and Miscellaneous Plastic Products					
3011	Rubber and misc. plastic products	1,955,781	430,465	2,386,246	190,802	2,577,048
	Leather and Leather Products					
3111	Leather and leather products	872,728	89,183	961,911	31,761	993,672
	Stone, Clay, Glass and Concrete					
3211	Glass and clay, glass and concrete	385,203	311,079	696,282	41,950	738,232
3241	Building cement	13,681,485	125,049	13,806,534	10,391,259	24,197,793
3251	Structural clay products	852,649	131,875	984,424	49,072	1,033,596
3271	Lime	6,043	3,355	9,398	1,523,256	1,532,654
3281	Cut stone and stone products	563,398	11,428	574,826	14,289	589,115
3291	Misc. nonmetallic mineral products	722,563	333,505	1,056,068	284,143	1,340,211
	Primary Metal Products					
3311	Pig iron	678,651	57,652	736,303	595,535	1,331,838
3312	Slag	123,414	79	123,493	1,823,062	1,946,555
3313	Coke, petrol pitches, asphalts, S	0	0	0	3,063	3,063
3314	Iron and steel primary forms	2,140,423	26,771	2,167,194	2,628,244	4,795,438
3315	Iron, steel shapes, exc sheets	11,824,284	52,323	11,876,607	1,352,805	13,229,412
3316	Iron and steel plates, sheets	99,377	970,872	1,070,249	671,512	1,741,761
3317	Iron and steel pipe and tube	2,765,960	219,627	2,985,587	1,232,255	4,217,842
3318	Ferroalloys	1,496,619	35,306	1,531,925	1,078,523	2,610,448
3319	Iron and steel products, NEC	407,920	101,219	509,139	638,408	1,147,547
3321	Nonferrous metals, NEC	191,517	95,926	287,443	28,201	315,644
3322	Copper alloys, unworked	525,005	267,333	792,338	45,040	837,378
3323	Lead and zinc, unworked	320,794	43,363	364,157	26,221	390,378
3324	Aluminum and alloys, unworked	557,610	531,585	1,089,195	22,030	1,111,225
	Fabricated Metal Products, Except Ordnance, Machinery, and Transportation Equipment					
3411	Fabricated metal products	3,329,711	480,568	3,810,279	1,801,520	5,611,799

TABLE V–C3.—WATERBORNE COMMERCE OF THE UNITED STATES 1988—Continued

Code	Commodity	Imports	Exports	Total foreign	Domestic	Total
	Machinery Except Electrical					
3511	Machinery, except electrical	4,045,518	1,699,143	5,744,661	1,080,242	6,824,903
	Electrical Machinery, Equipment and Supplies					
3611	Electrical machinery and equipment	2,941,854	851,471	3,793,325	142,261	3,935,586
	Transportation Equipment					
3711	Motor vehicles, parts, equipment	6,506,754	1,222,392	7,729,146	856,977	8,586,123
3721	Aircraft and parts	11,935	19,967	31,902	1,060	32,962
3731	Ships and boats	50,269	73,141	123,410	24,992	148,402
3791	Misc. transportation equipment	28,293	48,245	76,538	136,194	212,732
	Instruments, Photographic and Optical Goods, Watches and Clocks					
3811	Instruments, time, photo, optical goods	273,431	153,199	426,630	39,642	466,272
	Miscellaneous Products of Manufacturing					
3911	Misc. manufactured products	1,469,020	99,011	1,568,031	1,753,983	3,322,014
	Waste and Scrap Materials					
4011	Iron and steel scrap	235,367	8,758,209	8,993,576	3,252,161	12,245,737
4012	Nonferrous metal scrap	769,193	1,036,363	1,805,556	81,087	1,886,643
4022	Textile waste, scrap, sweep	8,097	278,171	286,268	7,618	293,886
4024	Paper waste and scrap	11,412	4,284,777	4,296,189	1,672	4,297,861
4029	Waste and scrap, NEC	0	0	0	24,948,936	24,948,936
	Special Items					
4111	Water	0	0	0	2,576,239	2,576,239
4112	Commodities, NEC	594,006	491,874	1,085,880	3,927,648	5,013,528
4119	Empty containers	0	0	0	119,552	119,552
9999	Department of Defense	462,674	102,071	564,745	0	564,745
	Total	549,897,111	426,323,874	976,220,985	1,111,772,499	2,087,993,484

Source: Department of the Army, Corps of Engineers, Waterborne Commerce of the United States: Part 5: National Summaries, 1988.

LNG/LPG tankers are vessels designed for the carriage of liquified natural gas, liquified petroleum gas, and other manufactured gases and natural gas products.

Break bulk vessels include carriers of refrigerated and unrefrigerated general cargo. Among the break bulk cargoes are fruits and vegetables, lumber, glass, and machinery. Break bulk vessels may be used to transport standardized containers but they are not designed to specialize in containerized cargo.

Roll-On/Roll-Off (RO–RO) vessels are those that are specially designed to carry automobiles, other vehicles, wheeled containers or trailers. RO–RO ships only use the RO–RO method for loading and unloading, in which vehicles are actually driven on or off the ship. Containerized cargo may be transported on the upper deck of a RO–RO vessel.

Containerships are vessels designed exclusively for the carriage of standardized containerized cargo. These vessels are equipped with permanent container cells and have little or no space for other types of cargoes. Containers are loaded and unloaded usually by cranes and toploaders.

Break bulk, RO–RO vessels and containerships are often grouped together in a broad class as freighters. Both break bulk and RO–RO vessels may be partial containerships, in which one or more, but not all, compartments are fitted with permanent container cells; the remaining compartments are used for other types of cargo. In break bulk and RO–RO vessels, containers are often transported on the upper deck and other cargo is carried in lower decks.

Non-self-propelled vessels—Bulk barges are large double hulled cargo holds without an internal means of propulsion. Virtually all barges used on the inland river system are 195 feet long by 35 feet wide. Most bulk barges have a total draft of 12 feet and are typically loaded so that they draw 9 feet of water. Barges can carry virtually any dry cargo and have an average capacity of 1,500 tons or about 52,500 bushels. Also classified as barges are scows and ocean-going barges. These barges tend to be much larger, have a higher freeboard and carry most of their cargo on deck.

Tanker barges are double hulled barges designed exclusively to haul liquid cargos. Tanker barges lack an internal means of propulsion but do contain pumping systems and equipment for unloading their cargoes. Tanker barges are not of standardized size but are on the order of 240 feet long by 75 feet wide. Tanker barges do have a standard draft of 12 feet.

Gang size. Table V–C4 summarizes average stevedoring gang size by type of operation. Separate crew sizes are itemized for various ship and barge operations. Crew sizes vary dramatically by type of operation and type of vessel. Crew size may range from 2 for tanker barge loading to 64 for RO–RO operations. Also in Table V–C4 are average cargo handling rates per stevedore work-hour.

Market structure. Table V–C5 shows the annual revenues and profits related to longshoring operations by region. Most economic activity occurs in the Gulf/Mississippi region. This region accounts for about 51 percent of total

industry revenues and profits. Annual revenues and profits related to longshoring activities total $8.6 billion and $0.3 billion, respectively.

TABLE V–C4.—SUMMARY OF AVERAGE STEVEDORING GANG SIZE AND CARGO HANDLING RATES

Type of operation	Average total on-site stevedores	Average handling rate per stevedore work-hour (tons/hour)
Ship:		
Break bulk operation	16	5.0
Container operation	18	12.9
Bulk carrier/conveyor loading	13	96.2
Clamshell unloading operation	6	41.7
Tanker operation	6	300.0
Special tanker operation	6	75.0
Fruit and hand loaded cargo	64	1.0
RO–RO car carrier	22	4.1
Barge:		
Barge break bulk operation	8	14.1
Barge bulk unloading with clamshell	3	50.0
Barge conveyor loading	8	56.3
Barge tanker operation	2	180.0

Source: Centaur Associates, Inc., Field Visits.

TABLE V–C5.—ESTIMATED ECONOMIC ACTIVITY RELATED TO LONGSHORING ACTIVITIES

[Millions of dollars]

Region	Annual revenues	Estimated profits
Atlantic	$1,456	$52.6
Gulf/Mississippi	3,317	142.6
Great Lakes	370	15.9
Pacific	1,411	60.7
Totals	6,554	281.8

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis, based on Kearney/Centaur.

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3. Employee Exposures and Benefits

Introduction. Employee exposures to the substances included in the scope of this rulemaking are associated with a wide variety of acute and chronic conditions and illnesses. These include sensory irritation, narcosis, organ system dysfunction, chronic respiratory disease, neurological impairment, allergic sensitization, and cancer. Since OSHA's adoption of existing Federal and consensus standard limits in 1971, toxicologic evidence has become available that shows that adverse health effects can occur as a consequence of exposure to many of the substances listed in OSHA's Z tables, and that such health effects occur even when exposures are maintained at the current Z-table limits. In addition, many substances that have come into widespread use or been introduced since 1971 have been shown to be potentially hazardous in the workplace environment. OSHA thus believes that reducing worker exposure to such substances by lowering existing exposure limits or by adding limits for previously unregulated substances will result in a significantly reduced risk of illness to workers.

This section describes both the methodology used to identify maritime workers potentially exposed to the hazardous substances included in this rulemaking and the expected benefits to those workers resulting from lowering permissible exposure levels. An important existing data base for identifying employees potentially exposed to hazardous substances was OSHA's Integrated Management Information System (IMIS). The IMIS data were used to project expected benefits resulting from lowering permissible exposure levels of the substances being regulated.

Exposure Profile

Methodology. SIC 3731: Shipbuilding and repairing. For each facility inspected, the IMIS files contained information on the number of employees at the facility, the occupation of each employee sampled at the facility, and the number of employees observed to be potentially exposed to each substance for which personal air samples were collected at the facility.

For each substance sampled within each four-digit SIC, the estimated number of employees potentially exposed to that substance in that SIC was determined by the following formula:

$$\frac{\Sigma P_f}{\Sigma E_f} \times W = P$$

where

P_f=number of employees observed to be potentially exposed to the substance at a facility;

E_f=number of employees at each facility inspected by IMIS;

W=number of workers in the industry in 1989; and

P=estimated number of employees potentially exposed to the substance in the industry.

The estimated number of employees currently exposed above the limits for each substance was calculated using the following formula:

$$\frac{\Sigma S_f}{\Sigma T_f} \times P = Z$$

where

S_f=number of samples that exceeded the limit for the substance at all facilities inspected by IMIS in the industry sector;

T_f=total number of personal samples taken for the substance at all facilities inspected by IMIS in the industry sector;

P=estimated number of employees potentially exposed to the substance in the industry sector; and

Z=estimated number of workers in the industry sector currently exposed above the limits for the substance.

Some data for shipyards were collected during the survey conducted for the general industry PEL update. The following information was provided:

- The total number of production employees at the facility;
- The number of employees involved in each process used at the facility;

- The substances used or present in each process;
- The exposure limits used as internal targets or goals at the facility (i.e., OSHA's current limits, ACGIH limits, NIOSH limits, or "Other" limits such as those from material safety data sheets or insurance carriers); and
- Whether employee exposures exceeded the targeted limits for each process/chemical combination present at the facility.

To estimate the number of employees potentially exposed to a given substance, OSHA assumed that all employees who are involved with processes in which the substance is used or present are potentially exposed. Thus, the formula for estimating the number of employees potentially exposed to a substance in the shipbuilding and repairing industry is:

$$\frac{\Sigma X_f}{\Sigma T_f} \times W = P$$

where

X_f = number of employees at the facility who are involved in processes using a given substance;

T_f = total production workforce at the facility;

W = the number of production workers in the industry sector; and

P = estimated number of employees potentially exposed to the substance in the industry sector.

To estimate the number of employees currently exposed above the final limits, OSHA relied on survey responses that indicated whether exposure measurements associated with a process exceeded the facility's internal exposure limits. OSHA interpreted the survey responses as follows:

(1) OSHA assumed that none of the potentially exposed employees are currently exposed above the limit for any substance associated with a process if:

(a) the revised limit is an ACGIH TLV and respondents indicated that exposure measurements did not exceed ACGIH, NIOSH, or some "other" set of limits; or

(b) the revised limit is a NIOSH REL and respondents indicated that exposure measurements did not exceed NIOSH limits.

(2) OSHA also assumed that all of the potentially exposed employees are currently exposed above the limits for all substances associated with a process if:

(a) the revised limit is an ACGIH TLV and respondents indicated that exposure measurements did exceed OSHA, ACGIH, or some "other" set of limits, or

(b) The revised limit is a NIOSH REL and respondents indicated that exposure measurements did exceed OSHA, ACGIH, NIOSH, or some "other" set of limits.

The number of overexposed workers were then summed for each substance across all facilities that responded to the survey in SIC 3731. In instances where the survey data yielded no information on whether or not employees are exposed above the proposed limit for a substance, and no exposure data were available from IMIS on that substance, OSHA assumed that no workers are overexposed to that substance. It is likely, however, that in some of these cases there are employees exposed above the proposed limits, OSHA believes that it has not necessarily accounted for all employees who are exposed above the proposed PELs for the substances included in this analysis. OSHA, thus, believes that the number of overexposed employees may be understated.

A separate analysis of existing IMIS data was done to determine the effect of proposed limits on MEL (mixture exposure limit) overexposures. To estimate the number of workers over the MEL, *employees* rather than incidents with either PEL or MEL overexposures were identified. Each sample in the IMIS database listed the occupation of the employee sampled, all substances and levels at which the worker was exposed, and the number of workers similarly exposed. Potential exposures were calculated by adding the total number of full-time equivalent workers in each occupational group potentially exposed to any combination of substances affected by the proposed rulemaking.

The estimated number of employees currently exposed above the MEL for any combination of substances, which includes one or more substances affected by the proposed rule, was calculated using the following formula:

$$\frac{\Sigma S_f}{\Sigma T_f} \times P = Z$$

where

S_f = number of samples that exceeded the MEL at all facilities identified by IMIS for the industry sector. (Note: This number includes any worker exposed above the PEL for any substance, since, by definition, any worker over a PEL would be overexposed to the MEL);

T_f = total number of personal samples involving substances affected by the proposed rulemaking taken at all facilities identified by IMIS for the industry sector;

P = estimated number of potentially exposed employees in the industry sector; and

Z = estimated number of workers in the industry sector currently exposed above the limits.

SIC 4491: Marine cargo handling. Data from IMIS, a study conducted by Kearney/Centaur, commodity and tonnage data from the U.S. Army Corps of Engineers, and expert judgements were used to estimate worker exposures in SIC 4491. The intermittent work schedules of stevedores caused difficulty in making typical 8-hour TWA measurements. To determine the exposures to hazardous substances in the marine cargo handling industry, OSHA looked at each commodity shipped by water and the number of full-time equivalent workers involved in the processes. Every imported and/or exported, as well as each domestically shipped commodity, was examined for potential exposures.

OSHA looked at the form in which each commodity is shipped, whether in containerized, break bulk, liquid bulk or dry bulk cargo form. OSHA also analyzed commodities requiring the RO–RO method for loading and unloading.

Procedures for loading and unloading the various commodities, potential exposures, and the percentage of stevedores likely to be overexposed were determined.

Once the areas of potential exposure were determined, OSHA estimated the number of full-time equivalent employees involved in the processes and potential exposures.

Estimates of the Number of Potentially Exposed Employees

SIC 3731

The IMIS data base and the survey provided information on the substances posing potential hazards to specific occupational groups in SIC 3731. Table V–C6 shows the occupational groups identified in IMIS, the hazardous substances to which each group is potentially exposed, and the adverse health effects related to exposure.

One of the most widely recognized hazards associated with exposures to airborne substances in the maritime industry involves tank cleaning. Tank cleaners may be potentially exposed to any substance carried in bulk liquid form. Table V–C7 lists liquid substances transported in bulk which are affected by the proposed rulemaking. OSHA estimates that all 843 tank cleaners may be potentially exposed to the substances listed in Table V–C7. Due to the potentially serious nature of the hazards involved and the requirements imposed

by relevant existing standards, employers are generally aware of the need to protect employees during this activity. OSHA believes that the requirements imposed by this proposed rulemaking would not require significant changes in current practices. OSHA requests public comments on this preliminary conclusion.

To obtain an overall estimate of the extent of employee exposures to substances used in SIC 3731, OSHA combined the estimates derived separately from the IMIS and survey data. Where estimates for a given substance could be derived from one data set but not the other, the assessment uses the available estimates without adjustment. Where estimates could be derived from both data sets for the same substance, the combined assessment is based on the average of the available estimates; this approach has the effect of giving equal weight to estimates derived from either the IMIS or survey data. The results are shown in Table V–C8.

TABLE V–C6.—RELEVANT SUBSTANCES AND EFFECTS BY OCCUPATION SIC 3731

Occupation	Substance	Primary basis for limit
Blaster	2-Butoxyethanol	Systemic Toxicity.
	N-Butyl Alcohol	Neuropathy.
	Cobalt, Metal, Fume & Dust	Sensitization.
	Epichlorohydrin	Sensory Irritation.
	Hexone	Liver and kidney Effects.
	Molybdenum, Insoluble	Physical Irritation.
	Nickel (Soluble Compounds)	Respiratory Effects.
	Petroleum Distillates	Miscellaneous Effects.
	Toluene	Narcosis.
	Trimethyl Benzene	Systemic Toxicity.
	Vanadium (V205) fume	Sensory Irritation.
Boilermaker	Cobalt, Metal, Fume & Dust	Sensitization.
	Nickel (Soluble Compounds)	Respiratory Effects.
	Vanadium (V205) Fume	Sensory Irritation.
	Welding Fumes	Systemic Toxicity.
	Zinc Oxide Fume	Systemic Toxicity.
Burner	Cobalt, Metal, Fume & Dust	Sensitization.
	Nickel (Soluble Compunds)	Respiratory Effects.
	Vanadium (V205) Dust	Sensory Irritation.
Carpenter	Cobalt, Metal, Fume & Dust	Sensitization.
	Nickel (Soluble Compounds)	Respiratory Effects.
	Vanadium (V205) Dust	Sensory Irritation.
Electrician	N-Butyl Alcohol	Neuropathy.
Fitter	Cobalt, Metal, Fume & Dust	Sensitization.
	Nickel (Soluble Compounds)	Respiratory effects.
	Styrene	Narcosis.
Grinder	Cobalt, Metal, Fume & Dust	Sensitization.
	Molybdenum, Insoluble	Physical Irritation.
	Nickel (Soluble Compounds)	Respiratory Effects.
	Zinc Oxide Fume	Systemic Toxicity.
Handyman	Cobalt, Metal, Fume & Dust	Sensitization.
	Nickel (Soluble Compounds)	Respiratory Effects.
	Vanadium (V205) Dust	Sensory Irritation.
Laborer	Acetone	Sensory Irritation.
	Carbon Monoxide	Biochemical/Metabolic Effects.
	Styrene	Narcosis.
Mechanic/tool operator	Nickel (Soluble Compunds)	Respiratory Effects.
Painter	2-Butoxyethanol	Systemic Toxicity.
	N-Butyl Alcohol	Neuropathy.
	N-Butyl Glycidyl Ether	Systemic Toxicity.
	Cobalt, Metal, Fume & Dust	Sensitization.
	Cyclohexanone	Liver and Kidney Effects.
	Epichlorohydrin	Sensory Irritation.
	Hexone	Liver and Kidney Effects.
	Methyl Isoamyl Ketone	Analogy.
	Molybdenum, Insoluble	Physical Irritation.
	Naphthalene	Ocular Effects.
	Nickel (Soluble Compounds)	Respiratory Effects.
	Petroleum Distillates	Miscellaneous Effects.
	Stoddard Solvent	Narcosis.
	Toluene	Narcosis.
	Vanadium (V205) Fume	Sensory Irritation.
	Xylene (o-, m- and p- isomers)	Sensory Irritation.
Plastics fabricator	N-Butyl Alcohol	Neuropathy.
	Epichlorohydrin	Sensory Irritation.
	Trimellitic Anhydride	Respiratory Effects.
	Sensory Irritation.	
Steel worker	Cobalt, Metal, Fume & Dust	Neuropathy.
	Nickel (Soluble Compunds)	Sensory Irritation
	Molybdenum, Insoluble	Respiratory Effects.
	Vanadium (V205) Fume	
Sensitization.	Respiratory Effects.	Sensitization.
	Physical Irritation.	Respiratory Effects
	Sensory Irritation.	Physical Irritation
		Sensory Irritation.
Tank cleaner	(See Table V–C7)	

TABLE V-06.—RELEVANT SUBSTANCES AND EFFECTS BY OCCUPATION SIC 3731—Continued

Occupation	Substance	Primary basis for limit
Welder	Cobalt, Metal, Fume & Dust	Sensitization.
	Molybdenum, Insoluble	Physical Irritation.
	Nickel (Soluble Compounds)	Respiratory Effects.
	Styrene	Narcosis.
	Vanadium (V205) Fume	Sensory Irritation.
	Welding Fumes	Systemic Toxicity.
	Zinc Oxide Fume	Systemic Toxicity.

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis, based on IMIS data.

TABLE V-C7.—BULK LIQUID CARGOES COVERED BY THE PROPOSED RULE

Substance	Primary basis for limit
Acetaldehyde	Sensory Irritation.
Acetic Anhydride	Analogy.
Acetone	Sensory Irritation.
Acetonitrile	Systemic Toxicity.
Acrylamide	Cancer.
Acrylic acid	Analogy.
Allyl alcohol	Sensory Irritation.
Allyl chloride	Liver and Kidney Effects.
Ammonia	Sensory Irritation.
Aniline	Biochemical/Metabolic Effects.
Asphalt	Respiratory/Effects.
Butane	Narcosis.
N-Butyl acetate	Sensory Irritation.
Butyl acrylate	Analogy.
sec-Butyl alcohol	Narcosis.
tert-Butyl alcohol	Narcosis.
n-Butyl alcohol	Neuropathy.
p-tert-Butyl toluene	Miscellaneous Effects.
Caprolactum	Sensory Irritation.
Carbon disulfide	Cardiovascular Effects.
Carbon tetrachloride	Cancer.
Chlorine	Sensory Irritation.
Chloroform	Cancer.
beta-Chloroprene	Systemic Toxicity.
o-Chlorotoluene	Miscellaneous Effects.
Cyclohexanone	Liver and Kidney Effects.
Cyclohexylamine	Systemic Toxicity.
p-Dichlorobenzene	Analogy.
Dichloroethyl ether	Sensory Irritation.
Dichloromonofluoromethane	Analogy.
1,3-Dichloropropene	Liver and Kidney Effects.
Dichloropropionic acid	Sensory Irritation.
Dicyclopentadiene	Liver and Kidney Effects.
Diethanolamine	Miscellaneous Effects.
Diethyl phthalate	Miscellaneous Effects.
Diethylamine	Sensory Irritation.
Diethylene triamine	Analogy.
Diglycidyl ether	System Toxicity.
Diisobutyl ketone	Sensory Irritation.
Dioxane	Liver and Kidney Effects.
Epichlorohydrin	Sensory Irritation.
Ethanolamine	Systemic Toxicity.
Ethyl acrylate	Respiratory Effects.
Ethyl benzene	Sensory Irritation.
Ethyl ether	Sensory Irritation.
Ethylene chlorohydrin	Systemic Toxicity.
Ethylene dichloride	Liver and Kidney Effects.
Ethylene glycol	Sensory Irritation.
Ethylene norbornene	Sensory Irritation.
Formamide	Analogy.
Furfural	Sensory Irritation.
Furfuryl alcohol	Sensory Irritation.
Gasoline	Narcosis.

TABLE V-C7.—BULK LIQUID CARGOES COVERED BY THE PROPOSED RULE—Continued

Substance	Primary basis for limit
Glutaraldehyde	Sensory Irritation.
Glycerin	Physical Irritation.
Heptane	Narcosis.
Hexane	Neuropathy.
Hexone	Liver and Kidney Effects.
Hexylene glycol	Sensory Irritation.
Hydrogen fluoride	Sensory Irritation.
Isoamyl alcohol	Narcosis.
Isobutyl alcohol	Analogy.
Isophorone	Narcosis.
Isophorone diisocyanate	Sensitization.
Isopropyl acetate	Sensory Irritation.
Isopropyl alcohol	Sensory Irritation.
Isopropylamine	Sensory Irritation.
Mesityl oxide	Sensory Irritation.
Methacrylic acid	Analogy.
Methyl acetate	Miscellaneous Effects.
Methyl acetylene	Analogy.
Methyl alcohol	Ocular Effects.
Methyl bromide	Neuropathy.
Methyl chloride	Narcosis.
Methyl ethyl ketone	Sensory Irritation.
Methyl formate	Analogy.
Methyl isobutyl carbinol	Sensory Irritation.
alpha-Methyl styrene	Sensory Irritation.
Morpholine	Analogy.
Naphthalene	Ocular Effects.
Nitric acid	Analogy.
2-Nitropropane	Cancer.
Nitrotoluene	Analogy.
Nonane	Analogy.
Octane	Narcosis.
n-Pentane	Narcosis.
Perchloroethylene	Cancer.
Phosphoric acid	Sensory Irritation.
Phthalic anhydride	Analogy.
Propionic acid	Analogy.
n-Propyl acetate	Analogy.
n-Propyl alcohol	Analogy.
Propylene dichloride	Liver and Kidney Effects.
Propylene glycol monomethyl ether.	Sensory Irritation.
Propylene oxide	Analogy.
Styrene	Narcosis.
Sulfur dioxide	Respiratory Effects.
1,1,2,2-Tetrachloroethane.	Liver and Kidney Effects.
Tetrahydrofuran	Sensory Irritation.
Toluene	Narcosis.
Toluene 2,4-Diisocyanate	Sensitization.
1,2,4-Trichlorobenzene	Sensory Irritation.
Trichloroethylene	Narcosis.
1,2,3-Trichloropropane	Liver and Kidney Effects.

TABLE V-C7.—BULK LIQUID CARGOES COVERED BY THE PROPOSED RULE—Continued

Substance	Primary basis for limit
Triethylamine	Sensory Irritation.
Trimethyl phosphite	Miscellaneous Effects.
n-Valeraldehyde	Analogy.
Vinyl acetate	Sensory Irritation.
Vinylidene chloride	Systemic Toxicity.
Xylene (o-, m-, p-isomers).	Sensory Irritation.

Because an employee may be exposed to more than one substance in a given industry, aggregate estimates of the size of the exposed population are presented as an average of minimum and maximum estimates of potential and overexposures. Maximum estimates of the size of the exposed population assume that no employee is exposed to more than one substance; minimum estimates assume the greatest possible extent of multiple chemical exposure. When minimum and maximum estimates are averaged, the result is OSHA's best estimates of actual exposures. For the shipbuilding and repairing industry, the minimum and maximum estimates of potentially exposed workers are 5,856 and 49,040, respectively. The minimum and maximum estimates of the number of workers overexposed are 1,245 and 4,315. OSHA's best estimates of the actual number of potentially exposed and overexposed workers are 27,448 and 2,780, respectively.

The majority of overexposures are to welding fumes and methyl isoamyl ketone. The activities for which these exposures occur are welding and painting. OSHA believes that overexposures may occur during grinding operations. IMIS data, however, do not provide documentation to support this. OSHA requests information on this subject.

TABLE V–C8.—POTENTIAL AND OVER EXPOSURES SIC 3731

Substance	Assessment from IMIS		Assessment from survey		Combined Assessment	
	Workers potentially exposed	Workers above limits	Workers potentially exposed	Workers above limits	Workers potentially exposed	Workers above limits
Acetone	785	0			785	0
2-Butanone (MEK)	1,132	0			1,132	0
2-Butoxyethanol	270	0			270	0
N-Butyl Alcohol	286	38			286	38
N-Butyl Glycidyl Ether	74	0			74	0
Carbon Monoxide	654	0			654	0
Cobalt, Metal, Fume & Dust	5,856	325			5,856	325
Cyclohexanone	366	366			366	366
Epichlorohydrin	225	0			225	0
Hexone	88	0			88	0
Methyl Isoamyl Ketone	1,719	1,031			1,719	1,031
Molybdenum, Insoluble	5,621	0			5,621	0
Naphthalene	75	0			75	0
Nickel (Soluble Compounds)	3,153	104			3,153	104
Petroleum Distillates	1,935	0			1,935	0
Stoddard Solvent	1,875	525	1,012	144	1,444	385
Styrene	4,040	735			4,040	735
Toluene	244	0			244	0
Trimellitic Anhydride	20	10			20	10
Trimethyl Benzene	966	0			966	0
Vanadium (V205) Dust	5,570	0			5,570	0
Vanadium (V205) Fume	5,583	0			5,583	0
Welding Fumes	2,822	1,162	3,036	1,328	2,929	1,245
Xylene (o-, m- and p-isomers)	519	0	7,084	0	3,852	0
Zinc Oxide Fume	1,610	153	1,012	0	1,311	77
Bulk Liquid Cargoes*	843	0			843	0

*The 843 workers in this category represent tank cleaners which may be potentially exposed to bulk liquid cargoes listed in Table V–C7.
Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

When the MEL methodology was used (instead of single or specific substance exposure incident data described above) 22,267 workers were estimated to be potentially exposed to one or more substances affected by the proposed rule, and 2,717 workers had exposures which exceeded proposed PELs or MELs. About 734 of these overexposures exceeded the MEL without exceeding individual PELs. The MEL approach resulted in slightly less control technology and cost than controls needed for specific substance incidents. The reason for electing the more conservative substance specific cost methodology was influenced by the need to develop substance specific benefits.

Since overexposure results from the application of a mathematical formula, the attribution of health effects (and consequently benefits) becomes a problem. There is very little health science data on the combination effects of chemicals acting synergistically (formaldehyde and wood dust, asbestos and tobacco smoke are notable exceptions). Traditionally the health effects related to individual substances have provided the basis with which to develop potential benefits of proposed OSHA rules. The potential benefits of avoiding MEL overexposure are very difficult to quantify since individual substances within a mixture often produce different health effects. There is a practical problem of correctly apportioning risk reduction factors across all chemicals in a mixture. While mathematically possible, it is unclear that health science can provide guidance as to what risk reduction factors can be assigned to the individual components in a mixture. Absent this guidance, OSHA focused on risks posed by individual substances and has made benefit projections on this basis. This focus on individual substances influenced the cost methodology and insured consistency on the separate sides of the cost to benefit equation (i.e., costs related to individual substance control and benefits unique to individual substance exposures).

On balance, this approach probably has the effect of slightly exaggerating estimated costs of the proposed PELs. Benefits may also be exaggerated to the extent that multiple overexposures are concentrated on a few workers (the methodology adopted assumes they are not). As health science advances, and the true risk of mixture exposures becomes better defined, future analysis may find higher risk and, as a result, potential benefits in reducing MEL overexposures.

SIC 4491

Exposure potentials exist from the loading and unloading of a wide variety of commodities.

Risks of exposure during the loading and unloading of containerized cargoes exist only if there is a breakage. Leaks in containerized cargoes are rare. If a leak does occur, all longshoremen are removed from the site and specialists are called in to clean up the spills.

There is also little risk of worker exposure to hazardous substances during the loading and unloading of bulk liquid cargoes. The loading or unloading of liquid cargoes involves connecting pipes from the valve of the tank holding the cargo to the valve of the tank to which the cargo is to be loaded. The valves are opened and the cargo is pumped only after hook-up is complete. The operation of the pumps is done from a console located away from the deck. Stevedores are involved only in the hook-up and disconnection of the pipelines and are not required to be present during the actual transfer of cargo. Any potential exposures would be negligible and of short durations.

The loading and unloading of dry bulk cargoes creates a potential for exposure to various dusts, fumigants and pesticides. The loading and unloading of grain, for example, creates a potential for exposure to grain dust, as well as to any pesticide or fumigant applied to the grain. Generally, grain handling poses little hazard to stevedores. Grain is usually loaded using a system of conveyors and chutes or spouts. Grain is

moved by electric conveyor from dock to ship and dropped through the spout into the hold of the ship [2]. This procedure may take several days, depending on the size of the cargo. Besides someone to observe the grain loading, no other workers are involved during this procedure. Workers may be called upon to level the grain in the hold of a vessel only after the grain is loaded. Any exposures to grain dust would be of short duration. A potential does exist for overexposures to occur during the breakdown of conveyors or other equipment necessary in this loading procedure. However, OSHA has no evidence of any overexposures and requests additional information.

There has been concern about worker exposures to fumigants, such as ethylene dibromide (EDB), which are known carcinogens or known to cause serious illnesses such as permanent liver and kidney diseases [3]. However, risks of exposures have declined dramatically since the move away from liquid applications of pesticides and fumigants and the banning of EDB use in the United States. Phosphine and methyl bromide are the fumigants commonly used in grain handling which are affected by the proposed rulemaking. These substances are in the form of pellets which are dropped through tubes into the grain in the hold of a vessel. The pellets dissipate while the cargo is in transit. This procedure for pesticide/fumigant application greatly reduces the risk of exposure to fumigants and pesticides.

Roughly 79 million tons of grain are handled by stevedores each year. The average handling rate per stevedore work hour for conveyor loading is 96.2 tons. Applying this rate to the total tonnage handled and assuming a 2,000 hour work-year yields 411 full-time equivalent workers potentially exposed to grain dust, phosphine and methyl bromide each year. No stevedores are likely to be overexposed to these substances.

Significant exposures exist during RO–RO and forklift operations. Of the substances affected by the proposed rulemaking, the most significant exposures in the marine cargo handling industry are to carbon monoxide and portland cement.

The RO–RO method of loading and unloading creates a risk of exposure to substances such as carbon monoxide and carbon dioxide. Carbon monoxide exposures are the most significant. According to Kearney/Centaur approximately 1,847 full-time equivalent employees are involved in RO–RO operations. Most RO–RO ships have good ventilation systems which reduce carbon monoxide levels.

The loading and unloading of break bulk cargoes involving the use of forklifts also create a potential for exposure to carbon monoxide. Although most break bulk ships are well ventilated, some older break bulk ships lack sufficient ventilation. There has, however, been a move towards the use of electric and diesel powered forklifts. There are roughly 2,000 full-time equivalent workers involved in forklift operations.

OSHA estimates that, under existing conditions, approximately 20 percent of RO–RO and forklift operators would not be in compliance with the proposed PEL for carbon monoxide. IMIS data support this estimate. Applying this percentage to the 3,847 full-time equivalent workers potentially exposed to carbon monoxide yields 769 overexposed workers.

Overexposures to portland cement are likely to occur during the unloading of dry bulk cement. Approximately 24.1 million tons of building cement are unloaded each year. Of this amount, roughly 90 percent (21.7 million tons) is shipped in dry bulk cargo form. Dry bulk cement is unloaded either by pneumatic means (vacuums) or by clamshell buckets. Roughly 98 percent of the tonnage (21.3 million tons) is vacuumed and 2 percent (0.4 million tons) is unloaded by clamshell.

The average handling rate per stevedore work-hour is 96.2 tons per hour for dry bulk operations and 45.9 for clamshell operations. Applying these rates to their respective tonnages and assuming a 2,000 hour work-year yields 115 full-time equivalent workers potentially exposed to portland cement each year. OSHA believes that roughly 50 percent of these workers would be under the proposed PEL for portland cement, leaving 58 full-time equivalent workers overexposed.

Potential exposures also exist during the loading of dry bulk cement. Approximately 10.5 million tons of building cement are loaded each year. Of this amount, roughly 9.5 million tons are in dry bulk cargo form. Applying the 96.2 tons/hour handling rate yields about 49 full-time equivalent workers potentially exposed to portland cement annually. None of these workers are likely to be overexposed.

Once the cargo is loaded or unloaded, the hold is usually either cleaned in preparation for new cargo, or trimmed to level the cargo in the hold. Approximately 31.2 million tons of bulk cement were loaded or unloaded in 1989. The average barge or bulk cargo ship has four holds and carries 30,000 tons. Assuming that it takes three workers and 2.5 hours/worker to clean or trim a hold, 16 full-time equivalent workers are potentially exposed to portland cement. Roughly 8 of these workers are estimated to be overexposed.

TABLE V–C9.—POTENTIAL AND OVER EXPOSURES SIC 4491

Substance	Process	Primary basis for limit	Potential exposures (FTE workers)	Over exposures
Carbon Monoxide	RO—RO Operations Forklift Operations	Biochemical/Metabolic Effects	3,847	769
Coal Dust	Sweeping/Trimming	Respiratory Effects	2,673	0
Cobalt, Metal, Fume and Dust	Longshoring	Sensitization	2,735	0
Grain Dust	Grain Handling	Respiratory Effects	411	0
Hydrogen Fluoride	Longshoring	Sensory Irritation	2,317	0
Methyl Bromide	Grain Handling	Neuropathy	411	0
Phosphine	Grain Handling	Systemic Toxicity	411	0
Portland Cement	Dry Bulk Cement Handling	Physical Irritation	180	66
Vanadium (V205) Dust	Longshoring	Sensory Irritation	97	0
Total			13,082	835

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

Based on IMIS data, consultation with industry experts and data compiled by consultants under contract with OSHA, other exposures are likely to exist in addition to those mentioned above. These include exposures during various other bulk cargo handling activities; these estimates are shown in Table V–C9, along with the exposures discussed earlier.

Summary

As shown in Table V–C10, there are an estimated 40,530 workers potentially exposed and 3,615 workers overexposed to substances affected by the proposed rulemaking.

TABLE V–C10.—SUMMARY OF POTENTIAL AND OVER EXPOSURES MARITIME INDUSTRY

Industry sector	Potential exposures	Over exposures
Shipbuilding and Repairing SIC 3731	27,448	2,780
Marine Cargo Handling SIC 4491	13,082	835
Total	40,530	3,615

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

BENEFITS

Illnesses

Illness rates are derived from the 1989 BLS Occupational Injuries and Illnesses data base. These rates are expressed as the annual number of illnesses per 100 full-time equivalent employees. Reducing employee exposures to hazardous substances to levels below those associated with adverse health effects will result in a decrease in the number of illnesses.

OSHA estimated the number of annual illnesses that would be averted by applying current rates to the estimated number of overexposed workers in each SIC.

Based on the Bureau of Labor Statistics 1989 publication of Occupational Injuries and Illnesses in the United States, the occupational illness rate for the shipbuilding and repairing industry is 5.8 per 100 full-time workers (total cases) and 1.8 per 100 full-time workers (lost workday cases). Approximately 30 percent of the illnesses represented by these illness rates (1.74 total and 0.54 lost workday) are addressed by the proposed rule, while 70 percent of the illnesses are the result of physical agents, repetitive motion, etc. The illness rates published by BLS only include cases documented and recorded by employers. In addition, a large number of chronic illnesses resulting from exposures in the workplace may never be reported, since long latency periods make it difficult for employers and physicians to diagnose illnesses as being work related. Illness rates, therefore, tend to understate the actual number of occupationally related illnesses. OSHA's best estimate is that these illness rates may understate the actual number of occupationally related illnesses by a factor of 5.

Applying these illness rates to the average number of overexposed workers in SIC 3731 yields 242 total illnesses, of which 75 are lost workday cases. OSHA's best estimate is that 80 percent (194 total and 60 lost workday) of these illnesses would be avoided annually as a result of the proposed rule.

The occupational illness rate for the marine cargo handling industry is reported to be 0.2 per 100 full-time workers (total cases) and 0.1 per 100 full-time workers (lost workday cases). Approximately 40 percent of the illnesses accounted for are addressed by the proposed standard. A factor of 5 was applied to these illness rates to account for the understatement.

Applying the illness incidence rates to the number of overexposed workers in SIC 4491 yields 3 total illnesses, 2 of which are lost workday cases. Assuming an 80 percent effectiveness rate, the proposed rule should prevent 2 lost workday illnesses.

The estimated number of annual illnesses prevented by the proposed rule for both industry subsectors are presented in Table V–C11. In addition to OSHA's best estimate of an 80 percent effectiveness level, a sensitivity analysis using 70 percent and 90 percent effectiveness rates is also presented.

Fatalities

TABLE V–C11.—REDUCTION IN ILLNESSES

Industry subsector SIC code	Number of illnesses resulting from exposures		Illnesses prevented by effectiveness					
			70%		80%		90%	
	Total	LWD	TL	LWD	TL	LWD	TL	LWD
Shipbuilding and Repairing SIC 3731	242	75	169	53	194	60	218	68
Marine Cargo Handling SIC 4491	3	2	2	1	2	2	3	2
Total	245	77	171	54	196	62	221	69

Sources: U.S. Department of Labor, Bureau of Labor Statistics, 1989 Occupational Injuries and Illnesses in the United States.
U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

There are no statistical data on fatalities from work related illnesses in the maritime industry. However, there are studies which show that fatalities do result from exposures to substances included in OSHA's Z-Table. For example, a study of silicosis in sandblasters conducted in the early seventies showed that deaths did result from silica exposures in New Orleans shipyards [9]. Although the actual level for silica remains unchanged, OSHA believes the new formula, which is easier to understand and allows for more accurate monitoring, will increase compliance.

There are an estimated 1,245 workers overexposed to welding fumes, for which there is currently no PEL. Welding fumes often cause metal fume fever and sometimes damage the small airways, causing interstitial pneumonia at levels above the proposed PEL.

Carbon monoxide, is known to cause fatalities at exposures above the proposed PEL.

Because some substances are known to cause fatalities at high exposure levels, OSHA believes that the proposed rule will prevent fatalities. OSHA estimates that annual fatalities avoided could average 1–2 annually.

Summary

OSHA estimates that 196 total illnesses and 62 lost workday illnesses would be avoided annually as a result of the proposed rule. OSHA also believes that fatalities will be prevented as a result of the proposed rule, probably 1–2 on average, annually.

SOURCES

1. CONSAD Research Corporation. Final Report—Data to Support a Regulatory Analysis of the Proposed Standard for Shipbuilding and Repairing. Prepared for the U.S. Department of Labor, Occupational Safety and Health Administration. Under Contract No. J–9–F–4–0024.

2. Finlay, William. Work on the Waterfront: Work Power and Technological Change in a West Coast Port. Philadelphia: Temple University Press, 1988.

3. Kearney/Centaur. Economic Assessment of Draft Revisions to OSHA Longshoring Standard. Prepared for the U.S. Department of Labor, Occupational Safety and Health Administration. Under Contract No. J–9–F–8–0032. March 1990.

4. Mahaney, Francis X. Jr. "Pesticide Exposure in Grain Industry Raises Cancer Risks." Journal of the National Cancer Institute. Vol. 82, No. 10. May 16, 1990.

5. U.S. Department of the Army. Corps of Engineers. Waterborne Commerce of the United States. 1988.

6. U.S. Department of Commerce. Bureau of the Census. County Business Patterns. 1988.

7. U.S. Department of Labor. Bureau of Labor Statistics. Employment and Earnings, 1990.

8. U.S. Department of Labor. Bureau of Labor Statistics. Occupational Injuries and Illnesses in the United States by Industry, 1988.

9. Ziskind, Morton et al., "Silicosis in Shipyard Sandblasters". International Shipyard Conference, University of Southern California School of Medicine, Dec. 13–15, 1970.

4. Assessment of Nonregulatory Alternatives

For a full discussion on the need for and the appropriateness of a regulatory solution to address occupational exposures to air contaminants, see section V.B.4. of this preamble.

5. TECHNOLOGICAL FEASIBILITY

Introduction. This section presents a technological feasibility analysis of the maritime industry's ability to meet OSHA's proposed permissible exposure limits (PELs).

The control of workplace exposures to toxic chemicals involves combining a variety of standard techniques to solve problems unique to each situation. OSHA believes that proposed levels can be met using currently available controls and equipment and work practices. Consistent with OSHA regulations and policy, this section examines the feasibility of engineering controls and work practices to control employee exposures, in preference to personal protective equipment. However, under certain circumstances, respiratory protection may be necessary.

Engineering controls. Engineering controls involve the use of local exhaust ventilation, general ventilation, isolation of the worker and enclosure of the source of emissions process modifications, equipment modifications, and substitution of non-hazardous chemicals. These methods may be used alone or in combination of any two or more controls depending upon the needs of the specific situation.

Perhaps the most widely used technique for controlling chemical exposures is the use of ventilation. General ventilation uses the movement of air within the general work space to displace or dilute the contaminant with fresh outside air. General ventilation is not typically the preferred control method in most operations due to the large volumes of air movement required. Local exhaust ventilation is concentrated on moving smaller volumes of air, exhausted from the point at which contaminants are generated thereby removing contaminants at the source.

Personal protective equipment. Where it is impractical to apply engineering or work practice controls, or where their application will not consistently reduce employee exposures below the proposed PELs, personal protective equipment such as respirators or dust masks may be used to prevent or reduce exposures.

SIC 3731: Shipbuilding and repairing. The major air contaminants in the shipbuilding and repairing industry result from welding and painting.

Welding. OSHA has determined that welding activities cause overexposures to the following substances (shown with proposed PELs):

- Cobalt, Metal, Fume & Dust (0.05 mg/m³)
- Nickel (Soluble Compounds) (0.1 mg/m³)
- Styrene (50 ppm or 215 mg/m³)
- Welding Fumes (5 mg/m³)
- Zinc Oxide Fume (10 mg/m³ STEL)

OSHA believes that all of the proposed PELs for these substances can be achieved using portable, local exhaust systems comprised of a vane-axial suction fan, flexible duct, and portable capture hood.

Painting. OSHA determined that overexposures during painting activities exist for the substances shown below (with their proposed PELs):

- N–Butyl Alcohol (50 ppm (C) or 150 mg/m³(C) SKIN)
- Cyclohexanone (25 ppm or 100 mg/m³ SKIN)
- Methyl Isoamyl Ketone (50 ppm or 240 mg/m³)
- Stoddard Solvent (100 ppm or 525 mg/m³)

OSHA believes that the proposed PELs for painters can be met by using a portable exhaust system consisting of a vane-axial suction fan, flexible duct, and portable capture hood. In cases where ventilation cannot maintain the chemical concentration below the PEL, painters would require respirators. Gloves should be worn to reduce dermal exposure to substances for which OSHA has a skin notation.

Other exposures. OSHA also estimated that a very small number of plastics fabricators are overexposed to trimellitic anhydride which has a PEL of 0.005 ppm or 0.04 mg/m³. This PEL could be achieved using engineering controls similar to those identified for welding and painting.

SIC 4491: Marine cargo handling—RO–RO operations and break bulk forklift operations. Carbon monoxide overexposures result from RO–RO and break bulk forklift operations. The new PEL for carbon monoxide is 35 ppm or 40 mg/m³ TWA and a ceiling level of 200 ppm or 229 mg/m³. This PEL can be achieved using a vane-axial fan and flexible duct.

Clamshell operations. Clamshell operations have been determined to cause overexposures to portland cement. Sealed lips and dust covers are available to seal the openings in the clamshell buckets through which particles are likely to escape and reach the clamshell operator. These controls should be sufficient to reduce exposures below the proposed 10 mg/m³ total dust and 5 mg/m³ respirable fraction levels.

Trimming/hold cleaning. All overexposures which result from trimming (leveling dry bulk cargoes in hold) and cleaning holds can be brought below the proposed PELs using personal protective equipment. The use of engineering controls in these cases is believed to be impractical.

Summary. Since all proposed PELs can be achieved using currently available engineering controls and personal protective equipment, OSHA has determined that the proposed standard is technologically feasible.

6. Costs of Compliance

Introduction. Costs are related to engineering controls and personal protective equipment needed for specific processes which involve the use of hazardous substances. These costs are presented in this section. Costs of compliance result from the purchase, installation, operation and maintenance of equipment to maintain workers' exposures at or below the levels specified in the proposed standard.

Costs of Compliance. The following operations have the potential for worker overexposure and will incur costs:

SIC 3731

- Welding
- Painting/coating

SIC 4491

- Loading and Unloading of Bulk Cement

—Clamshell Operations
—Trimming/Cleaning

- Non-Electric Forklift Operations within vessels
- RO–RO operations

This section summarizes for each process: (1) The extent of current worker exposure, (2) the additional controls that will be needed to reduce worker overexposures, and (3) the costs that facilities in the maritime industry will incur to come into compliance with the proposed PELs.

SIC 3731—Welding. Welding is an integral part of the shipbuilding and repairing industry. Fumes and gases from welding and cutting cannot be easily classified. In welding procedures, the composition of the fumes is determined by the electrode or consumables themselves, coatings on the work (e.g., paint, galvanizing, or plating), contaminants in the atmosphere (e.g., halogenated hydrocarbon vapors from cleaning and degreasing activities), etc. Thus, fumes include those originating from volatilization, reaction, or oxidation of consumables, base metals and coatings, and the atmospheric contaminants. In Section 3, it was determined that welders are currently overexposed to cobalt, nickel, styrene, welding fumes and zinc oxide fume.

It is assumed that exposures are a result of welding in semi-enclosed environments (i.e. the hull of a ship). An estimated 1,612 workers or 58 percent of all overexposed workers in SIC 3731 are welders. To control these exposures, OSHA believes it will be necessary to install portable, local exhaust systems comprised of a vane-axial suction fan, flexible duct, and portable capture hood. Costs for these systems are broken down as follows: $1,850 for a vane-axial suction fan which pulls 2000 cfm and uses a 0.5 hp electric motor (including purchase, installation/hookup); $262 for the 100 feet of duct made of a flexible fiberglass coated fabric with a corrosion resistent metal helix and a 5" diameter (including purchase, installation/hookup) @ $2.62 per foot; and $150 for a portable hood with a 5"×10" outlet and a 5" diameter inlet (including purchase, installation/hookup). This results in a total investment cost of $2,262. Assuming a useful life of 5 years, and a 10 percent discount rate, the annualized cost for a portable local exhaust system is $597.

Annual costs consist of labor to move the system and manipulate the hood during welding, electricity to power the fan, and annual maintenance costs. Labor required to move a portable system into position and manipulate the hood during welding is estimated to be $780 per year, per system. This is derived by assuming that it takes 10 minutes to set up the system. If the system is set up daily throughout the year, this is equivalent to 43.33 hours a year [(10 mins./day)/(60 mins./hour)]×(5 days/week)×(52 weeks/year). Assuming the labor rate for a nonsupervisory worker in SIC 3731 with 30% benefits is $18 per/hour, the labor cost to set up the system is estimated to be $780 per year (43.33 hrs./year×$18 per/hr.). Maintenance costs are estimated to be 10% of the capital cost. In the case of this system, it is equivalent to $226 per year (0.1×$2,262).

The cost of electricity for operating the system is estimated to be approximately $39 per year. Since one horse power (hp) is equivalent to 0.746 kilowatts, the 0.5 hp generated by fan uses 0.373 kilowatts per hour. Applying the average national electricity rate of $0.05/kwh, for five days a week for the entire year, the cost is equivalent to $39 per year (0.373 kw/hr.×8 hrs./day×5 days/wk.×52 wks/yr.×$.05/kwh). The total annual operating and maintenance cost for the portable local exhaust system is calculated to be $1,045 per year ($780+$39+$226).

The total annualized investment cost plus the annual operating and maintenance cost is $1,642 per year ($597+$1,045). It was assumed that one portable local exhaust system will be needed for each of the 1,612 overexposed welders. The total annualized cost of preventing welding overexposures to substances for which new or updated PELs are proposed is estimated to be $2,646,904 (1,612 workers overexposed × $1,642 per year).

Painting and plasticizing. Painting is also an integral part of the shipbuilding and repairing industry. Chemicals present in paints or coatings are a potential source of overexposure. In Section 3, it was determined that painters are currently overexposed to several different chemicals for which new or updated PELs are being proposed. These chemicals are n-butyl alcohol, cobalt, cyclohexanone, methyl isoamyl ketone, nickel, and stoddard solvent.

(There is minimal exposure in plastics fabrication. A small number of plastics fabricators (10) in the shipbuilding industry were estimated to be over exposed to trimellitic anhydride, a colorless solid used in preparation of resins, adhesives, and polymers. The data obtained for plastics fabricators and painters are combined in this analysis since the controls required per exposed worker are roughly the same.)

Overexposures result from inadequate engineering controls or the non-use of full protection respirators. Of the workers in SIC 3731 who are overexposed to a combination of chemicals affected by the proposed rulemaking, roughly 42 percent (1,168 workers) are painters. OSHA believes that exposures can be reduced to the proposed levels using portable, local exhaust systems comprised of a vane-axial suction fan, flexible duct, and a portable capture hood. In cases where ventilation cannot maintain the chemical concentration below the proposed PEL, the painter (or plasticizer) must be provided with the proper respiratory equipment. Gloves will be necessary to reduce dermal exposure where skin notations are present. Costs for putting together a system to control exposure are broken down as follows: $1,975 for a 4,000 cfm vane-axial fan which uses a 1 hp electric motor (including purchase, installation/hookup), $262 for 100 feet of duct made of a flexible fiberglass coated fabric with a corrosion resistent metal helix and a 5" diameter (including purchase, installation/hookup) @ $2.62 per foot, and $150 for a portable hood with a 5"x10" outlet and a 5" diameter inlet (including purchase, installation/hookup). Engineering controls may not be capable of maintaining chemical concentrations below the proposed PELs in all operations. For some maintenance and repair work and work in confined spaces, full-face powered air purifying respirators will be needed to supplement engineering controls. OSHA estimates that of the 1,168 overexposed workers in this activity, roughly 30 percent will need such respiratory protection. Respirator costs are estimated to be $495 each. Engineering controls and PPE result in a total investment cost of $2,882 ($1,975+$282+$150+$495). Assuming a useful life of 5 years, and a 10 percent interest rate, the annualized cost for the system is $760.

Annual costs include labor time to move the system and manipulate the hood during painting, electricity to power the fan, and annual maintenance costs. Labor required to move the portable system for work and to manipulate the hood during painting is estimated to be $1,170 per year, per system. This is derived by assuming that 15 minutes is required to set up the system. If set up daily throughout the

year, total time is equivalent to 65 hours a year [(15 mins./day)/(60 mins./hour)]×(5 days/week)×(52 weeks/year). Assuming the labor rate for a nonsupervisory worker in SIC 3731 with 30% benefits is $18 per hour, the labor cost to set up the system is estimated to be $1,170 per year (65 hrs./year×$18 per/hr.). Maintenance costs are estimated to be 10% of the capital cost. In the case of this system, it is equivalent to $288 per year (0.1×$2,882).

The cost of electricity for operating the system is estimated to be approximately $78 per year. The 1 hp fan uses 0.746 killowatts per hour. Applying the $0.05 electricity rate and assuming that the system is operated eight hours each day for five days a week for the entire year, the cost is equivalent to $78 per year (0.746 kw/hr.×8 hrs./day×5 days/wk.×52 wks/yr.×$.05/kwh).

The total annual operating and maintenance cost for the portable local exhaust system is calculated to be $1,536 per year ($1,170+$78+$288). The total annualized investment cost plus the annual operating and maintenance cost is $2,296 per year ($760+$1,536). It is assumed that each overexposed worker will also require one full-face powered air purifying respirator. Overexposures to n-butyl alcohol and cyclohexanone will require the use of gloves to prevent dermal exposures. The unit cost for a pair of gloves is $4.33; each pair is assumed to last 5 days. Applying these rates to the 404 overexposed workers results in an annual cost of $87,466. Thus, the total annual cost of preventing painting overexposures to substances affected by the proposed rulemaking is estimated to be $2,769,194 [(1,168 workers overexposed × $2,296 per year)+$87,466].

Summary. Table V–C12 shows the compliance costs for SIC 3731. Total annualized costs are $2,646,904 for welding operations and $2,769,194 for painting. Overall cost of compliance for SIC 3731 is $5,416,098.

SIC 4491.—RO–RO and Break Bulk Forklift Operations. Carbon monoxide exposures occur during RO–RO and forklift operations which are conducted within the confines of ships lacking adequate ventilation. An estimated 769 workers are overexposed to carbon monoxide.

The control system for carbon monoxide exposures consists of a vane-axial fan, tripod, transport cart, duct storage cannister and wired supported flexible duct. The average cost of this control system is a $2,590 per unit investment cost. Amortizing this cost at 10 percent over 5 years results in an annualized investment cost of $683.

TABLE V–C12.—COMPLIANCE COSTS FOR SIC 3731 BY OPERATION

Cost	Welding	Painting		
		Respiratory protection	Skin protection	Total
One-time investment cost per unit	$2,882	$2,882		
Annualized investment cost per unit	597	760		
Annual operating and maintenance costs/unit	1,045	1,536		
Total annualized cost per unit	1,642	2,296	4.33	
Number of units required	1,612	1,168	20,200	
Total cost	$2,646,904	$2,681,728	$87,466	$5,416,098

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

The labor required to move each unit is $1,625 per year. The cost was determined assuming an average wage rate of $25, including benefits and assuming that 15 minutes is required to set up the unit [(15 mins./day)/(60 mins./hr.)×(5 days/wk.)×(52 wks./yr.)×$25/hr].

The electricity required to run each unit is $78. This cost was determined by applying the $0.05 per kwh rate to the electricity required to power the 1 hp motor of the unit [(1 hp)×(0.746 kw/hp)×(8 hrs./day)×(5 days/wk.)×(52 wks./yr.)×(0.05/kwh)]. The operating costs, estimated to be 10 percent of the investment costs, equal $259 per unit.

The annual recurring costs of compliance per unit total $1,962 ($1,625+$78+$259).

The annualized cost, per unit, to reduce exposures to carbon monoxide is $2,645 ($683+$1,962).

The average gang size for RO–RO operations and break bulk forklift operations is 12 workers, which yields approximately 64 gangs. Assuming that one control is required per gang, the total annualized cost for reducing exposures to carbon monoxide is $169,280.

Portland cement exposures. Exposures to portland cement in marine cargo handling (SIC 4491) occur during the loading and unloading of dry bulk cement. Overexposures occur during clamshell operations. An estimated 66 workers are overexposed to portland cement (58 during clamshell operations and 8 during trimming and cleaning).

Sealed lips and dust covers can be used to reduce portland cement exposures for clamshell operators. The average cost of a sealed lip and dust cover is $3,650. Amortizing this investment cost at 10 percent over 5 years yields a $963 annualized cost.

Annual maintenance and operation costs for each unit are estimated to be 10 percent of the investment cost or $365.

The annualized cost for clamshell operations, therefore, is $1,328 per unit. Assuming that 58 units will be required, the total annualized cost to reduce portland cement exposures during clamshell operations is $77,024.

Overexposures to portland cement during trimming and cleaning operations will require the use of disposable dust masks to reduce exposures. Approximately 34.6 million tons of portland cement are loaded and unloaded per year. Of this amount, about 90 percent or 31.2 million tons are transported in dry bulk cargo form. Assuming that the average barge has 4 holds and carries 30,000 tons, holds are trimmed or cleaned approximately 4,160 times per year. The average cost of a dust mask is $1.59 and an average of 3 workers are required to trim or clean each hold. This indicates that 12,480 disposable dust masks and $19,843 will be required annually to reduce exposures to portland cement.

Summary. Table V–C13 shows compliance costs for SIC 4491. Total costs are $169,280 for RO–RO and forklift operations, $77,024 for clamshell operations and $19,843 for trimming and cleaning operations. Total annualized cost for SIC 4491 is $266,147.

TABLE V–C13.—COMPLIANCE COSTS FOR SIC 4491 BY OPERATION

Cost	RO–RO/ forklift operations	Clamshell operations	Trimming/ cleaning	Total
One-time investment cost per unit	$2,500	$3,650	$0	
Annualized investment cost per unit	683	963	0	
Annual cost per unit	1,962	365	1.59	
Total annualized cost per unit	2,645	1,328	1.59	
Number of unit required	64	58	12,480	12,602
Total cost	169,280	$77,024	$19,843	$266,147

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

Summary. Total costs resulting from the proposed rule are presented in Table V–C14. A total of $5,682,245 would be required annually for firms to come into compliance with the proposed PELs.

TABLE V–C14.—MARITIME INDUSTRY COSTS OF COMPLIANCE (1989 DOLLARS)

SIC group operation	Costs
SIC 3731:	
Welding	$2,646,904
Painting	2,769,194
Subtotal	5,416,098
SIC 4491:	
RO–Ro/forklift operations	169,280
Clamshell operations	77,024
Trimming/ cleaning	19,843
Subtotal	266,147
Total costs	$5,682,245

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

Sources

1. CONSAD Research Corporation. Final Report—Data to Support a Regulatory Analysis of the Proposed Standard for Shipbuilding and Repairing. Prepared for the U.S. Department of Labor, Occupational Safety and Health Administration. Under Contract No. J–9–F–4–0024.

2. Kearney/Centaur. Economic Assessment of Draft Revisions to OSHA Longshoring Standard. Prepared for the U.S. Department of Labor, Occupational Safety and Health Administration. Under Contract No. J–9–F–8–0032. March 1990.

3. Means Facility Cost Data, 1990.

4. U.S. Department of the Army. Corps of Engineers. Waterborne Commerce of the United States. 1988.

5. U.S. Department of Commerce. Bureau of the Census. County Business Patterns. 1988.

6. U.S. Department of Labor. Bureau of Labor Statistics. Employment and Earnings. 1990.

7. U.S. Department of Labor. Bureau of Labor Statistics. Occupational Injuries and Illnesses in the United States by Industry. 1988.

8. U.S. Department of Labor. "Shipboard Ventilation for Hazardous Atmospheres." Safety in Industry: Maritime Safety Data.

7. Economic Impacts

Introduction. This section will assess the economic impact of the proposed standard on firms involved in maritime operations.

Economic impact—Compliance costs. As shown in Table V–C15, compliance costs total $5,682,245, with 96% borne by shipbuilding and repairing facilities and 4% borne by longshoring facilities. The greatest portion of this dollar amount will be incurred by shipbuilding and repairing facilities to reduce welding and painting exposures to levels below the proposed PELs.

The costs per affected employee at risk for SIC 3731 and SIC 4491 are shown in Table V–C16.

Costs as a percent of revenue and profit. Revenue and profit estimates, as well as estimates of costs as percentages of revenues and profits, for the maritime industries are shown in Table V–C17. Estimates of costs as percentages of revenues are less than 0.07 percent for both industry subsectors. Compliance costs as percentages of profits for both SIC groups range from 0.09 percent and 1.13 percent, with an average of 0.75 percent.

TABLE V–C15—COMPLIANCE COSTS FOR MARITIME INDUSTRY

Industry sector	Compliance costs
SIC 3731:	
Welding	$2,646,904
Painting	2,769,194
Total SIC 3731	5,416,098
SIC 4491:	
RO–RO and forklift operations	169,280
Dry bulk cement operations	96,867
Total SIC 4491	266,147
Total	$5,682,245

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

TABLE V–C16.—UNIT COSTS PER AFFECTED EMPLOYEE AT RISK

Industry subsector	Annualized costs	Number of workers	Average cost per worker
Shipbuilding and Repairing SIC 3731	$5,416,098	125,625	$43
Marine Cargo Handling SIC 4491	266,147	57,912	5
Maritime industry	5,682,245	183,537	31

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

TABLE V–C17.—ESTIMATED IMPACT OF COMPLIANCE COSTS ON MARITIME INDUSTRY PROFITS AND REVENUES

Maritime industry subsector	Estimated annual revenues (millions)	Estimated annual profits (millions)	Cost as percent of revenues	Cost as a percent of profits
SIC 3731	$9,053	$480	0.06	1.13
SIC 4491	6,554	282	0.00	0.09
Total	$15,607	$762	0.04	0.75

Source: 1989 Dun & Bradstreet Financial Data.
U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

These costs are clearly feasible. Most shipbuilding costs are born by the U.S. government for the purchase of warships. Federal contracts require compliance with OSHA regulations, so shipbuilding costs will be passed forward. In any event, costs are tiny in relation to sales and small in relation to profits. The ship repair costs for the civilian sector will be tiny and clearly feasible if absorbed, but are likely to be passed forward because repairs are done in the U.S. when the place of the breakdown or scheduling makes it necessary to be done here. The costs for longshoring, being so tiny as to be inconsequential, are clearly feasible.

Regulatory flexibility. Pursuant to the Regulatory Flexibility Act (Pub. L. 96–353, 94 Stat. 1164 [5 U.S.C. 60 et seq.]), the Assistant Secretary has made an assessment of the standard and has concluded that it will not have a significant impact upon a substantial number of small entities. If a small firm is defined as one that employs fewer than 20 workers, then nearly 50 percent of the firms in maritime would be classified as "small".

The important criterion of a regulatory flexibility analysis is whether the standard will impose significant costs upon small entities, whether it will have relatively greater negative impact on small entities than on large ones, and whether it will benefit large firms, thereby putting small firms at a competitive disadvantage.

If the costs of compliance for small firms are relatively minor and proportional to the size of the firm, then there is no significant differential effect. The standard does not require any significant capital expenditures, and the compliance costs will tend to be proportional to the level of employment. The analysis showed that declines in profitability expected to result from the rule are not expected to be significant.

Any differential impact on small firms is expected to be insignificant in view of the very small magnitude of compliance costs relative to the overall operating costs for all firms. The significance of the differential impact on profits is further reduced by the likelihood that all firms in the industry should be able to pass a substantial portion of their compliance costs on to consumers. For these reasons, OSHA concludes that small entities will not be significantly affected by the standard.

Other Effects

International trade. The standard is not likely to have any effect on international trade because of the extremely small magnitude of any price increase that will be required for passing forward compliance costs.

Environmental. The rule and its major alternatives have been reviewed in accordance with the requirements of the National Environmental Policy Act (NEPA) of 1969 (42 U.S.C. 4321, et seq.), the regulations of the Council on Environmental Quality (CEQ) (40 CFR part 1500), and OSHA's DOL NEPA Procedures (29 CFR part 11).

The OSHA standard contains provisions for reducing exposures to hazardous substances, and is not expected to have any significant environmental effects.

Sources

1. Dun and Bradstreet Financial Data. 1989.

2. Kearney/Centaur. *Economic Assessment of Draft Revisions to OSHA Longshoring Standard.* Prepared for the U.S. Department of Labor, Occupational Safety and Health Administration. Under Contract No. J–9–F–8–0032. March 1990.

3. U.S. Department of Commerce. Bureau of the Census. *County Business Patterns.* 1988.

4. U.S. Department of Labor. Bureau of Labor Statistic. *Employment and Earnings.* 1989.

D. Preliminary Regulatory Analysis for Agriculture

1. Industry Profile

Introduction. This chapter examines sectors affected by the proposed permissible exposure limit (PEL) update for Agriculture. Based upon a review of several sources, it has been determined that over 782,000 workers, employed at approximately 74,000 establishments, within SICs 01–09, are at risk of potential exposure to substances found on OSHA's Z tables. This chapter summarizes the affected sectors and presents an industry profile detailing the number of establishments, employment and financial characteristics of establishments potentially affected by OSHA's proposed rule. Specifically considered in this industry profile are establishments categorized within SIC 01, agricultural crop production; 02, agricultural livestock production; and 07, agricultural services. Forestry, 08 and fishing, hunting and trapping, 09 are also covered in this report.

Introducing permissible exposure limits in agriculture will not only bring the United States into line with such countries as the United Kingdom and the Federal Republic of Germany regarding the protection of agricultural workers from airborne contaminants, but it will provide consistency in coverage and protection to all U.S. workers from airborne contaminants.

Profile. Industries involved SICs 01 through 09 in the United States are composed of a large group of diverse establishments, all of which are engaged in various aspects of production and service. The establishments range in physical size from a few acres to thousands of acres, and their purpose and organization can be vastly different (ranging from a small dairy farm in Maine to a huge wheat farm in Kansas).

Concerning the agricultural sector, a continuing trend has been a reduction in the number of employees necessary for operations through increased reliance on automation. Although this trend has leveled off in the past few years, the net result has been that a few large producers with many employees account for a very large share of total production.

Summary of affected establishments and population at risk. Estimates of the number of establishments potentially affected by the proposed standard were developed by Meridian Research Inc.[1], based on the *Census of Agriculture*[2], Farm Labor[3], and the *Agricultural Work Force of 1987*[4]. Due to the seasonality of agricultural production and the inability to obtain data which precisely identified the number of agricultural establishments employing more than 10 workers at any one time during the year, SIC groups affected by the proposal were separated into two sections, (1) agricultural production and (2) agricultural services, forestry, fishing, hunting and trapping.

Agricultural Production

Cash Grains. For the grain sector (SIC 011), the number of workers on large farms was derived by relying on information describing labor requirements for grain production and a size breakout of the industry by acreage. Between 1981 and 1985, 3 hours of labor were required to produce and harvest 1 acre of grain[5]. Knowing that employees at grain facilities work an average of 10 hours per day and 90 days per year[2], it is estimated that a workforce of 11 employees can be expected to produce and harvest 3,300 acres of grain. Applying this figure to statistics from the 1987 Census of Agriculture[2] it is estimated that there were 576 grain farms with over 3,000 acres or more in 1987, representing .1 percent of all grain farm establishments. Because estimates of employment on large farms are not available from any

published sources, it is assumed that each of the large grain farms employed slightly more than 11 employees in 1987, this equates to approximately 6,350 workers.

Crops Other Than Grain. To calculate the estimated number of potentially exposed workers and establishments in crops other than grain (SICs 013 through 018), estimates developed in support of OSHA's Field Sanitation standard were used[6]. Table V–D1 shows the estimated number of workers and establishments presented for SIC codes 013 through 018 in the Regulatory Impact Analysis for the Field Sanitation standard. It is estimated that 492,000 employees work on the estimated 58,000 farms that employ 11 or more workers in these sectors.

For the livestock-producing sectors (SICs 021 through 029), the same methodology used to estimate worker population and establishment numbers in the grain sector was relied on. Meridian based its estimates on the amount of labor required per unit of production.

Beef Cattle. It is estimated there are 34,200 employees working on 1,710 cow-calf production establishments which employ more than 10 workers. This figure is based on the estimate that it requires 7.16 hours of hired labor to produce each calf or feeder cow[7]; farmhands work an average of 157 days per year and 10 hours per day[7]; and according to the 1987 Census of Agriculture[2], there are an estimated 1,710 calf establishments that sold 2,500 or more cows each in 1987. Based on these figures, Meridian estimated that a farm employing roughly 11 employees would be capable of producing approximately 2,400 cows each year (2,400=11 employees×157 days/year×10 hours/day×1 cow/7.16 hours). Because this is roughly equivalent to the base production capacity of the 1,710 farms identified by the Bureau of the Census, it is estimated there are 1,710 farms in cow-calf production establishments employing more than 10 workers.

Based on the labor required to operate large commercial feedlots it is estimated that 9,000 workers are employed on the estimated 300 feedlots likely to employ 11 or more workers. Approximately 1 employee is necessary for every 1,000 head of cattle in inventory, (based on Meridian's personal communication with a livestock commodities specialist, USDA Economic Research Service 1991). Hence, a feedlot which employs 11 or more workers is likely to have the feedlot capacity to accommodate over 11,000 cattle. Based on data from Schertz[9], 422 feedlots had a capacity of 8,000 or more head, of these, 140 had a capacity from 16,000 to 31,999 head and 61 had a capacity over 32,000 head. From this data, it is reasonable to assume 300 feedlots have a capacity of 11,000 head or more and thus are likely to employ 11 or more workers. Because it is believed that an average of 30 workers are employed on large feedlots (representing an average feedlot capacity of 30,000 head), it is estimated that a total of 9,000 workers are employed on the 300 establishments likely to employ more than 10 workers.

Dairy Farms. Dairy farm employment and establishment estimates were derived from data reported in the 1987 Census of Agriculture[2], Agricultural Statistics 1986[5] and information from site visits by OSHA's contractor Meridian. Using a methodology based on sales, it is estimated that there are 700 dairy farms employing 10,500 workers. Meridian estimated that an average dairy farm which has more than 10 employees has sales greater than $2 million[1]. Because the 1987 Census of Agriculture[2] does not estimate the number of dairy farms with annual sales of $2 million or more, Meridian used data on the number of farms with sales between $.5 and $1 million (2,940) and the number of farms with sales in excess of $1 million (1,457). Based on a straight line basis, it was estimated that in 1987 there were 700 farms with sales over $2 million (1,457/2) (farms in excess of $1 million are roughly half the farms with sales between $.5 and $1 million; therefore, it is assumed that farms with sales over $2 million are roughly half the number of farms with sales in excess of $1 million). Assuming each farm employs an average of 15 employees[1] it is estimated there are 10,500 workers employed at the 700 dairy farms which employ more than 10 persons.

Hog Farms. It was estimated by Meridian that there are currently only 10 hog farms in the United States which employ 11 or more workers each. This is based on data by Schertz[9] who estimated that there were 10 exceptional U.S. hog producing facilities (those which produce more than 30,000 head per year). It is believed that since 1979, this number has not changed because the number of farms in the 1987 Census of Agriculture[2] found in the 5,000 head plus category has not substantially increased. Based on site visit data, it is assumed that an average of 39 workers are employed at each of the large facilities. Hence, the total number of workers employed at these 10 large farms is estimated to be 390.

Poultry and Egg Producing Facilities. Because only one person-year of labor is required to produce approximately 150,000 to 200,000 broiler chickens or 50,000 to 60,000 turkeys each year[9] it is believed a poultry farm employing 11 or more workers would be capable of producing 2 million broilers annually. Data on chicken and turkey producing facilities indicate that no facilities exist in the United States which are of this size. It was determined, however, that 11 or more workers are occasionally engaged in bird catching operations at poultry farms, but that the bird catchers actually work for poultry processors (e.g., for firms such as Perdue or Holly Farms, which are classified in general industry rather than agriculture).

It is estimated that there are 15 chicken egg farms employing a total of 450 workers that fall into the scope of this study. These estimations are based on information that an egg farm employing 11 or more workers would be capable of housing half a million to several million chickens a year and would employ an average of 30 workers[9] (personal communication with poultry specialist from the North Carolina Agricultural Extension Service). Currently, there are approximately 15 such facilities in the United States. Therefore, it is believed these establishments employ roughly 450 workers (15 x 30).

From Table V–D1 the estimated number of farms and the number of workers employed in the agricultural production sector that would be affected by the proposal are shown to be 63,792 and 570,890 respectively. In addition, Table V–D1 shows the types of establishments, the number of employees and the number of establishments expected to fall under the scope of the proposed rule by primary SIC (determined by the commodity which accounts for at least 50 percent of the value of the farm's sales). In reviewing Table V–D1, it is interesting to note that the total number of farms is roughly equivalent to the total number of workers. One explanation for this is many of the small farms are operated by individuals whose major source of income is derived from non-farm activities, in which case their establishment may be classified as a farm, but they may not be classified as a farm worker. Also, non-paid workers on farms, eg. family members, are not counted by the sources from which the estimates were derived. Affected establishments are not divided into small or large sizes because when concerned with agriculture, the proposed rule is restricted to farms with more than 10 employees. Farms with more than 10 employees are considered large farms.

TABLE V–D1.—ESTIMATED NUMBER OF FARM WORKERS EMPLOYED ON ESTABLISHMENTS WITH MORE THAN 10 WORKERS, BY TYPE OF FARM

Principal product [1] and corresponding SIC [2]	Total number of farms [1]	Number of workers	Estimated number of farms likely to employ more than 10 workers	Estimated number of farmworkers employed on farms with more than 10 employees
Grain:				
011 Cash Grains	450,306	558,000	578	6,350
0111 Wheat	65,126			
0112 Rice	7,396			
0115 Corn	125,557			
0116 Soybeans	125,533			
0119 Cash Grains NEC	134,784			
Cotton:				
0131 Cotton	27,674	105,000	850	[3] 9,000
Tobacco:				
0132 Tobacco	87,776	136,000	19,225	[3] 98,000
Other Crops:				
013 Other Field Crops	128,178	317,000	1,600	[3] 33,000
0133 Sugarcane	4,625			
0134 Irish Potatoes	5,168			
0139 Field Crops NEC	116,385			
Vegetables and Melons:				
016 Vegetables & Melons	28,801	178,000	8,165	[3] 137,000
Fruits and Tree Nuts:				
017 Fruits & Tree Nuts	56,123	240,000	22,287	[3] 175,000
0171 Berry Crops	7,905			
0172 Grapes	11,375			
0173 Tree Nuts	14,364			
0174 Citrus Fruits	14,163			
0175 Deciduous Tree Fruits	21,547			
0179 Fruits & Nuts NEC	18,969			
Horticultural Specialty:				
018 Horticultural Specialty	31,469	99,000	5,954	[3] 40,000
0181 Ornamental Floriculture and Nursery Products	30,573			
0182 Food Crops Grown Under Cover	896			
Livestock except dairy, poultry, and animal specialties				
021 Beef Cattle	892,267	407,000		
0211 Beef Cattle, feedlot	72,515		[4] 300	[4] 9,000
0212 Beef Cattle, except feedlot	643,831		[4] 1,710	[4] 34,200
0213 Hogs	107,929		[4] 10	[4] 390
0214 Sheep & Goats	32,385			
0219 General Livestock	35,607			
Dairy Farms:				
024 Dairy Farms	138,311	259,000	[4] 700	[4] 10,500
Poultry and Eggs:				
025 Poultry & Eggs	38,494		[4] 0	
0251 Broiler Fryer	19,264		[4] 15	[4] 450
0242 Chicken Eggs	13,343			
0253 Turkey & Turkey Eggs	3,239			
0254 Poultry Hatcheries	865			
0259 Poultry & Eggs NEC	2,263			
Animal Specialties:				
027 Animal Specialties	87,855			
0271 Fur-bearing & Rabbits	2,652			
0272 Horses and Other Equine	75,393			
0273 Animal Aquaculture	2,089			
0279 Animal Specialties NEC	7,721			
Other Crops and Livestock:				
019 and 029 General Farms	80,215	162,000	2,600	18,000
0191 General Farms, Primarily Crops	57,800			
0291 General Farms, Primarily Livestock	22,327			
Total	2,087,759	2,461,000	63,792	570,890

[1] Source: USDA Farm Labor Survey [2]. Numbers may not compute due to rounding.
[2] Source: *1987 Census of Agriculture* [3].
[3] Based on RIA for Field Sanitation Standard [6].
[4] Based on *1987 Census of Agriculture, Economic Indicators of the Farm Sector, 1985, Agricultural Statistics, 1986*, site visit reports, and telephone interviews with USDA experts. Source of Table: Meridian Research Inc., [1].

Financial Profile. Financial data are not broken out for farms with more than 10 employees; however, Meridian used data profiling the largest farms as a proxy for those farms employing more than 10. Table V–D2 shows aggregate net farm income and the relative share of total income earned by each sales class. Farms with sales in excess of $250,000 per year, the farm category most likely to have more than 10 employees at any time during the year, accounted for 63.2 percent of net farm income in 1988.

Table V–D3 depicts average farm balance sheet data by farm sales size. In general, while debt/asset ratios are highest for the largest farms, which may indicate a weaker financial position, their return on assets is much higher than smaller farms.

TABLE V–D2.—AGGREGATE AND AVERAGE PER FARM NET INCOME BY SALES CATEGORY, 1988

Sales category (annual sales/farm)	Aggregate net farm income		Number of farms (in 000s)	Average per farm, 000s		
	Billions of dollars	Percent of total		Gross income	Production costs	Net income
$500,000 and up	21.6	43.3	30	1,907.6	1,195.2	712.4
$250,000 to $499,999	8.9	19.8	76	409.1	279.2	129.9
$100,000 to $249,999	10.8	21.6	216	184.4	134.5	49.9
$40,000 to $99,999	5.6	11.2	320	82.8	63.4	19.4
<$39,999	2.0	4.0	1,554	16.9	15.7	1.2
Total	49.9	100.0	2,196			

TABLE V–D3.—AVERAGE FARM BALANCE SHEETS (IN 000S) AND DEBT/ASSET RATIOS, BY FARM SALES CLASS, 1988

Item	Farm Sales Class				
	>$500,000	$250,000 to $499,999	$100,000 to $249,999	$40,000 to $99,999	<$39,999
Assets:					
Real Estate	2,452.5	1,079.5	589.1	325.6	101.4
Livestock, Crops	501.5	182.1	100.2	57.5	14.6
Machinery	219.1	128.6	85.8	51.5	14.9
Liquid Assets	22.3	18.6	6.1	4.8	3.5
Other	275.3	58.9	26.5	11.8	2.0
Total	3,470.7	1,467.7	807.7	451.2	136.4
Debt:					
Real Estate	472.2	167.6	87.7	44.4	10.6
Other	580.0	134.4	64.1	30.8	6.5
Total	1,052.2	302.0	151.8	75.2	17.1
Equity	2,418.5	1,165.7	655.9	376.0	119.3
Debt/Asset Ratio	0.303	0.205	0.188	0.166	0.125
Return on Assets	0.205	0.088	0.062	0.043	0.010

Source (Table V–D2) Meridian Research Inc., Exhibit 2–7[1]
Source (fTable V–D34 Meridian Research Inc., Exhibit 2–8[1].
Numbers may not compute due to rounding.

Agricultural Services, Forestry, Fishing, Hunting and Trapping. Agricultural Services include establishments in SIC 07 primarily engaged in performing soil preparation services, crop services such as cotton ginning, other animal services, farm labor and management services, and landscape and horticultural services for others on a contract or fee basis.

Forestry includes establishments in SIC 08 primarily engaged in the operation of timber tracts, tree farms, forest nurseries, and related activities such as reforestation services and the gathering of gums, barks, balsam needles, maple sap, moss, and other forest products.

Fishing, hunting and trapping includes establishments in SIC 09 primarily engaged in the catching or taking of finfish, the catching or taking of shellfish, the operation of fish hatcheries or preserves, commercial hunting and trapping, and the operation of game preserves.

For most operations in SIC 07 through 09, OSHA's proposed rule would not have an adverse impact. Many of these operations do not involve PEL chemicals, do not give rise to potentially hazardous exposures, or are covered by EPA regulations.

A summary of the number of establishments in SICs 07 through 09, taken from County Business Patterns[7], is presented in Table V–D4. Forestry, fishing, hunting and trapping are considered by OSHA to be exclusive of agriculture and hence not restricted by the congressional rider restricting OSHA's jurisdiction to agricultural establishments with more than 10 persons. For this reason, the number of establishments and employees for SICs 08 and 09 under the establishments with 10 or more employees heading does not reflect the subtraction of establishments with 10 or fewer employees. Within sectors SICs 07–09, the dominant subsector is landscape and horticultural services (SIC 0781, 0782, and 0783). Estimates of the number of establishments and employees potentially affected by the proposed rule are presented in Table V–D5. As indicated on Table V–D5, approximately 10,000 establishments and 218,172 employees are likely to be affected by the proposed rule. Total annual payroll for these establishments is nearly $2.7 billion, or an average of $270,000 per establishment. Financial information on agricultural services and forestry was compiled by Meridian from Dun and Bradstreet's Industry Norms and Key Business Ratios[10] to give a general overview of the entire industry (data on 0831, Forest Nurseries and Gathering of Forest Products, were not available). Financial data for fishing, hunting and trapping was gathered from Dun and Bradstreet's Industry Statistics and Financial Analysis[11]. These 1989 data do not include all establishments within the industry, but instead represent only a sample. Table V–D6 presents totals for reporting firms within each four-digit subgroup. Fishing, hunting and trapping has the largest return on assets (16.5%), followed by cotton ginning (11%) and lawn and garden services (10.7%).

TABLE V–D4.—NUMBER OF AGRICULTURAL SERVICE AND FORESTRY ESTABLISHMENTS IN 1987, BY SIC

Type of establishment (SIC)	No. of establishments	Percent of all establishments
Agricultural Services:		
Soil Preparation Services (071)	581	1
Crop Services (072)	3,309	6
Animal Services, except veterinary (075)	8,086	13
Farm Management Services (0762)	506	1
Landscape and Horticultural Services (078)	40,543	76
Forestry (08):	1,796	3
Fishing, Hunting and Trapping (09)	1,826	4
Total	55,447	105

Source: Meridian Research Inc., Table 2–10[1].

TABLE V–D5.—NUMBER OF ESTABLISHMENTS, EMPLOYMENT, AND PAYROLL FOR AGRICULTURAL SERVICES AND FORESTRY, 1987

Industry (SIC)	Total industry		Establishments employing 10 or more [1]		
	No. of establishments	No. of employees	No. of establishments	No. of employees	Annual payroll ($ mil.)
Soil Preparation Services (0711):	581	4,498	104	3,212	50.7
Crop Services (0721, 0722, 0723, 0724)	3,309	33,271	564	26,516	346.5
Animal Services (0751, 0752)	8,086	31,510	563	16,049	217.3
Farm Management Services (0762)	506	4,059	93	3,110	48.3
Landscape and Horticultural Services (0781, 0782, 0783)	40,543	216,965	4,813	143,169	2,098.2
Forestry (0811, 0831, 0851):	1,796	17,692	[2] 1,796	[2] 17,696	234.6
Fishing, Hunting and Trapping (0912, 0913, 0919, 0921, 0971)	1,826	8,420	[2] 1,826	[2] 8,420	171.9
Subtotal	55,447	316,415	9,959	218,172	3,165.5
Agricultural Production (01, 02)	2,087,759	2,461,000	63,792	570,890	NR
Total	2,143,206	2,777,415	73,751	789,062	NR

NR—Not reported.
Source: Meridian Research Inc. Table 2–11[1].
[1] USDC, *1990 County Business Patterns*, specifies its employment category as 10 or more rather than more than 10. No adjustment was made for this anomaly; thus the estimated number of establishments employing more than 10 may be slightly inflated.
[2] Establishments and employees in SICs 08 and 09 have traditionally been considered by OSHA to be outside of the agricultural sector. Establishments and employees in these SICs fall completely within OSHA's jurisdiction and are not subject to the congressional rider on OSHA's appropriations.

Summary

Combining agricultural production, agricultural services, forestry, fishing, hunting and trapping estimates, OSHA calculates that a total of 73,751 establishments are potentially affected by the proposed standard (Table V–D5). This total is strictly for large establishments (farms employing more than 10 workers) [1] which make up roughly 3.4% of all U.S. farm operations in SICs 01–09. The total population at risk is an estimated 789,062 employees (Table V–D5) which constitute 28% of all workers in SICs 01–09. Three major groups could be affected by the proposed rule. These groups are SIC 01—agricultural crop production; 02—agricultural livestock production; 07—agricultural services. Forestry, fishing, hunting and trapping may also be affected. Industry segment SIC 01, Agricultural Crop Production, is the largest affected sector; over 58,457 establishments and 498,350 employees. This seems reasonable in light of the fact that this segment is the most labor intensive segment of all the SICs.

TABLE V–D6.—SELECTED FINANCIAL DATA FOR VARIOUS GROUPS WITHIN THE AGRICULTURAL SERVICES INDUSTRY (SIC 07) AND FORESTRY (SIC 08), 1989

Name of industry group	SIC No.	No. of reporting firms	Average per firm, in $000s					Ratios for median firm		
			Total assets	Total liabilities	Net worth	Net sales	Net profit after tax	Current ratio	Return on sales	Return on assets
Soil Preparation Services	0711	176	289.6	131.5	158.1	637.4	48.6	1.9	6.6	9.7
Crop planting, cultivating, and protecting	0721	295	300.6	17.8	282.8	455.0	39.6	2.3	7.6	9.0
Crop harvesting	0722	56	491.8	206.1	285.7	1,1150.0	61.0	2.2	3.8	5.4
Crop preparation	0723	663	1,308.5	671.2	637.3	2,312.0	76.6	1.6	2.7	4.3
Cotton ginning	0724	339	1,062.2	397.3	664.9	1,651.4	153.6	2.0	6.7	11.3
Livestock services	0751	153	256.7	111.7	145.0	700.0	42.7	2.3	4.6	8.3
Animal specialty services	0752	160	195.7	75.3	120.4	347.0	30.5	2.7	5.5	7.5
Farm management services	0762	97	569.9	281.5	288.4	964.8	52.1	1.6	4.9	8.8
Landscape counseling	0781	889	194.8	100.5	94.3	524.4	35.1	1.8	5.2	10.6
Lawn and garden services	0782	1,528	163.1	84.2	78.9	500.0	33.5	1.7	5.3	10.7
Ornamental shrub and tree services	0783	415	191.2	93.1	98.1	486.1	29.7	1.7	4.9	9.3

[1] Excluding fishing, hunting and trapping.

TABLE V–D6.—SELECTED FINANCIAL DATA FOR VARIOUS GROUPS WITHIN THE AGRICULTURAL SERVICES INDUSTRY (SIC 07) AND FORESTRY (SIC 08), 1989—Continued

Name of industry group	SIC No.	No. of reporting firms	Average per firm, in $000s					Ratios for median firm		
			Total assets	Total liabilities	Net worth	Net sales	Net profit after tax	Current ratio	Return on sales	Return on assets
Timber tracts	0811	123	1,410.4	561.3	849.1	1,063.0	86.7	2.6	12.4	3.8
Forestry services	0851	127	240.8	112.2	138.6	880.0	40.8	1.7	0.5	7.3
Fishing, hunting and trapping [1]	0900	60	608.8	115.0	493.8	1,003.0	18.0	5.6	12.7	16.5

[1] Derived from Dun and Bradstreet, [10], [11]
Source: Meridian Research Inc., Table 2–12[1].

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2. Employee Exposure and Benefits

Introduction. Employee exposures to the substances included in the scope of this rule making are associated with a wide variety of acute and chronic conditions and illnesses. These include sensory irritation, narcosis, organ system dysfunction, chronic respiratory disease, neurological impairment, allergic sensitization, and cancer. OSHA believes that reducing worker exposures to such substances by applying limits where none existed previously will result in significantly reduced levels of exposure and disease. Section I–F of this preamble provides additional discussion of exposures in agriculture.

This chapter describes both the methodology used to identify workers potentially exposed to the hazardous substances included in this rule making and the expected benefits to those workers resulting from the application of permissible exposure limits to the agricultural sector.

Methodology. The diversity of agricultural work places, the large number of substances included in the proposed rule making, and the variety of operations and processes in the agricultural sector required Meridian, OSHA's contractor, to develop a methodology to permit the identification of (1) non-pesticidal hazards [2] regulated by OSHA that are of concern in the agricultural environment, (2) the number of workers employed on large farms exposed to these hazards, and (3) the magnitude of their exposures to the substances of concern.

Meridian, OSHA's contractor for this project, relied on the professional judgment of experts in the agricultural field to identify specific processes and operations widely recognized as presenting non-pesticidal toxic-substance exposure hazards to agricultural workers. Hazardous processes in the following industries were identified:

- Silo Operations
- Cotton Ginning
- Grain Handling (Post-Harvest)
- Grape Harvesting

For some operations, preliminary investigation revealed that the exposures of concern fell outside the scope of this project, either because the problem was the result of a pesticidal use of a toxic substance (as was the case with the mushroom industry), because it was related largely to a confined-space problem (as was the case with silo gases) or due to the fact that problematic exposures primarily occur on farms with fewer than 11 employees (as was the case with swine confinement and poultry operations [3] and many other agricultural operations). OSHA requests that any information on exposures not identified by this methodology be submitted to the public record of this standard for review.

Description of Data Sources Used. To derive a baseline of current exposures and to assess the quantitative benefits associated with reducing those exposures, the following data sources were used:

- OSHA compliance case files
- NIOSH Health Hazard Evaluations
- Agriculture extension agents in 10 of the major agricultural states.
- The Institute of Agricultural Medicine at the University of Iowa
- Agricultural economists at DPRA, Meridian's subcontractor
- The USDA library in Beltsville, MD
- OSHA's Integrated Management System (IMIS)
- Discussions with industrial hygienists at the Department of Labor and Industries, Washington State
- Discussions, observations, and exposure measurements from Meridian's site visits
- Discussions with experts at USDA's Research Unit and with individuals at the California Department of Food and Agriculture

[2] Currently the Environmental Protection Agency has jurisdiction over the application and use of pesticides pursuant to an EPA label. However, OSHA has authority over the non-pesticidal use of pesticide chemicals, use not permitted by the label or when farmers make their own pesticides. Benefits from these activities are not covered in this analysis.

[3] In a study by Morris, Lenhart and Service published in the February 1981 *American Journal of Industrial Medicine*, it was determined that 88 percent of chicken catchers reported at least one acute respirator symptom associated with work in poultry houses. Although these chicken catchers do not fall into the scope of the proposed standard, they are employed by general industry firms and are already subject to PELs, their exposures give an indication of respiratory problems occurring on all farms in the poultry confinement industry; an industry which is not analyzed in this study because no poultry confinement establishment is large enough to employ 11 or more workers.

Estimates of the Number of Potentially Exposed Employees

Silo Operations (SICs 0212, 0214, 0219, 024)

Exposure. Agricultural workers who work in and around silos are exposed to the dust that is generated by the movement of the silage during the loading and unloading of silos. In a study conducted by May[1], employee breathing zone samples for total and respirable dust were collected during five silo operations. The respirable dust exposures of employees working at three of the five silos ranged from 0.3 to 1.4 mg/m³, significantly less than the 5 mg/m³ PEL. In the fourth silo, two personal breathing zone samples showed respirable dust exposures of 10.1 and 10.5 mg/m³, both greater than the 5 mg/m³ PEL. At the fifth silo, the respirable dust exposures of two employees were measured as 24.6 and 40.3 mg/m³, respectively. Total dust measurements taken inside the chute using area sampling were less than 15 mg/m³ in three silos. In the fourth silo, total dust concentrations ranged from 13.5 to 25.2 mg/m³. Seven area samples for total dust were collected at the fifth silo, all of which exceeded the 15 mg/m³ PEL; these samples ranged from 86.8 to 154 mg/m³. These data indicate that some silo workers are exposed to respirable dust in excess of OSHA's 5 mg/m³ PEL for respirable dust and the 15 mg/m³ PEL for total dust; two fifths of the silo operations studied had respirable dust levels resulting in overexposure to all of their employees. Therefore, it is assumed that 40% of all persons employed at silo operations are overexposed to total and respirable dust. (Only two employees at any one time are needed for the silo operation; however, because hired farm hands participate in all aspects of a farm's activities, it was assumed that each farmhand will be involved in silo operations during their employment.)

Population at Risk. To estimate the number of silo workers potentially exposed to dust during silo unloading operations, sectors using silage as winter feed were identified. Based on observations and discussions during site visits, Meridian assumed that silos are present in both of the following industries: SIC 0212, Beef cattle, except feedlots and SIC 024, Dairy cattle. It was believed that silos are not likely to exist at cattle feedlots, poultry operations and hog farms because both use a high-calorie mixture of grains and other ingredients to achieve rapid weight gain in animals.

Exposures to Dust at Cattle Establishments. Data presented in chapter II indicate that there are roughly 1,700 beef cattle, except feedlot, establishments (SIC 0212) employing more than 10 workers and that these establishments have a total employment of 34,200 employees. No data are available to determine how many of these workers are actually exposed to dusts; it was assumed that all of these workers are potentially exposed to dusts during their employment.

Exposures to Dust at Dairy Farms. Meridian estimated that there are 700 large dairy farms employing 10,500 workers in the United States (see Table V–D1, chapter II). Although no data exist to indicate how many of these farms actually have silos, it is assumed that all of the establishments are likely to have silos and that all of the 10,500 employees at these establishments are potentially exposed to the hazards associated with silo operations.

Summation of Exposures for Silo Operations. Based on the estimates above, it is believed a total of 44,700 workers are exposed to silo-related hazards in 2,400 establishments that employ more than 10 workers. Due to personal exposure data reported by May[1] Meridian estimated that two fifths or 40 percent of these facilities and workers (960 and 17,800 respectively) are currently exposed above OSHA's 5 mg/m³ PEL for respirable dusts. It is likely that these workers are also exposed above OSHA's 15 mg/m³ PEL for total dust since it is unlikely that the respirable mass fraction of this dust represents a substantial fraction of the total dust mass.

Cotton Ginning (SIC 0724)

Exposure. Cotton ginning is seasonal and operations potentially expose workers to cotton dust, pesticide residue [4] and particles not otherwise classified (PNORs), i.e. nuisance dust. Two NIOSH publications describe employee exposures to these substances. One of these studies is a Health Hazard Evaluation describing three cotton gins in Arizona[2] in which cotton dust measurements and personal samples for total airborne particulates were taken. The study concluded that employees working at the gin stand and bale press were exposed above 1 mg/m³ for cotton dust (this is roughly equivalent to the 500 μg/m³ vertical elutriator [VE] PEL OSHA is proposing). In contrast, these data do not suggest that cotton gin employees are exposed above OSHA's 15 mg/m³ PEL for total dust (PNORs). In addition, NIOSH analyzed bulb cotton samples taken from various process locations for pesticide residues, including residues of the Table Z–1–A substances monocrotophos and methyl parathion. The analysis of bulb samples revealed detectable levels of monocrotophos and methyl parathion residues on the bulk samples; however, the data as reported do not permit an assessment of the levels of employee exposure to these substances.

The second publication concerned with cotton dust exposure is an extensive study of worker exposure to cotton dust during ginning operations. It was conducted by NIOSH[2] and included cotton dust measurements taken at 35 gins located in five different regions of the country. For each cotton gin, NIOSH reported the geometric mean and geometric standard deviation of cotton dust levels. These exposure profiles were analyzed by Meridian along with exposure distributions reported by Zey[3]. The latter studies findings suggest there is a 10-percent average probability that a cotton dust measurement will be found to exceed the 1 mg/m³ (500 μg/m³ VE) PEL for cotton dust.

Population at Risk. During the 1986–87 season there were 1,162 cotton gins in active operation[4]. According to Zey[3], a typical cotton gin has three or four gin stands and roughly 11 production workers. Meridian estimated that 12,782 workers (1162 gins x 11 estimated workers per gin) are potentially exposed to cotton dust, pesticide residues, and total dust (PNORs). In the study conducted by Zey, a 10-percent average probability that a cotton dust will be found to exceed the 1 mg/m³ PEL was estimated. Therefore, OSHA estimates that 10 percent of the estimated 12,782 cotton gin workers, or 1,278 employees are currently exposed above OSHA's proposed limit for cotton dust. Based on the exposure data reported in the NIOSH HHEs, it is believed that none of these employees are currently exposed above OSHA's 15 mg/m³ PEL for total dust(PNORs). As explained above, the data contained in these HHEs describing levels of pesticide residues is not suitable for estimating the pesticide levels to which employees are exposed. Hence, no estimate can be derived for pesticide residue exposure.

Post-harvest Grain Handling Operations

(SICs 011, 0711, 0721, 0722, 0723 [5]).

[4] The residues of pesticides listed on Table Z–1–A is not a situation covered under EPA's FIFRA and is therefore considered to fall within the scope of this study.

[5] It should be noted that SIC 0723 (crop preparations for market except for cotton ginning) is

Continued

Exposure. Most of the data describing employee exposures to dust during grain handling operations are relevant only when looking at large grain elevator operations, which are not within the scope of OSHA's present rule making (these elevators are classified in SICs 4221 and 5253 and were addressed by the 1989 Air Contaminants standard for general industry). There are no data that adequately describe the dust exposures of workers during grain handling operations at agricultural facilities classified in SIC 011 (Cash Grains). However, it is believed that these exposures can be approximated on the basis of data that describe the dust exposures of workers during the loading of trucks at small grain elevator facilities. Burg[5] studied such an operation and reported that the dust concentrations (as determined from personal air samples) were 15.3, 145, and 2,346 [6] mg/m³ during the loading of trucks with corn; because corn is not as dusty as wheat, oats and other grains it is believed that the exposure potential represented by corn dust somewhat underestimates dust exposures typically encountered by workers during operations that involve the transport and handling of wheat, oats and other grains usually found in storage bins located at agricultural facilities. It is estimated that grain handling often causes dust exposures that exceed OSHA's 10 mg/m³ PEL for grain dust (applicable to the handling of oats, wheat, and barley) and the 15 mg/m³ PEL for total dust (applicable to dust arising from all other types of grain).

Population at Risk. The loading and unloading of grain from storage bins is not generally a labor-intensive operation. Therefore, it is assumed that no more than one or two employees at each grain farm are involved in these operations and are thus exposed above the OSHA PELs for grain dust or total dust(PNORs); this would mean approximately 864 farm workers (average # of persons x 576 establishments) at the 576 grain farms estimated to have more than 10 employees (see Table V–D1). According to the 1986 Agricultural Statistics[6], the production of wheat, oats, and barley constituted 25 percent of total 1985 U.S. grain production; it is therefore believed that 216 or 25 percent of the estimated 864 workers engaged in grain handling operations are currently exposed above OSHA's 10 mg/m³ PEL for grain dust and that the remaining 75 percent (or 648) are currently exposed above OSHA's 15 mg/m³ PEL for total dust.

Grape Harvesting (SIC 0172)

Exposure. There is a potential for exposure to inorganic dusts, including free silica, which are released when foliage on the vine is disturbed during harvesting of grapes. After the spring rains, soil dust that is generated during the cultivation of the crop accumulates on the leaves of the plant; this accumulation continues throughout the drier summer until harvest time, when the dislodgeable portion of the dust is released into the air by the harvesting operation. The amount of dust released at harvest time depends on the specific crop and the local weather conditions prevailing at the time of harvest. Because grape crops require extensive cultivation regardless of where they are grown, workers harvesting grapes anywhere in the country are potentially exposed to airborne dust. In addition, because grapes are typically grown in areas having sillicacious soil (i.e., rocky or sandy soil), grape workers are potentially exposed to free silica.

In a California study by Popendorf and Spear[7], average personal total dust exposures of 13.5 mg/m³ for grape pickers were reported. Another study by Popendorf[8] indicated that a substantial fraction of the grape picker population is exposed to dust levels that exceed both OSHA's 15 mg/m³ PEL for total dust (PNORs) and its .1 mg/m³ PEL for respirable crystalline silica. Data from these studies can be seen in Tables V–D7 and V–D8.

Table V–D7.—Summary of Worker Exposures to Total Dust and Respirable Silica During Grape Harvesting

Crop	Number of total dust samples taken	Number of facilities surveyed for total dust	Percent of total dust samples exceeding 15 mg/m³	Percent of respirable dust samples exceeding .1 mg/m³ of free silica [a]
Grapes (SIC 0172)	80	10	75	60

[a] Based on the collection of 50 respirable dust samples at 19 facilities.
Source: Meridian Research Inc., Table 3–5[10].

Table V–D8.—Estimates of the Number of Workers Currently Exposed Above OSHA's PELs for Respirable Silica and Total Dust During Grape Harvesting

Crop	Estimated number of potentially exposed employees	Percent of workers exposed above total dust PEL [b]	Number of workers exposed above total dust PEL	Percent of workers exposed above respirable silica PEL	Number of workers exposed above respirable silica PEL
Grapes (SIC 0172)	8,873 [a]	75	6,655 (8873×.75)	60	5,324 (8873×.60)

[a] Total for the United States (excluding California).
[b] Data from Popendorf et al. (1982).
Source: Meridian Research Inc., Table 3–6[10].

covered by a settlement agreement between OSHA, the National Food and Grain Association (NFGA) and the AFL–CIO which retains the 10 mg/m³ limit but permits use of respirators in certain operations.

[6] Out of 74 observations, 2,346 was the highest recorded exposure, 513.1 and 176.1 were the next highest exposures.

Population at Risk. To estimate the size of the potentially exposed population, it was assumed that all grape pickers in the U.S. are conceivably exposed to total dust (PNORs), as well as to respirable free silica. According to the 1987 Census of Agriculture[9], grape farms (SIC 0172) constitute approximately 13 percent of all fruit and tree nut facilities in the United States. Assuming that the proportion of large fruit and tree nut establishments producing grapes is the same as that for all fruit and tree nut establishments, it is estimated that there are 1,495 large grape producing establishments (11,375×13 percent) employing 11,830 workers (91,000×13 percent).[7] No data on the proportion of the work force actually engaged in the harvesting of grapes were available; however, because most of these workers are likely to be involved in grape harvesting, it was assumed that 75 percent of the work force, or 8,873 workers, are potentially exposed to total dust and respirable silica during grape harvesting.

Estimates of the number of grape workers who are currently exposed above OSHA's PELs for respirable silica and total dust were derived from the employment estimates presented above and from the exposure data reported by Popendorf[8]. The derivation of these estimates is presented in Table V–D8 which shows that 6,665 workers (8,873×.75) are currently exposed above OSHA's PEL for total dust of 15 mg/m³ and 5,324 (8,873×.60) are exposed above the .1 mg/m³ PEL for respirable silica during the harvesting of grapes (Table V–D8).

Summary. Table V–D9 presents a summary of employees at risk of overexposure. There are an estimated 32,000 employees (some workers may be double counted) on 5,643 farms who are at risk of exposure to substances included in this rule making.

TABLE V–D9.—SUMMARY OF EMPLOYEES AT RISK OF OVEREXPOSURE

Activity	Industry	Substances	No. of establishments	Estimated population at risk
Silo Operations	SIC 0212: Beef cattle, except feedlots	Respirable dust (5 mg/m³)	2,410	17,880
	SIC 024: Dairy cattle	Total particulate (15 mg/m³)		
Ginning	SIC 0724: Cotton ginning	Cotton dust (500 μ/m³	1,162	1,278
Loading/Unloading Grain	SIC 011: Cash grains	Grain dust (10 mg/m³)	576	216
		Total particulate (15 mg/m³)		648
Harvesting Grapes	SIC 0172: Grape harvesting	Total particulate (15 mg/m³)	1,495	6,655
		Respirable silica (1 mg/m³)		5,324
Total	5,643	32,001		

Employee Benefits. In addition to estimating the number of employees exposed to the substances in this analysis, OSHA also estimated the number of employees who are at risk of experiencing particular types of adverse health effects. To conduct this analysis, each substance in which overexposure was determined, was assigned to a health hazard category; these assignments were based on the primary health effects expected from reducing a previous limit or establishing a new limit for a particular substance. In the benefit analysis described below, substances were classified according to their primary health effect and the operations in which they were present. When direct health effects evidence was not available, analogous chemicals were used to develop benefit estimates. OSHA believes that farm workers may be exposed to other air contaminants, such as CO_2, solvents and fertilizers, which were not identified in this analysis due to the limited data available concerning agricultural exposures. As a result, benefits for the reduction of these exposures were not quantified even though OSHA believes some unspecifiable benefits will result.

OSHA's contractor, Meridian Research Inc., developed an approach that relied on substance-specific toxicologic information that quantitatively related exposure to prevalence of disease for the specific substances of interest. It did not rely on general statistical data describing the illness mortality and morbidity experience of agricultural workers for two principal reasons. First, general illness mortality and morbidity rates reflect disease risks that are associated with exposure to a number of substances that are not generally covered by this rule making action, notably pesticides, herbicides, insecticides, fungicides, defoliants and disinfectants. (It should be noted however, that activities where a pesticide was misused according to its EPA label or where farmers make their own pesticides are instances covered by OSHA. OSHA has not been able to quantify benefits from the regulation of these instances but believes benefits will result in the form of some reduction in illnesses.) Second, such statistics reflect the health experience of farmers who work on small farms (i.e. those with 10 or fewer employees), which together employ most of the agricultural workers in the United States. In order to avoid the problem of using under-representative data, a substance-specific approach using toxicologic information relating quantitative exposure to prevalence was adopted. Substances, and the agricultural operations in which occupational exposures have been documented, include exposure to: silage dust during silo unloading operations; respirable silica during grape harvesting; grain dust during grain handling operations; and cotton dust during cotton ginning operations.

Silage Dust

The principal health hazard associated with excessive exposure to silage dust is a condition known as organic toxic dust syndrome, an illness in which the affected individual suffers fever, malaise, headaches, and chest tightness[3]. In a study conducted by May[3], exposures to silage dust generated by unloading operations in five vertical silos were analyzed and found to exceed OSHA's 5 mg/m³ PEL at one of the five silos. Assuming the survey is representative of current industry conditions, such exposures may be expected to occur among roughly 20

[7] These calculations exclude California. Currently, CAL–OSHA enforces PELs in agriculture which are stricter than OSHA's proposed PELS. For this reason, Californian establishments and employees were excluded from the population-at-risk estimate.

percent [8] of the workers engaged in silo unloading operation with sufficient magnitude (more than 5 times the PEL) to cause symptoms of organic toxic dust syndrome at least once during the silo unloading season.

There is no evidence that organic toxic dust syndrome is likely to occur when exposures to respirable dust conditions are maintained at or below the 5 mg/m³ PEL[3]. Therefore, it is estimated that compliance with OSHA's 5 mg/m³ will result in the prevention of at least 8,940 cases of organic toxic dust syndrome (44,700 silo workers × 20 percent) per year. Based on the severity of symptoms caused by organic toxic dust syndrome (OTDS), OSHA estimates that 25% of all OTDS cases (roughly 2,235 cases) will result in lost workdays.

Respirable Silica

Exposure to respirable dusts is known to cause silicosis, a fibrotic lung disease that can severely compromise pulmonary function. In a study conducted by Popendorf in 1982[8], it was determined that respirable silica exposure frequently exceeds OSHA's 0.1 mg/m³ PEL, usually averaging 0.15 mg/m³ during grape harvesting. Calculating the lifetime cumulative-silica exposure of the worker populations, Meridian estimated that about two percent of grape harvesters who are overexposed to respirable silica are likely to develop reduced pulmonary function as a consequence of exposure. This corresponds to a population of 106 grape harvesters (2% of the 5,324 workers estimated to be overexposed to respirable silica) at risk of acquiring silicosis. Meridian states in its report that through the use of approved dust masks having a protection factor of 5 (see Chapter VI), the risk of acquiring silicosis will be reduced to less than one percent. (Meridian used a methodology where average silica exposures were translated into the number of dust years exposed in order to define the current incidence level. Using the formula:

$$\text{Number of dust-years exposed}^{9} = \frac{\text{Average Silica Exposure}}{0.05\ \text{mg/mg}^3} \times \frac{\text{No. days/year exposed}}{365}$$

And assuming grape harvesters work an average of about 20 years and that these workers are exposed to respirable silica over an average of 40 days per year during grape harvest, Meridian calculated that grape harvesters who are currently exposed above OSHA's PEL for respirable silica accumulate approximately 6.6 dust-years of exposure to silica over their working lifetime. Applying this information to a dose response curve[11] between granite dust and quartz [both silicacious substances] versus pulmonary forced vital capacity, Meridian was able to determine that two percent or 106 harvesters over a 20 year working life were at risk of acquiring silicosis in grape harvesting.) Therefore, it is estimated that compliance with the 0.1 mg/m³ PEL will prevent pulmonary function reduction in approximately 106 grape harvesters over a 20-year working life (an average of 6 cases avoided per year). Based on the severity of symptoms caused by silicosis (silicosis can result in death), OSHA estimates that all of the cases will result in lost workdays.

Grain Dust

The respiratory and other effects of exposure to grain dust are varied and include both acute and chronic abnormalities. Workers exposed to grain dust frequently experience symptoms of cough, expectoration, wheezing and chest tightness; these symptoms may result from either an acute exposure or may reflect the development of chronic bronchitis from prolonged exposure. In addition, grain workers experience episodes of "grain fever", which occurs during or several hours after exposure.

As a result of a study conducted by Rankin[12], Meridian determined that workers exposed in excess of OSHA's 10 mg/m³ PEL for grain dust during the unloading of storage bins containing oats, wheat and barley are likely to experience adverse respiratory symptoms (Table V–D10). Because the workers are generally exposed to levels exceeding 15 mg/m³, it was assumed that the prevalence of respiratory symptoms in this group parallels that of the highest group studied by Rankin where prevalence for cough, expectoration, dyspnea and eye irritation was 89, 67, 44 and 44 percent, respectively. Assuming the prevalence for cough, expectoration, dyspnea and eye irritation in grain workers is the same as the group studied by Rankin it is believed that as a result of complying with OSHA's 10 mg/m³ PEL for grain dust and 15 mg/m³ for total dust, the prevalence of respiratory symptoms among these workers would fall to the same levels experienced by Rankin's 5–10 mg/m³ and 10–15 mg/m³ exposure group.[10] Hence, the anticipated reduction for cough, expectoration, dyspnea and eye irritation is expected to be 49; 14; 3; and 11 percent for those over exposed to grain dust and 22; 0; 0 and 0 percent for those over exposed to total dust. After applying these reductions in symptoms to the estimated 216 farm workers (Chapter 3) who are currently overexposed to grain dust and the 648 over exposed to total dust, it is estimated that coughing, expectoration, difficulties in breathing, and eye irritation will be avoided in 236, 30, 80, and 24 workers respectively. Based on the mild severity of the symptoms caused from over exposure to grain dust, it is estimated that none of these cases will result in lost workdays.

[8] While 40 percent or two fifths of all employees at silo operations are overexposed, 20 percent of all employees are exposed at a level (roughly 5 times the PEL) high enough to cause organic toxic dust syndrome.

[9] This formula relies on the concept of dust-years of exposure defined by Theriault, Buress, DiBernardini, et al. in their 1974 study and by Peters in his 1986 paper.

[10] The 5–10 mg/m³ group had roughly the same prevalence rates as the 0–5 mg/m³ group which is not presented in table V–D10. Thus, although it is probable that exposures will be lowered below 5 mg/m³ due to the use of respirators, there will be no further decline in prevalence.

TABLE V-D10.—SUMMARY OF THE PREVALENCE OF RESPIRATORY SYMPTOMS EXPERIENCED BY GRAIN DUST WORKERS, BY EXPOSURE LEVEL

Range of exposure level (mg/m³)	Prevalence of respiratory symptoms (percent of workers studied)*			
	Cough	Expectoration	Dyspnea	Eye irritation
5–10	40	53	7	13
10–15	67	67	50	50
>15	[illegible]	67	44	44

* Data reported only for those symptoms for which the prevalence in the high exposure group was significantly different from that in an unexposed control group.

Source: Meridian Research Inc. Table 5-3[10].

Cotton Dust

Byssinosis is the general term applied to acute and chronic respiratory disease caused by excessive exposure to textile vegetable dusts, which include cotton, flax and hemp fibers. As the disease worsens, affected individuals experience chest tightness and/or breathlessness both on return to work and on subsequent days and show decrements in pulmonary flow that become progressively more pronounced; at this stage, a clear picture of chronic obstructive lung disease can be seen. In a study conducted by Zey among 521 non-smoking cotton gin workers employed in 37 gins [3], an exposure-related increase in the prevalence of chronic bronchitis symptoms and textile vegetable dust disorders was discovered. Using regression analysis, Meridian constructed a mathematical expression relating exposure to prevalence of these disorders.

To estimate the potential benefits associated with promulgation of the 500 $\mu g/m^3$ VE cotton dust PEL in cotton gins, Meridian assumed that the 1,278 workers who are overexposed to cotton dust are exposed to an average TWA concentration of 1.5 mg/m^3 (overexposures generally occur in the range of 1.1 to 2.0). Meridian also assumed that cotton gin workers work an average of 20 years in gins. Using the exposure-response relationships, (Exhibit 5-4 [10]) Meridian estimated that the current prevalences of chronic bronchitis and textile vegetable dust disorders among the 1,278 workers estimated to be overexposed are 28 and 43 percent respectively. By reducing these workers exposures to the 500 $\mu g/m^3$ VE PEL, OSHA estimates prevalence will be reduced to 17 percent for chronic bronchitis (i.e. a reduction of 11 percent) and 24 percent for textile vegetable dust disorders (i.e. a reduction of 19 percent).[11] This analysis suggests that the promulgation of the 500 $\mu g/m^3$ PEL for cotton dust in cotton ginning operations will prevent symptoms of chronic bronchitis from developing in 141 workers (1,278×11 percent) and symptoms of textile vegetable dust disorders in 243 workers (1,278×19 percent) over a 20 year working life. Hence, promulgation of the final rule will prevent chronic bronchitis from developing in approximately 7 workers (141/20), and textile vegetable dust disorders in roughly 12 workers (243/20) each year. Chronic bronchitis is a potentially crippling disease, greatly reducing the quality of life and the productivity of its victims, OSHA estimates that all cases of chronic bronchitis will result in lost workdays. No cases of textile vegetable dust disorders are expected to result in lost workdays.

Summary of Benefits and Exposure. As is evident, agricultural workers are exposed to a variety of air contaminants in a number of different operations. It is estimated that over 17,800 workers are overexposed to total dust as well as respirable dust in silo operations; 1,278 employees are currently overexposed to cotton dust in cotton gins; 216 workers are exposed above OSHA's PEL for grain dust and 648 are exposed above OSHA's PEL for total dust during post-harvest grain operations; 5,324 grape harvesters are overexposed to respirable silica; and 6,655 workers are overexposed to total dust during grape harvesting.

The benefits presented in this chapter show that the proposed rule is necessary and that it will provide quantifiable benefits to the agricultural community. As a result of the promulgation of this rule, 8,940 annual occurrences of febrile organic toxic dust syndrome will be prevented; 5 silica-induced pulmonary function declines will be eliminated each year; acute respiratory symptoms occurring in 24 to 106 workers will be avoided annually; and 7 cases of chronic bronchitis as well as 12 cases of textile vegetable dust disorders will be prevented annually. OSHA estimates that 2,248 (24%) of these cases will result in lost workdays while 7,090 (76%) will be non-lost workday cases.

[11] This reduction is based on data from the American Textile Manufactures Institute (ATMI) which showed that after dust control and medical surveillance were initiated, the prevalence rate for byssinosis dropped to less than 1.0% and the prevalence rate for bronchitis ranged from 6-7%. OSHA believes that through the use of the controls outlined, reductions approximate to half of these can be achieved since a medical surveillance program will not be required. [13]

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3. Assessment of Nonregulatory Alternatives

For a full discussion on the need for and the appropriateness of a regulatory solution to address occupational exposures to air contaminants, see section V.B.4. of this preamble.

4. Technological Feasibility

Introduction. This chapter presents a technological feasibility analysis of the agricultural sector's ability to meet OSHA's proposed permissible exposure limits (PELs) for a wide range of occupational health hazards. These PELs include limits on airborne concentrations of substances, and in some cases, direct contact on the skin with the substance.

The control of workplace exposures to toxic chemicals involves combining a variety of standard techniques to solve a situation-specific problem. For a variety of situations where air contaminants are encountered by workers in agriculture, compliance can be achieved by applying known engineering control methods. In addition, work practice improvements and personnel protective equipment usage may also be required.

This chapter examines the feasibility of engineering controls, work practices and personal protective equipment needed to reduce workers' exposure levels to total dust, respirable dust, cotton dust and respirable silica.

Types of Controls

Engineering Controls. Engineering controls involve the use of local exhaust ventilation, general ventilation, isolation of the worker, enclosure of the source of emissions, process modifications, equipment modifications, and substitution of non-hazardous chemicals. These methods may be used alone or in combination of any two or more controls depending upon the needs of a specific situation. Variations in situations usually result from the type of process being used and the number of chemicals in the air. However, these controls are considered standard techniques which will effectively control these variables either by themselves, or coupled with changes in work practices.

Ventilation. Perhaps the most widely used technique for controlling chemical exposures is the use of ventilation. General ventilation uses the movement of air within the general work space to displace or dilute the contaminant with fresh outside air. General ventilation is not typically the preferred control method in most operations due to the large volumes of air movement required. Local exhaust ventilation uses much smaller volumes of air, exhausted from the point at which contaminants are generated to remove the contaminant at the source.

Work Practices. Work practice controls include housekeeping procedures, material handling procedures, training and personal hygiene. In many cases, it is possible to bring about substantial reductions in employee exposures by applying work practice controls. A work practice control could be as simple as keeping a distance from a source of emission.

Personal Protective Equipment. Where it is impractical to apply engineering or work practice controls, or where their application will not consistently reduce employee exposures below the proposed PELs, personal protective equipment such as respirators, may be required in order to prevent or reduce exposures.

Feasibility. This chapter presents examples of feasible methods for controlling exposures to hazardous substances encountered in processes used in the SICs for which costs (Chapter VI) and benefits (Chapter 3) have been identified. Unit costs for these controls are used as the basis for the cost projections in Chapter 5.

Feasibility determination is made for the following operations which have been identified as having activities which result in overexposure to certain hazardous substances:

- Silo operations
- Cotton ginning
- Post-harvest grain handling operations
- Grape harvesting

Silo Operations. Employee exposures to silo dust occur when workers unload silage to feed animals during the winter months. These exposures result from dust generated by silage falling down the silo chute into a truck or onto a conveyor for subsequent transport to the animal feed area. Dust exposure may also occur when employees climb up the chute and enter the silo to maintain or repair the silo's automatic loading device.

The exposure data presented by May[1] and described in Chapter III indicate that employee exposures to dust exceed the 5 mg/m^3 PEL for respirable dust about 40 percent of the time during silo operations. It is believed, by OSHA's contractor, that the 15 mg/m^3 PEL for total dust is also exceeded at these times.

Feasibility Assessment. At present, no controls are in place in silos to reduce worker exposures to dust. The implementation of a simple work practice, such as training workers to stand upwind during unloading, would reduce dust exposures to some extent, although the percentage of reduction achievable by this approach is not known. Wetting, a commonly employed dust suppression technique, cannot be used with silage because wetting would interfere with the flow of silage during unloading and would also promote spoilage. Ventilation, another common technique for controlling airborne particles, and the only engineering control applicable to this situation, cannot be configured for such a large area and could even create more of an exposure problem in this particular environment. Reliance on disposable dust respirators is seen as a practical approach to achieve compliance with the PELs for respirable and total dust during silo operations.

Cotton Ginning

Feasibility Assessment. Based on NIOSH's extensive 35-gin survey[2], 86 percent of all gins are already achieving mean employee cotton dust exposures at or below the 500 $\mu g/m^3$ level most of the time. These data demonstrate that compliance with the 500 $\mu g/m^3$ cotton dust PEL is technologically feasible for the cotton ginning industry and is already being achieved by most ginning operations at the present time. Nevertheless, it was estimated in Chapter III that 10 percent of the ginning workforce, or 1,278 employees, continue to be exposed above the PEL. The exposures of most of these workers are within a factor of 2 of the PEL; in a few rare cases, however, overexposures may exceed the PEL by a factor of 6[3].

Based on these data, there is no issue of technological feasibility for facilities in this sector, since the overwhelming majority of ginning operations are already well below the cotton dust PEL. For those few facilities still above the PEL, OSHA's contractor, Meridian, concluded the implementation of simple housekeeping measures will achieve the 1 mg/m^3 PEL. Meridian's reasoning was based on the following: The exposure data presented by Zey[4] and described in Chapter III strongly suggest that employee exposures to cotton dust can

be maintained below 1 mg/m³ by applying consistent work practices[4]. The authors report that "The accumulated thrash inside the plant[s] [is] inevitably a secondary source of airborne dust"[4]. Second, NIOSH reports that the dust raised during the dry sweeping of facilities is responsible for the dustiest conditions in these gins[2]. And lastly, the Zey[2] data show that at least one of the readings taken in half of the 35 gins examined were above 1 mg/m³; however, these high readings were infrequent, occurring only in about 10 percent of the cases overall. It is Meridian's opinion that this pattern of infrequent high particulate exposures is consistent with the failure of work practice controls rather than with inadequate engineering controls (which would be reflected in plant-wide high readings). In other words, NIOSH's finding that employee exposures to cotton dust exceed 1 mg/m³ on an infrequent basis in many cotton gins suggests that work practices, such as the regular cleaning of work areas and equipment, are being applied in an inconsistent manner or are being performed improperly by those facilities having high readings.

Post-Harvest Grain Handling Operations. Employee exposures to grain dust during post-harvest grain handling occur during the transfer of grain from storage bins to trucks[5], and the exposure data presented in Chapter III show that these exposures range from 15.3 to 2,346 [12] mg/m³. These data were derived from personal samples taken on the operator, who was standing on top of a truck directing the flow of grain from a chute that dropped grain into the truck from a distance of several feet. The actual 8-hour TWA exposure of the employee sampled by Burg[5] is difficult to gauge because the duration of the sampling and the employee's pattern of exposure were not described in the Berg article. Because this employee was not reported to be wearing respiratory equipment during the unloading operation, it is believed that the employee's exposure did not exceed a few hundred mg/m³ as an 8-hour TWA; exposures higher than approximately 200 mg/m³, if sustained over a full workday, would cause considerable discomfort in the absence of respiratory protection. Therefore, it is believed the 8-hour TWA dust exposures of workers during bin unloading operations are unlikely to exceed about 200 mg/m³.

Feasibility Assessment. Meridian's final report states that compliance with OSHA's 15 mg/m³ 8-hour TWA PEL for total dust and with OSHA's 10mg/m3 PEL for grain dust (in those instances where oats, wheat, or barley are being handled) can be achieved by the use of full-facepiece respirators equipped with high-efficiency particulate (HEPA) filters. The use of such respiratory equipment, which affords a protection factor of 50[6], will provide sufficient protection where 8-hour TWA exposures to grain dust or to total dust do not exceed 500 mg/m³ or 750 mg/m³, respectively. In those rare instances where exposures exceed these values, it is believed that the observance of some simple work practices, such as lowering the loading chute to reduce the drop distance or having the operator stand away from the truck during the initial loading period, will reduce operator exposures to within a factor of 50 of the respective total dust and grain dust PELs.

Grape Harvesting

Feasibility Assessment. Unlike the harvesting of grain and other crops, the harvesting of grapes is primarily a manual activity. Workers are potentially exposed to total dust and respirable silica in these operations at levels in excess of OSHA's PELs for these substances (see Chapter III). Some reduction in dust exposures could be achieved in instances where it is possible to wet the fields and vines prior to picking; however, this procedure is not feasible in the majority of cases because crop spoilage increases if the fruit is picked and packed wet. Therefore, it is believed that the owners of these farms will rely on respiratory protection (NIOSH approved dust masks) to reduce their employees' exposures to total dust to the 15 mg/m³ PEL and to respirable silica to the 1.0 mg/m³ PEL.

Engineering controls such as ventilation, oil conveyors and pneumatic dust control mechanisms have proven effective in controlling grain dust in other sectors involving grain handling. It is possible that these controls could be used for post-harvest grain handling. However, OSHA's contractor, Meridian, has reviewed some dust control systems and found that they may not be effective in the open areas post-harvest grain handling takes place in and may even cause additional problems by increasing the level of grain dust in the air. As a result, Meridian has recommended to OSHA that some workers may be required to wear respirators some of the time as part of their duties.

Summary. Based upon the analysis in this chapter and the supporting documentation, OSHA believes that controlling exposures on large agricultural establishments to substances found on the Z–Table is technologically feasible through the use of engineering controls, work practices and personal protective equipment. OSHA believes that these controls will be effective in reducing routine exposure to the substances posing a threat to worker health on these agricultural establishments.

[12] Out of 74 observations, 2,346 was the highest recorded exposure, 613.1 and 170.1 were the next highest exposures.

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5. Costs of Compliance

Introduction. Costs are related to recommended engineering controls, work practices and personal protective equipment needed for specific processes in order to maintain workers' exposures at or below the levels specified in the proposed standard. Given the large number of substances being regulated, an assessment was required that would examine a large number of operations over the entire agricultural sector.

Work practice requirements were identified by industrial hygienists based on their experience and research on operations and exposure situations in each operation. Also, included in the cost calculations are costs for all equipment required for exposure control. These latter costs were developed on a per person basis. Capital costs for HEPA vacuums and full facepiece respirators were annualized over 5 years using a 10 percent cost of capital and adding annual operating and maintenance costs estimated at 10 percent of the capital cost.

An annual cost was developed for each operation. These costs are

presented in Table V–D11. As shown, annual compliance costs are estimated to total $2.7 million. These costs represent an estimate of compliance costs for large operations affected by the PEL extension. Operations (identified in chapter III) which have the potential for worker overexposure and will incur costs include:

- Silo operations;
- Cotton ginning;
- Post-harvest grain handling operations; and
- Grape harvesting

This chapter summarizes for each process: (1) The extent of current worker exposure and the current baseline level of controls, (2) the additional controls that will be needed to reduce worker overexposures, and (3) the costs that facilities in the agricultural sector will incur to achieve compliance with the PELs for the airborne contaminants of concern.

TABLE V–D11.—ANNUALIZED COSTS OF COMPLIANCE

Operation	SIC	Cost per facility	No. of facilities	Annual cost
Silo operations	0212, 024, 0214, 0219	$270	964	$260,280
Cotton ginning	0274	3,334	581	1,937,054
Post-harvest grain handling	011, 0711, 0721, 0722, 0723	155	576	88,992
Grape harvesting	0172	267	1,495	398,300
Total			3,616	2,585,526

Numbers may not compute due to rounding.

Costs—Silo Operations (SICs 0212, 024, 0214, 0219). The exposure data described by May[1] indicate that the dust exposures of employees involved in silo unloading exceed the 5 mg/m³ PEL for respirable dust in two of five (or 40 percent) of the facilities having silos. In Chapter III, it is estimated that silo unloading operations are conducted at approximately 2,400 agricultural facilities that employ more than 10 workers. Therefore, employee overexposures to dust during silo unloading currently occur in an estimated 964 (2,410×0.4) agricultural facilities. It is believed that, at most, two employees per facility are engaged in silo unloading operations on any given day during the approximately 90-workday winter feeding season.

Current Baseline Controls. Meridian derived its data for baseline controls from observations made during site visits and discussions with representatives of agricultural extension services and the University of Iowa Institute of Agricultural Medicine and Occupational Health. Currently, no controls are used to reduce exposure to respirable dust in this process.

Estimated Costs of Controls. Assuming that each such facility will purchase 180 disposable respirators (2 employees×90 workdays), at a unit cost of $1.59[2], the estimated annual cost per facility to achieve the respirable and total dust PELs for silo unloading operations is $270. Meridian estimates that the total annual cost for all affected facilities will be $260,280 ($270 per facility×964 facilities).

Cotton Ginning (0724). Based on NIOSH's survey of 35-gin operations[3], 86 percent of all gins are already achieving mean employee cotton dust exposures at or below the 500 μg/m³ level most of the time. However, Meridian estimated (Chapter III of its final report) that 10 percent of the ginning workforce, or 1,278 employees, continue to be exposed above the PEL. The exposures of most of these workers are within a factor of 2 of the PEL; in a few rare cases overexposures may exceed the PEL by a factor of 6.

Current Baseline Controls. An industrial hygiene/medical evaluation conducted by NIOSH from October 1980 through February 1981 dealt with three cotton gins on the Colorado River Reservation in Arizona[3]. The three gins studied in this investigation had moderately effective to no engineering controls for reducing airborne concentrations of cotton dust. One gin was processing first- and second-picked cotton at the time of the evaluation and had some local exhaust ventilation equipment to help control dust. Another gin was processing only first-picked cotton and had no exhaust ventilation equipment. The third gin was processing rood or last-picked cotton and also had no exhaust ventilation equipment.

Reduction of worker exposure to dust that results from the processing of cotton is being achieved in some facilities by the use of engineering controls[4]. These include the isolation of open processes and specific dust generation points with shutters to prevent contamination of other areas; use of conveyers and material transfer points; location of exhaust systems and air cleaner discharge points downwind and at a distance from building air inlets; and hooding.

Work practices are also used to reduce cotton dust levels. Examples of such practices include routine maintenance of local exhaust hoods, ductwork, dust collection equipment, and fans so that such equipment works at maximum efficiency and effectiveness; good housekeeping activities that are performed systematically and on a regular basis (e.g., vacuum cleaning rather than dry sweeping); and common-sense procedures, such as regular laundering of work clothes and keeping cotton away from the face.

Respiratory protection is also used during dusty activities (e.g. the use of compressed air cleaning in "blow down" operations). During NIOSH's multi-plant survey, personal protective equipment in evidence were hard hats and disposable paper respirators, which were (not NIOSH-approved) worn by about 10 percent of the employees in the survey.

Estimated Costs of Compliance. The exposure data presented in Chapter III from the Zey report[4] suggest that 50 percent of the estimated 1,162 cotton gins in the United States (or 581 facilities) must implement additional exposure control procedures to achieve the 500 μg/m³ PEL for cotton dust. As described in Chapter V, it is believed that the 500 μg/m³ PEL can be achieved in these facilities by improving housekeeping practices, and in

particular, by cleaning the ginning work area and equipment at the end of each shift. In facilities currently performing end-of-shift cleanup, all employees spend 30 minutes performing this activity before going off-shift[4]. Meridian believes that effective cleanup will require the use of HEPA-filtered vacuums rather than the dry sweeping or use of compressed air that is likely to be the method of choice in these plants at present. This recommendation is consistent with NIOSH's recommendation[4].

According to NIOSH[4], cotton gins employ 10 to 18 workers (or an average of 14 workers), and these workers work on two 12-hour shifts. It is therefore assumed that, on average, seven employees will be available per shift to perform cleanup activities. Assuming that a typical cotton gin operates 7 days per week over an 8-week season[4], it is estimated that a total of 392 hours per facility per year will be required to perform post-shift cleanup activities (7 employees/shift×0.5 hour/shift×2 shifts/day×56 days). At an average labor cost of $6/hour[5], the estimated annual per-facility labor cost for end-of-shift cleanup is $2,352.

In addition, it is assumed that each of the 581 cotton gins estimated to need this kind of routine cleanup will purchase three HEPA-filtered vacuums at an estimated capital cost of $2,700[2] to use for post-shift cleanup. Assuming a useful life of 5 years, a 10-percent interest rate, and an annual maintenance cost of 10 percent of capital cost, an annualized cost for HEPA-filtered vacuum equipment is estimated to be $982 for each affected facility. Thus, the total annualized cost, including labor costs for cleanup, estimated to be incurred by each affected facility to achieve compliance with the 500 $\mu g/m^3$ cotton dust PEL is $3,334 ($982+$2,352). The total annualized cost of compliance for this sector as a whole is estimated to be $1,937,054 ($3,334×581 facilities).

Post-Harvest Grain Handling Operations (SICs 011, 0721, 0722, 0723)

Current Baseline Controls. Employee exposures to grain dust during post-harvest grain handling occur when grain is transferred from storage bins to trucks[6]. The exposure data presented in Chapter III show that these exposures range from 15.3 to 2,346 mg/m^3. Currently, it is believed that no baseline controls are used to reduce employee exposures to OSHA's 10 mg/m^3 PEL for grain dust.

Estimated Costs of Compliance. In Meridian's final report, it is estimated that 864 employees on 576 grain farms employing more than 10 workers are overexposed to either grain dust (i.e., oats, wheat, or barley) or total dust as a result of unloading grain from storage bins (see Chapter III). The unloading of storage bins does not occur year-round; instead, bins are unloaded when the demand and price of grain are sufficiently high to warrant the shipment of grain to market. Consequently, the number of days on which workers are exposed to dust during this operation varies from facility to facility and from year to year. For costing purposes, it is assumed that employees are overexposed an average of 30 days per year and that every employee will be provided with a NIOSH/MSHA-approved full-facepiece respirator at an estimated cost of $165 apiece[2]. Assuming a 5-year life expectancy for these masks and a 10-percent interest rate, the annualized cost for each respirator is estimated to be $43. In addition, every employee will have to be furnished with a new pair of HEPA filters, at a cost of $8.00 per pair[2], every 4 days that unloading is performed. Assuming that employees perform this task on 30 days per year, the annual cost for HEPA filters is estimated to be $60 per employee ([30 days/4]×$8.00 pair). The total annualized per-employee cost of compliance is therefore estimated to be $103 ($43 and $60). The industry wide annualized cost to provide this equipment to all 864 employees believed to be overexposed during grain unloading operations is estimated to be $88,962.

Grape Harvesting (SIC 0172)

Estimated Exposure. For the grape industry sector, data presented in Chapter III suggest that 8,873 workers are potentially exposed to respirable silica and total dust during the manual harvesting of grapes. Of these, 5,324 workers are estimated to be overexposed at present to both respirable silica and total dust. In addition, 6,655 employees are estimated to be overexposed above the 15 mg/m^3 PEL for total dust alone; this yields a maximum total of 6,655 overexposed workers in this sector (see Table V–D9).

Current Baseline Controls. Meridian found no controls being used to control exposure to respirable silica and total dust during the manual harvesting of grapes.

Estimated Costs of Compliance. Based upon available exposure data[7], no grape harvest worker is currently exposed to a concentration of respirable silica or of total dust that is greater than five times the respective PEL; therefore, employers will be able to achieve compliance both with the respirable silica and total dust PELs by ensuring that their employees wear disposable respirators, which have a protection factor of five[8]. Disposable respirators that are NIOSH/MSHA-approved for use against silica and total dust are available at a cost of $1.50 apiece[2–p. 99].

The grape harvest occurs in September and early October[7] and therefore, it is estimated that grape harvesters will need respirators over a period of about 40 work-days. The resulting cost per worker is estimated to be about $60 per year (40 days×$1.50/day). OSHA estimates that the total annualized costs that employers will incur to comply with the PELs for respirable silica and total dust in grape and harvesting operations is $399,300 ($60 per year per worker×6,655 workers exposed to silica and/or total dust).

Summary. Table V–D11 presents the estimated average cost of compliance per farm. Annual costs range from $155 for post harvest grain handling, up to $3,334 for cotton ginning operations. Total annual costs approach two million dollars for cotton ginning. The total annual cost for all affected farm operations is $2.7 million.

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6. Economic Impact, Regulatory Flexibility Analysis, and Environmental Impact Assessment

Introduction. This chapter investigates the potential economic impact on affected establishments as a result of the proposed standard. OSHA analyzed two alternative scenarios of the potential impacts of the proposed rulemaking. In the first, a total cost absorption or no cost passthrough scenario was assumed (all costs are borne entirely by affected establishments). In the second scenario, total cost passthrough was assumed (all compliance costs are passed on in the form of price increases). In this scenario, estimates of the maximum inflationary impact were calculated. These impacts were estimated using compliance costs developed in Chapter VI.

Economic Impact. In the first scenario, perfectly elastic demand or zero cost-passthrough was assumed. The estimated economic impact of the rule for firms potentially affected is summarized in Table V–D12. These estimates represent the maximum industry impact within a market scenario where none of the costs can be passed on to consumers and where there is no productivity offset to costs.

Table V–D12 shows that costs as a percentage of profits average .33%, with the highest impact in cotton ginning where costs as a percentage of profits is 1.4%. These results, however, represent a worst case, with the magnitude of the standard on profitability somewhat exaggerated because full absorption of the costs are not likely to occur. It should be noted that costs of this magnitude cannot affect the viability of even the smallest gins in this segment.

TABLE V–D12.—ECONOMIC EFFECTS: NO COST PASSTHROUGH SCENARIO

SIC	Industry	Annual cost per establishment	Average profit per [1] establishment	Costs as a percent of profit
011	Cash Grains	$155	$84,144	.18
0172	Grapes	267	270,301	.10
0212	Beef Cattle Except Feedlots	270	49,078	.55
0241	Dairy Farms	270	172,390	.16
0721	Crop Planting, Cultivating, Protecting	155	267,742	.06
0722	Crop Harvesting, Primarily by Machine	155	95,704	.16
0723	Crop Preparation Services for Market Except Cotton Ginning	155	325,580	.05
0724	Cotton Ginning	3,334	232,367	1.43

[1] Average profit before tax, Dun and Bradstreet, Dun's Insight Database, 1989.

TABLE V–D13.—ECONOMIC EFFECTS: NO COST PASSTHROUGH SCENARIO

SIC	Industry	Annual cost per establishment	Average sales per [1] establishment	Costs as a percent of sales
011	Cash Grains	$155	$1,083,682	0.01
0172	Grapes	267	3,329,803	0.01
0212	Beef Cattle Except Feedlots	270	604,599	0.04
0241	Dairy Farms	270	1,765,466	0.02
0721	Crop Planting, Cultivating, Protecting	155	3,298,281	0.00
0722	Crop Harvesting, Primarily by Machine	155	1,402,565	0.01
0723	Crop Preparation Services for Market Except Cotton Ginning	155	2,555,783	0.01
0724	Cotton Ginning	3,334	1,779,383	0.19

[1] Dun and Bradstreet, Dun's Insight Database.

In the second scenario, total cost pass-through was assumed. As demonstrated by the estimates summarized in Table V–D13, the impacts on market prices will not be significant. No price increase would exceed the .19% percent experienced by SIC 0724, cotton ginning. Price changes of this magnitude are not likely to result in market disruption or present an economic burden to consumers.

Based upon the data presented in Tables V–D12 and V–D13, OSHA has determined that the impact on the affected establishments is not significant to impair their economic viability and OSHA has made a preliminary determination that the proposed standard is economically feasible.

Regulatory Flexibility Analysis. In accordance with the Regulatory Flexibility Act (Pub. L. 96–353, 94 Stat. 1164 [5 U.S.C 601 et. seq.]), OSHA has assessed the impact of the rulemaking on small establishments. OSHA has determined that there will be no adverse economic impact on small agricultural establishments as a result of this rulemaking. This rulemaking is limited to agricultural establishments with 11 or more hired workers because of Congress's rider on OSHA appropriations which prohibits OSHA from using funds to proscribe, enforce or enact any standard that will affect farms with fewer than 11 employees. In Chapter II, it was determined that only 3.6 percent of all farm establishments have more than 10 employees; however, they produce more than 40% of all agricultural goods. Therefore, the proposed standard would only affect larger farms in the agricultural community. Small farms, which make up the remaining 96.4 percent of U.S. farms, may be indirectly affected by becoming more aware of the chemical hazards in agriculture, but because these farms employ fewer than 11 persons they will not be directly affected by this rulemaking. Hence, OSHA has assessed the possible impact on small farms and determined that no small farm will be adversely affected.

Environmental Impact Assessment. This assessment has been prepared in accordance with provisions of the National Environmental Policy Act (NEPA) (42 U.S.C. 4325 *et seq.*) as well as the regulations on the Council on Environmental Quality (40 CFR part 1500), and DOL–NEPA Compliance Procedures (29 CFR part 11).

OSHA has reviewed the standard and the information submitted by its contractor. As a result, OSHA has concluded that no significant adverse

environmental impacts are likely to occur as a result of this proposed standard. In fact, it is possible, that as a result of this rulemaking, persons using hazardous chemicals will be more aware of the possible consequences of exposure to these chemical. This could result in better controls and practices during the application of these substances. This in turn could have a beneficial environmental impact and OSHA invites public comment on this potential benefit.

International Trade Impact. Currently, some European countries have PELs which cover all aspects of the private sector, including agriculture. The United Kingdom has over 200 such PELs, one of which is a PEL of 10 mg/m³ for grain dust. The Federal Republic of Germany has a cotton dust PEL of 1.5 mg/m³. In addition, a committee empowered by the European Economic Community (EEC) is compiling list of PELs which members may someday be required to follow. Although their work is in its preliminary stages, once the list is completed it could be used as a market barrier by the EEC against U.S. agricultural products which were not produced by workers afforded protection from airborne contaminants. Currently, there is no trade barrier as such, however, one could develop if the United States does not afford its agricultural workers protection from air contaminants.

Summary. Based on the data summarized in Tables V–D12 and D–V13, OSHA has concluded that the economic impacts of the standard are clearly feasible for the agricultural sector and that the proposed rule is not expected to have an adverse affect on small establishments and the environment.

E. Preliminary Regulatory Impact Analysis for General Industry

1. Introduction

The Occupational Safety and Health Administration (OSHA) currently does not have specific permissible exposure limits (PELs) for mineral wool, fibrous glass, and asphalt fume. These substances are covered under the PEL for particulates not otherwise regulated (PNORs), which limits employee exposures to 15 mg/m³ total dust and 5 mg/m³ respirable dust. OSHA proposes to establish new PELs for these substances to protect the health of workers.

This section examines the potential effects associated with this proposed rulemaking. The nature and extent of occupational exposures are identified and the affected industries are profiled. The technological feasibility, the estimated costs and benefits, and the economic impacts of compliance with the proposed rule are presented.

2. Industry Profiles

Mineral wool and fibrous glass. An important consideration in discussing industries involved in the production and use of vitreous fibers is the use of appropriate definitions. For purposes of this analysis, the term man-made vitreous fiber (MMVF) is used as a generic term for a variety of fibers, including fibers made of rock, slag, minerals and glass. This analysis will consider mineral wool as a subset of MMVF, consisting of slag and rock wool. Fibrous glass is considered a separate subset of MMVF, distinct from mineral wool. OSHA intends that the proposed PEL for fibrous glass also applies to refractory ceramic fiber (RCF), and the exposures and potential impacts are analyzed for this substance as well. This preliminary analysis is based on research conducted by CONSAD Research Corporation [1], and on data supplied by the industry trade association, TIMA [2].

Standard industrial classification (SIC) codes 3296, 3297, and 3299 include over 70 establishments producing mineral wool, fibrous glass, RCF, and related products such as insulation and acoustical tile. In 1988, the latest year for which data are available, these plants employed over 28,000 workers and shipped over $3.2 billion worth of fiber products. Most of the shipments were fibrous glass products, including ordinary glass wool, insulation material, and continuous filament. Approximately 15 percent of the shipments were mineral wool products. The remainder was made up of special fine fiber and other vitreous fibers.

Mineral wool, fibrous glass, and RCF are available in a variety of different forms, reflecting a range of applications for these products. Blankets, batts, modules, textiles, boards, and vacuum-formed plastics are primarily fabricated in the bulk material manufacturing plants and can be made to customer specifications. Some bulk material is also sold to independent fabricators for making refractories.

Fiber-containing products are commonly used for insulation and fire protection applications. Fibrous glass is more prevalent in residential applications, and RCF tends to be used more in industrial and commercial applications. In addition to structural insulation, these products provide effective insulation for heating and cooling equipment, furnaces, tanks and storage facilities, pipes and ducts, and components of automobiles, ships, and airplanes. Fibrous products can also be used in reinforced plastics with applications in aerospace, appliances, construction, consumer goods, electrical equipment, protective gear, farming, and other areas.

Table V–E1 provides a summary of the industries associated with mineral wool, fibrous glass, and RCF products that involve employee exposures to airborne fibers. Most of the occupational exposure of concern in general industry occurs in the fiber manufacturing plants, which includes much of the fabrication of fiber-containing products. The potential for employee exposures may also exist for independent fabricators in the production of refractories and reinforced plastics where loose bulk fiber is used in the manufacturing process. The installation and removal of furnace linings and other insulation may involve employee exposures if fibers become airborne. The use of products made from or containing fibrous glass, RCF, or mineral wool generally does not result in significant occupational exposures during secondary manufacturing applications. For example, exposure to airborne fibers during the installation of preformed insulation in appliances or catalytic converters would be minimal.

TABLE V–E1.—ESTIMATED NUMBER AND REVENUES OF ESTABLISHMENTS PRODUCING OR USING MINERAL WOOL, FIBROUS GLASS, AND RCF

Industry revenues	Estimated number of establishments [1]	Estimated (millions)
Mineral Wool Production:		
Wool and Insulation Material	20	$303.5
Ceiling and Acoustical Tile	13	185.5
Fibrous Glass Production:		
Wool and Insulation Material	70	1,989.1
Continuous Filament	11	754.5
Special Fine Fiber	2	27.0
RCF and Other Fibers	12	75.0
User Industries: [2]		
Fabricators; Plastic and Refractory Manufacturers	20	50.0
Industrial Insulation Contractors	70	30.0

TABLE V-E1.—ESTIMATED NUMBER AND REVENUES OF ESTABLISHMENTS PRODUCING OR USING MINERAL WOOL, FIBROUS GLASS, AND RCF—Continued

Industry revenues	Estimated number of establishments [1]	Estimated (millions)
In-House Insulation Work	1,800	NA

[1] Plants may be included under more than one product with revenues divided proportionally.
[2] Products made from or containing fibers are used throughout industry in many applications with minimal potential for exposure to airborne fibers.

Source: Office of Regulatory Analysis, OSHA; based on CONSAD Research, 1991 [1].

Asphalt fumes. Asphalt is a constituent of most crude petroleum and is produced as part of the petroleum refining process. The total number of companies in the petroleum refining industry (SIC 2911) with the capacity to produce asphalt as a main product or byproduct in 1989 was estimated to be 72 [1]. According to statistics published by the U.S. Department of Commerce, the 309 establishments in SIC 2911 employed more than 50,000 production workers, and their total value of shipments in 1987 was $118.2 billion of which asphalt manufacturing represented a minor fraction [3].

Asphalt is used in the manufacture of a wide variety of products. The two main categories of products are paving mixtures (SIC 2951) and roofing products (SIC 2952). Asphalt may also be an ingredient for some paints and varnishes, but this minor application has not been found to be associated with any significant exposures [1].

Asphalt paving mixtures are produced at an estimated 4,000 establishments with total estimated sales of over $4.3 billion. Many different mixtures are available depending on the application and the specific properties desired. Asphalt paving mixtures are primarily used to pave streets, highways, and airfields.

Approximately 102 establishments produce an estimated $3.4 billion worth of roofing products annually. The products include asphalt saturated sheathing, boards, felts, and fabrics, asphalt roof cements and coatings, and asphalt shingles. Most of these products are used by roofing contractors, which are part of the construction industry.

Table V-E2 provides a summary of industries involved in the production of asphalt and asphalt products.

TABLE V-E2.—ESTIMATED NUMBER AND REVENUES OF PLANTS PRODUCING ASPHALT AND ASPHALT PRODUCTS

Industry	Estimated number of plants	Estimated revenues (millions)
Petroleum Refining (Asphalt Production)	72	$27,186.0
Paving Mixtures	4,000	4,346.2
Roofing Products	102	3,402.9

Source: Office of Regulatory Analysis, OSHA; based on CONSAD Research, 1991 [1].

3. Employee Exposures and Benefits

Mineral wool, fibrous glass, and RCF. Data on occupational exposures to these fibers are available from several NIOSH studies, from OSHA inspection experience, and from industry sources. Estimates of the numbers of exposed employees are based on figures reported by CONSAD and are derived from Department of Commerce data and industry sources [1].

An estimated 3,400 workers are potentially exposed to mineral wool (rock or slag wool) fibers in general industry. About 2,110 of these workers are involved in the production of mineral wool and mineral wool insulation materials. The use of mineral wool in the production of acoustical tile involves potential exposures to about 1,290 workers.

A NIOSH study of five mineral wool producers showed that worker exposures to respirable dusts and fibers were relatively low. Personal breathing zone sample results taken of production workers indicated that all exposures were below 1 f/cc. Mean fiber concentrations were reported to be 0.125 f/cc across all job categories, with the highest exposures occurring for laboratory and quality control workers at 0.196 f/cc. The mean of the total airborne particulate material concentrations for all facilities was 0.621 mg/m^3.

NIOSH also surveyed workers in two plants that used mineral wool in their products. One of the manufacturers produced industrial insulation and the other produced ceiling tiles. In the industrial insulation plant the mean fiber concentration for all exposed production workers was 0.208 f/cc as an 8-hour time-weighted average (TWA8). In the tile plant, occupational exposures ranged from 0.72 f/cc to 3.12 f/cc; these averages correspond to activities for which the sampling time may have been less than two hours. The highest exposures occurred during the mixing of raw materials.

These data are considered to represent upper bounds of current occupational exposures for two reasons. First, the NIOSH survey of the manufacturing facilities was done in 1980 and significant improvements in controls have been implemented since then. Second, the data reflect samples taken during activities that were considered to have the highest potential exposures and that may occur intermittently. The eight-hour time-weighted average (TWA8) exposures would be considerably lower.

More recent exposure data was collected by TIMA, the industry trade association. TIMA submitted a report to NIOSH in 1990 which included up-to-date industrial hygiene data of occupational exposures to vitreous fibers in five mineral wool manufacturing plants [2]. One of the plants also produced acoustical tile from mineral wool. The data were collected over a 17 month period from 1988 to 1989. None of the job categories had mean exposures above 0.6 f/cc TWA8. Area samples in tile manufacturing operations ranged from 0.31 f/cc to 0.64 f/cc.

Exposure monitoring data submitted by three different companies and covering 17 plants manufacturing mineral wool and/or acoustical tile show that most exposures are below 0.4 f/cc. Only two plants reported fiber concentrations above 0.5 f/cc.

In plants where employees are exposed to airborne mineral wool fibers, the use of personal protective equipment is widespread. Disposable respirators are the most common form of protection from the inhalation of fibers; in addition, some employees use face shields, earmuffs, and protective clothing.

Data on occupational exposures to fibrous glass and special purpose glassy fibers have been documented by NIOSH studies, collected from OSHA compliance inspections, and reported by TIMA in their July 10, 1990 document submitted to NIOSH.

An estimated 28,220 workers are potentially exposed to fibrous glass or RCF in primary manufacturing facilities. The majority of these workers (16,758) are involved in the production of ordinary fibrous glass wool and insulation material. Another 8,437 workers are estimated to be exposed during the production of continuous filament fibrous glass, and about 1,025 workers produce special fine fibers and other vitreous fiber products such as RCF.

The TIMA report summarizes a comprehensive database consisting of 1,570 samples of occupational exposure measurements taken between 1985 and 1989 in various types of manufacturing

facilities producing and using fibrous glass.

The mean occupational exposure during the production of glass wool was 0.11 f/cc. The production of continuous filament for use in fiberglass textiles resulted in mean occupational exposures of 0.03 f/cc. Samples taken during the production and use of very fine diameter glass fiber for special filtration products averaged 1.42 f/cc with over 77 percent of the samples falling below 1 f/cc. Occupational exposures during the production of RCF, a ceramic fiber, averaged 0.65 f/cc, and almost 80 percent of the samples collected were below 1 f/cc. As in mineral wool production, respiratory protection and other personal protective equipment is widely used in the production of fibrous glass and RCF.

Workers may be also exposed to fibers during a variety of secondary manufacturing applications. The TIMA database contains over 440 exposure samples taken in a variety of fibrous glass user industries. The mean fiber concentration found in the end use applications was 0.25 f/cc.

In general industry, the primary end use applications of concern include the installation and removal of insulation products with industrial applications, and the addition of fiber products in the production of refractories and plastics by independent fabricators. Based on the available data and on knowledge about the nature of the activities and materials involved, CONSAD concluded that uses of fibrous glass products in other secondary manufacturing applications in general industry would very rarely involve significant airborne fiber exposure.

The available NIOSH and OSHA data on occupational exposures to fibrous glass are primarily concentrated in the user industries. NIOSH conducted a study of a shingle manufacturing plant which produces shingles with fibrous glass cores. The results from the personal samples were reported by the laboratory as not detectable. The lower limit of detection was determined to be 0.01 f/cc. In a review of OSHA sampling results, the only job category in general industry found with mean exposures above 1f/cc was the saw operator in refractories production.

An estimated 200 employees are potentially exposed to fibers during the production of refractories and plastics in 20 facilities other than the primary manufacturing plants. Exposures for these workers average about 0.7 f/cc TWA8; the highest average exposure, 1.8 f/cc, was recorded for the saw operator [1].

The installation and removal of insulation in industrial applications is performed by maintenance employees and by independent contractors. Substantial insulation work for furnaces, pipes, process equipment, and fire protection occurs at about 1,800 facilities annually. In addition to over 10,000 employees at the facilities involved in this work regularly, jobs are also handled by about 70 independent contractors. A total of about 11,000 employees are engaged in this type of work, which generally entails potential exposure to RCF.

Exposure levels can vary significantly depending on the specific circumstances of a job and the type of product used. Health and safe handling information and outreach programs have been developed by RCF producers to assist users in adequately protecting employees. Monitoring results indicate that exposures are usually less than 1f/cc. Respirators are recommended when fiber levels are unknown.

Table V–E3 summarizes occupational exposure to fibrous glass, RCF, and mineral wool fibers in general industry. For each industry, the number of exposed employees and their typical exposure levels are presented. Exposure levels reflect ambient air concentrations in the employees' breathing zones and do not take the use of respiratory protection into account. Potential overexposures are most likely to occur during the production of acoustical tile, special fine fibers, and RCF; in the fabrication of refractories; and in the installation and removal of insulation.

The number of employees exposed above the proposed PELs is derived by estimating the probability of an overexposure among the workforce in each industry sector. Even in industries with mean exposures less than 1 f/cc the exposure data indicate that some employees may occasionally be exposed at levels greater than 1 f/cc or at levels exceeding the mixed exposure limit. Although detailed data on the specific distributions of exposures by job category are not available, the number of employees exposed above the proposed PELs can be estimated from information on the frequency distribution of sampling results.

TABLE V–E3.—SUMMARY OF OCCUPATIONAL EXPOSURES TO MINERAL WOOL, FIBROUS GLASS, AND RCF IN GENERAL INDUSTRY

Industry	Number of exposed workers	Mean exposure levels (f/cc)
Mineral wool production:		
Wool and insulation	2,110	[2] 0.4
Acoustical tile	1,290	.5
Fibrous glass production:		
Wool and insulation	16,758	[2] .2
Continuous filament	8,437	[2] .2
Special fine fiber	[illegible]	1.4
Other, including RCF	[illegible]	.7
User industries: [1]		
Fabricators: Plastics and refractories	[illegible]	.7
Insulation work	11,000	.6

[1] Products made from or containing fibers are used throughout industry in many applications with minimal potential for exposure to airborne fibers.
[2] Less than.

Source: Office of Regulatory Analysis, OSHA; based on CONSAD Research, 1991 [1].

OSHA estimates that compliance with the proposed PEL of 1 f/cc would result in lower exposures for about 2,000 employees working with fibers in general industry. This number represents about 1,500 workers in insulation work and about 100 workers each in the production of acoustical tile, special fine fiber, RCF, refractories, and plastics that would be overexposed at some time during the year without adequate respiratory protection. The full-time equivalent number would be 1,400 workers, including 1,000 insulation workers.

The benefits of establishing a lower PEL for mineral wool and fibrous glass include a reduction in cases of respiratory disease. Several epidemiological studies have shown significant excess mortality from such disease for workers that have been exposed to these fibers. More detailed information on the specific health effects associated with exposure to mineral wool and fibrous glass can be found in the health effects section of this preamble.

The epidemiological studies demonstrating significant excess mortality among cohorts with exposure to fibers typically show increases in incidence rates of 30 percent or more. The elevated incidence rates usually correspond to relatively high exposure levels, but the available evidence indicates that workers face a significant risk of disease at exposure levels above 1 f/cc. On average, the rate of occupationally induced respiratory disease should decline among the affected workers. If it is assumed that

the excess risk over a working lifetime would be reduced to less than one in one thousand, from two to four cases could be expected to be prevented over 45 years among the workers exposed above the proposed PEL.

Several studies link exposure to these fibers with illnesses such as chronic rhinitis, chronic coughs, bronchitis, fibrosis, and other conditions. A recent health hazard evaluation conducted by NIOSH at a fibrous glass manufacturer (RDHETA 90–145–2086, November, 1990) found that fifty to sixty percent of the workers participating complained of chronic cough, shortness of breath, and irritation of the eyes, skin, and upper respiratory tract (some of these symptoms may have been associated with smoking). Based on exposure monitoring data collected by NIOSH, exposure levels at the plant are representative of those in the industry.

Reductions in exposures as a result of achieving compliance with the proposed rule are estimated to prevent about 500 illnesses each year associated with exposures to fibers. Most of these illnesses are not expected to involve lost workdays; the annual number of lost workday illnesses potentially prevented by the proposed rule is estimated to be about 100. OSHA requests comments on the prevalence of health effects among workers exposed to fibrous glass and mineral wool, and on the effectiveness of compliance with the proposed rule in preventing such effects.

Based on research by Kip Viscusi, the implicit average value of avoiding each lost workday case would be about $40,000. This value is based on a willingness-to-pay methodology and takes existing compensation programs into account. The total potential benefit associated with the prevention of lost workday illnesses due to exposures to fibrous glass and mineral wool in general industry would be $4 million.

Asphalt fumes. Workers with occupational exposure to asphalt fume can be found in three industry categories within general industry. Approximately 144 workers may be exposed to asphalt fume during asphalt production; the production of paving mixtures involves approximately 12,000 production workers; and the manufacture of roofing products involves an estimated 7,100 production workers.

The production of asphalt in petroleum refineries is a highly automated process. In a typical plant, only two workers are required to operate the equipment. The production system is sealed and thus emissions of asphalt fume are minimal. Personal breathing zone samples were taken of workers during a site visit to an asphalt manufacturing refinery by CONSAD. The analysis of the samples revealed that the workers were exposed to the benzene soluble fraction of asphalt fumes at levels below 0.08 mg/m³.

Asphalt paving mixture manufacturing facilities are also generally automated, limiting the potential for employee exposures. OSHA inspection data for these facilities indicate that exposures range from 0.197 mg/m³ to 0.350 mg/m³. The National Asphalt Paving Association found that the mean exposure to the benzene soluble fraction of asphalt fume in two asphalt cement manufacturing plants was 0.297 mg/m³.

During the production of roofing products, employee exposures to asphalt fumes may be slightly higher. OSHA inspection data collected in five different plants manufacturing asphalt felts and coatings revealed exposures to the benzene soluble fraction of asphalt fume as high as 0.887 mg/m³. Exposures for most employees were below 0.4 mg/m³. Occupational exposures in plants manufacturing other roofing products, including shingles, are expected to be similar.

Table V–E4 summarizes the data on occupational exposures to asphalt fumes in general industry. The estimated number of exposed employees and the corresponding mean exposure levels are presented for each industry category. OSHA preliminarily concludes that employees involved in the production of asphalt, paving mixtures and roofing products are generally exposed below the proposed PEL of 5 mg/m³. However, most employees involved in the production of paving mixtures and roofing products may be potentially exposed above an alternative PEL of 0.2 mg/m³.

The potential benefits associated with compliance with the proposed standard of 5 mg/m³ or the alternative standard of 0.2 mg/m³ can be calculated by applying a quantitative risk assessment (QRA) developed for asphalt fume, which is discussed in detail in another section of this preamble. The QRA establishes a linear (proportional) relationship between lifetime cumulative exposure and risk of lung cancer, without threshold effects.

TABLE V–E4.—SUMMARY OF OCCUPATIONAL EXPOSURES TO ASPHALT FUMES IN GENERAL INDUSTRY

Exposure industry	Number of exposed workers	Mean levels (mg/m³)
Asphalt production	144	[1]0.1
Asphalt paving mixtures	12,000	.3
Roofing products	7,100	.5

[1] Less than.

Source: Office of Regulatory Analysis, OSHA; based on CONSAD Research, 1991 [1].

In order to derive the potential benefit of the alternative PEL of 0.2 mg/m³, the QRA is applied to exposures that would be lowered to comply with the standard. OSHA estimates that 12,000 full-time equivalent workers are exposed at an average of 0.3 mg/m³ in the production of paving mixtures, and that 7,100 workers are exposed at an average of 0.5 mg/m³ in the production of roofing products. If the QRA for asphalt fume is used to predict the excess cancer risk among these workers, the number of lung cancer cases expected to be attributable to these exposures over 45 years would be 36.

The exposures are likely to be reduced through the use of half-mask respirators with organic vapor cartridges and other controls which have an estimated protection factor of at least ten. Assuming full compliance with the standard would reduce average exposures to the benzene soluble fraction of asphalt for these workers to 0.05 mg/m³ over a working lifetime, the number of excess cancers expected to be prevented over 45 years would be 31.

With a PEL of 0.5 mg/m³, an estimated full-time equivalent of 2,000 workers producing paving mixtures and 3,000 workers producing roofing products would be exposed above the PEL. About 2,000 FTE workers in each industry would need respiratory protection. The number of excess cancers prevented over 45 years with full compliance would probably be fewer than 10.

With the proposed PEL of 5 mg/m³, it is unlikely that routine exposures would need to be reduced for any employees. Corresponding benefits would probably be close to zero.

4. Nonregulatory Alternatives

The new PELs for asphalt fume, mineral wool, and fibrous glass are proposed for general industry in conjunction with the accompanying proposed rulemaking for the construction industry.

The proposed construction industry regulation would add or revise PELs for over 400 substances, including asphalt fume, mineral wool, and fibrous glass. OSHA has determined that the proposed rule for the construction industry is necessary to ensure adequate protection of the health of construction workers. The analysis of nonregulatory and other alternative approaches for achieving this goal is included in the preamble of the proposed rule for construction.

New PELs are being proposed in the construction industry for asphalt fume, mineral wool, and fibrous glass; this proposed rule for general industry would ensure that the PELs are consistent for all workers. The alternative to this approach would be to establish different PELs for employees depending upon the industrial classification of the employer. OSHA has rejected this approach and has adopted a regulatory approach which recognizes the inherent hazardous nature of the substances being regulated and the desirability of affording equal protection to all workers exposed to the substances.

E. Technological Feasibility

OSHA has preliminarily determined that compliance with the proposed PELs for asphalt fume, fibrous glass, and mineral wool can be achieved with technology that is currently available and utilized in general industry. In operations where airborne concentrations cannot be reduced below the proposed PEL through the implementation of feasible controls, employees would be required to wear respirators. With a PEL of 0.2 mg/m^3, OSHA anticipates that such situations may occur in most facilities involved in the production of asphalt paving mixtures or roofing products. Additional respirator use would probably not be necessary to comply with a PEL of 5 mg/m^3 in any of the affected industries.

Data on occupational exposure to asphalt fume indicate that current occupational exposures are generally below 0.2 mg/m^3 during the production of asphalt. Employees control the highly automated process from air conditioned control booths with computerized equipment.

Recent technological advances have made the production of asphalt paving mixtures a relatively automated process at most plants. The facility operator can generally make most of the adjustments during a normal working day without leaving the control room. The ticket dispatcher usually coordinates and monitors the flow of trucks and loads of materials and may work in the control room or in a separate building. A front-end loader operator loads the feed bins with aggregates; such equipment can be outfitted with air conditioned cabs. Other employees work throughout the facility making necessary adjustments, performing preventative maintenance, and carrying out other responsibilities. Employees for whom airborne concentrations exceed the PEL could be adequately protected with an appropriate respirator.

Employees currently manufacturing asphalt roofing products may be exposed above a PEL of 0.2 mg/m^3 for asphalt. Most of these facilities already utilize feasible controls to reduce employee exposures, such as local exhaust ventilation at emissions sources. Although some increase in the effectiveness of controls or the application of additional controls may be possible, OSHA expects that many employees would be required to wear respirators. At the airborne concentrations found in this industry, respirators would be capable of providing sufficient protection from asphalt exposure.

The available data indicate that the production and use of mineral wool and mineral wool insulation in general industry usually does not involve exposures above 1 f/cc. During the manufacture of acoustical tile with mineral wool, some occupational exposures above 1 f/cc have been recorded, but these may not occur in all plants. Monitoring results from seven plants manufacturing and using mineral wool products showed no employee exposures above 0.3f/cc [2].

Occupational exposures during the production of fibrous glass wool, insulation, and continuous filament are generally below 0.2f/cc. Less than 2 percent of the workforce exposed to fibrous glass is involved in the production of special fine diameter fibers. Monitoring results during this process have averaged 1.42 f/cc, and over 77 percent of the observed fiber concentrations for employees exposed in these operations were less than 1 f/cc.

CONSAD Research has analyzed the circumstances of exposure in the production of fine diameter fibers and has concluded that the installation of additional engineering controls at work stations with exposures above 1 f/cc would enable exposures to be controlled below the proposed PEL [1]. The engineering controls could include improved general and local exhaust ventilation systems, portable vacuum systems, and enclosures. Improved work practices may also be able to reduce exposures in some cases. Respirators may be used to achieve compliance with the PEL if additional engineering controls are infeasible.

The available data on the production of RCF and the use of vitreous fibers in secondary manufacturing applications indicate that exposures are generally below 1 f/cc. In some exceptional circumstances exposures may be higher; the application of standard engineering controls alone or in combination with some supplemental respirator use would ensure compliance with the proposed PEL.

6. Costs of Compliance

Achieving full compliance with the proposed exposure levels for asphalt fumes, mineral wool, and fibrous glass would require additional expenditures on controls by some firms in general industry. Facilities with airborne concentrations above the proposed PELs would incur costs to reduce those levels.

The estimation of costs associated with the controls necessary to reduce exposures is consistent with the hierarchy of controls specified by OSHA. Respirators are to be used as a last resort if feasible engineering controls and work practices are insufficient.

The exposure profile and the feasibility of additional controls was evaluated by CONSAD Research Corporation under contract with OSHA. CONSAD identified the work stations and industries where additional controls would be needed, determined the effectiveness of the controls, and estimated the unit costs associated with the controls. The annualized incremental costs per plant and for each industry sector were calculated. OSHA supplemented the data in the CONSAD report with additional information submitted to NIOSH in response to the request for public comments on occupational asphalt exposure [F.R. Vol. 55, July 17, 1990].

Exposure monitoring data for employees involved in the production of asphalt indicate that the eight-hour time-weighted average exposures are sufficiently low to enable firms to be in compliance with the proposed PEL of 5 mg/m^3 and with the alternative PEL of 0.2 mg/m^3 without incurring costs for additional controls.

Mean exposure levels during the production of asphalt paving mixtures may exceed the alternative PEL of 0.2 mg/m^3, and additional controls would need to be implemented to ensure compliance in these establishments. Front-end loader operators could be protected by an enclosed cab with a filtered air supply which is estimated to cost $4,000. Assuming such a cab would

be necessary for an average of one loader at half of all affected asphalt pavement facilities, the resulting total estimated capital cost would be $8 million. Based on an expected life span of 10 years and an annual cost of capital of 10 percent, the corresponding annualized cost would be $1.30 million.

Additional engineering controls and improved work practices should be used to reduce exposures before relying on respiratory protection. Due to the current extent of automation and the mobile nature of the work at asphalt pavement facilities, the implementation of significant additional controls does not appear to be feasible. Employees outside of the control rooms for prolonged periods would probably have to wear half-mask respirators with organic vapor cartridges to comply with the alternative PEL of 0.2 mg/m³. An average of three employees per facility (a total of 12,000 employees) may be affected by this requirement on an average of two out of three work days. This would represent 8,000 FTE workers.

For purposes of estimating the cost of complying with the alternative PEL of 0.2 mg/m³, it is assumed that 12,000 respirators would be purchased for $22 each. With an average life span of five years, the annualized cost would be about $70,000. In addition, the respirator cartridges would need to be replaced on average for 8,000 workers every four days at a cost of $8 per set. This would amount to an annual cost of $4.16 million (260 work days per year divided by four, multiplied by $8 and by 8,000 workers). Total annualized costs of compliance for this industry are estimated to be $5.53 million.

With a PEL of 0.5 mg/m³, exposures for front end loaders may not need to be reduced, and the number of employees requiring respiratory protection is estimated to be a full-time equivalent of 2,000 workers. The purchase of 4,000 respirators would have an annualized cost of about $23,000, the replacement of cartridges for the equivalent of 2,000 workers every four days would cost $1.04 million, and the total annualized cost for this industry would be approximately $1.06 million.

Compliance with the proposed PEL of 5 mg/m³ is not expected to result in additional costs for the production of paving mixtures or for the manufacture of roofing products.

Manufacturers of roofing products would be required to provide additional exposure controls for most employees to achieve compliance with the alternative PELs of 0.2 mg/m³ and 0.5 mg/m³. Plants in this industry already use local exhaust ventilation systems at emission sources to some extent. New or improved 300 ventilation systems, other engineering controls, and changes in work practices would have to be implemented to the extent feasible in each plant to reduce exposures. Feasible controls generally include all practical and reasonable efforts to reduce exposures without resorting to building new plants or making drastic changes in the manufacturing process.

For purposes of estimating the costs of compliance that may be associated with the proposed standard, OSHA assumes that an average of $100,000 per plant could be spent on additional engineering controls, including new or improved ventilation systems, clean air islands, and enclosures. This amount represents potential incremental changes that may produce substantial reductions in exposures. For example, a study conducted by PACE, Incorporated for several other industries demonstrates that significant improvements in control technology can be achieved with such expenditures (Attachment to Exhibit 19–43 in OSHA docket H–057a). Annual costs associated with power and maintenance of these systems and with other elements of an exposure control program are estimated to be about $10,000 per plant. The estimated annualized cost of additional engineering controls for the industry would be $2.68 million.

The costs for additional engineering controls primarily represent an allowance for the costs of complying with the requirement to implement controls "whenever feasible." OSHA expects that plants in this industry have already implemented most feasible engineering controls and that further reductions in exposures through the implementation of additional controls would be marginal. OSHA invites comments on this preliminary assessment.

Although the implementation of additional controls would reduce exposure levels, it is likely that supplemental respiratory protection would be necessary for some employees. The data compiled by CONSAD indicate that two of seven job categories (the coater and the head saturator) have the highest average exposures and that engineering controls alone may not be able to achieve levels below 0.2 mg/m³ or 0.5 mg/m³ consistently in these operations. OSHA estimates that respiratory protection would have to be provided for about 2,030 employees on a full-time basis.

These employees would have to wear half-mask respirators with organic vapor cartridges. The respirators would cost about $22 each and have an estimated life span of five years; the annualized cost for these would thus be $11,781. The cartridges would need to be replaced once every four days on average for about $8 per set, for an annual cost of $1.06 million (260 work days per year, divided by 4, multiplied by $8 and by 2,030 workers). The annual cost for the industry for respiratory protection would be about $1.07 million. The total annualized cost of compliance for the manufacturers of roofing products with an alternative PEL of 0.2 mg/m³ would be an estimated $3.75 million.

During the production of acoustical tile from mineral wool, additional controls would be necessary in some plants to ensure compliance with the proposed PEL. CONSAD estimated that 13 plants would need to install additional controls at 3 work stations. The controls would primarily consist of local exhaust ventilation and would have an estimated annualized unit cost of about $3,000. In addition, three of these plants would need to supplement the engineering controls with respiratory protection for approximately 23 employees each. The total annualized costs of compliance for ceiling tile manufacturing would be about $131,000.

Two of the plants involved in fibrous glass production produce specialty fine fibers. Occupational exposures during this process may exceed the proposed PEL. An estimated six work stations in each plant would be affected. The installation of improved ventilation systems is the recommended method of control, and it is believed that these controls would be sufficient to ensure compliance with the proposed PEL. Improved housekeeping and changes in work practices may also reduce exposure levels. The total annualized costs for the two plants are estimated to be $18,660.

About 20 plants in secondary manufacturing applications may also need additional controls to meet the proposed PEL for fibrous glass. Plants most likely to be affected are associated with the production of refractories (including textiles and boards) and plastics. CONSAD estimated that each plant would have one affected work station that could be adequately controlled with standard ventilation systems. Annualized costs per plant are estimated to be about $3,000 and the estimated total annualized cost of compliance for these plants is $60,000.

The production of RCF involves potential exposures to airborne fibers. These plants have generally implemented feasible controls and respirator programs that adequately protect most workers from exposures above the proposed PELs. Additional

compliance costs as a result of this rulemaking would include providing respiratory protection for those employees intermittently exposed above 1 f/cc who currently do not wear respirators. Providing such protection for the equivalent of 60 full-time employees is estimated to cost $23,000 annually. Some plants may be able to implement additional engineering controls or improved housekeeping programs to reduce exposures below 1 f/cc; such alternatives would probably have comparable costs on an annual basis.

Workers may be exposed to RCF during the installation or removal of insulation products. The manufacturers of these products provide extensive information about the safe use of the products, including recommended work practices, potential health effects, and literature and videos on providing training and protecting the health of workers. Most employees involved in this type of work appear to be adequately protected with the appropriate combination of controls, work practices, and respirators. Additional costs for complying with the proposed PEL would be incurred for those employees who are exposed above 1 f/cc and not already protected. OSHA estimates that supplemental respiratory protection may be necessary for about 1,000 full-time equivalent employees at an annual cost of about $375,000.

Tables V–E5, V–E6, and V–E7 summarize the estimated costs of compliance with this rule with a proposed PEL for asphalt of 5 mg/m³ and with alternative PELs for asphalt of 0.2 mg/m³ and 0.5 mg/m³, respectively. OSHA requests that any comments regarding these preliminary estimates, including supporting or contradictory evidence, be submitted to the record to ensure that the potential regulatory impacts are accurately evaluated for the final rule.

The total annualized estimated cost of compliance for this rule with the proposed PEL for asphalt fume of 5 mg/m³ is $0.6 million, all of which would be attributable to the new PELs for fibers.

The total annualized estimated cost of compliance for this rule with an alternative PEL for asphalt fume of 0.2 mg/m³ in general industry would be $9.89 million. Most of these costs are attributable to compliance with the PEL for asphalt fume for which the primary exposure control measure would be the use of respirators. With an alternative PEL for asphalt fume of 0.5 mg/m³, the estimated total cost would be $5.42 million.

TABLE V–E5.—ESTIMATED COSTS OF COMPLIANCE FOR AFFECTED INDUSTRIES WITH A PEL FOR FIBERS OF 1 F/CC AND WITH A PEL FOR ASPHALT FUME OF 5 MG/M³

Affected industry	Number of affected plants	Total annualized cost of controls	Total annual cost of respirators	Annualized industry cost
Acoustical Tile	13	$117,000	$13,800	$130,800
Fine Diameter Fibers	2	18,660	0	18,660
Independent Fabricators	20	60,000	0	60,000
RCF Production	12	0	23,000	23,000
Insulation Work	1,500	0	375,000	375,000
Asphalt Pavement	4,000	0	0	0
Roofing Products	102	0	0	0
Total	5,649	195,660	411,800	607,460

Source: Office of Regulatory Analysis, OSHA; based on CONSAD Research, 1991 [1].

TABLE V–E6.—ESTIMATED COSTS OF COMPLIANCE FOR AFFECTED INDUSTRIES WITH A PEL FOR FIBERS OF 1 F/CC AND WITH A PEL FOR ASPHALT FUME OF 0.2 MG/M³

Affected industry	Number of affected plants	Total annualized cost of controls	Total annual cost of respirators	Annualized industry cost
Acoustical Tile	13	$117,000	$13,800	$130,800
Fine Diameter Fibers	2	18,660	0	18,660
Independent Fabricators	20	60,000	0	60,000
RCF Production	12	0	23,000	23,000
Insulation Work	1,500	0	375,000	375,000
Asphalt Pavement	4,000	1,300,000	4,230,000	5,530,000
Roofing Products	102	2,680,000	1,070,000	3,750,000
Total	5,649	4,175,660	5,711,800	9,887,460

Source: Office of Regulatory Analysis, OSHA; based on CONSAD Research, 1991 [1].

TABLE V–E7.—ESTIMATED COSTS OF COMPLIANCE FOR AFFECTED INDUSTRIES WITH A PEL FOR FIBERS OF 1 F/CC AND WITH A PEL FOR ASPHALT FUME OF 0.5 MG/M³

Affected industry	Number of affected plants	Total annualized cost of controls	Total annual cost of respirators	Annualized industry cost
Acoustical Tile	13	$117,000	$13,800	$130,800
Fine Diameter Fibers	2	18,660	0	18,660
Independent Fabricators	20	60,000	0	60,000
RCF Production	12	0	23,000	23,000

TABLE V–E7.—ESTIMATED COSTS OF COMPLIANCE FOR AFFECTED INDUSTRIES WITH A PEL FOR FIBERS OF 1 F/CC AND WITH A PEL FOR ASPHALT FUME OF 0.5 MG/M³—Continued

Affected industry	Number of affected plants	Total annualized cost of controls	Total annual cost of respirators	Annualized industry cost
Insulation Work	1,500	0	375,000	375,000
Asphalt Pavement	4,000	0	1,060,000	1,060,000
Roofing Products	102	2,050,000	1,070,000	3,750,000
Total	5,649	2,875,660	2,541,800	5,417,460

Source: Office of Regulatory Analysis, OSHA; based on CONSAD Research, 1991 [1].

7. Economic Impacts

The costs of compliance presented in the previous section were evaluated for their likely economic impacts in each of the affected industries. OSHA compared the estimated costs of compliance with data on annual revenues and profits for each sector.

The estimated costs necessary to achieve compliance with the proposed regulation are generally small in relation to revenues and profits. Table V–E8 shows the costs of compliance as a percentage of revenues and profits for each affected industry sector with an alternative PEL for asphalt fume of 0.2 mg/m³. The economic feasibility of a PEL for asphalt fume of 0.5 mg/m³ or 5 mg/m³ is established by demonstrating the economic feasibility of a PEL of 0.2 mg/m³.

The industry sectors affected by the proposed regulation would be the production of acoustical tile from mineral wool, the production of special application fine diameter glass fibers, the production of RCF, secondary manufacturing with vitreous fibers for refractories and plastics, insulation work in general industry, the production of asphalt pavement, and the manufacture of asphalt roofing products. The estimated compliance costs represent less than 0.15 percent of the revenues in each sector. The affected firms should be able to recoup the compliance costs with minimal price increases that would appear insignificant in comparison to other factors influencing production costs and pricing.

In the worst-case scenario where firms would not be able to bear any increase in prices without significant reductions in sales volumes, the estimated compliance costs would represent less than 4.5 percent of the pre-tax profits in each of the affected sectors. The resulting reductions in profitability would not be likely to cause significant impacts on the structure or viability of the affected sectors.

The compliance costs would probably be absorbed through some combination of slight price increases and reductions in profits. Most of the plants already have established safety and health programs to monitor the conditions of the work environment and control worker exposures to the extent necessary. This regulation would make it necessary for some plants to increase these efforts by targeting lower exposure levels corresponding to the proposed PELs.

In the mineral wool, fibrous glass, and RCF industries most employee exposures are already less than the proposed PEL of 1 f/cc; the compliance costs represent a marginal increase in the substantial efforts currently made to control exposures in these industries.

Compliance with the proposed PEL of 5 mg/m³ for asphalt would not require additional expenditures and thus is considered economically feasible.

Compliance with the alternative PEL of 0.2 mg/m³ for the benzene soluble portion of asphalt fume would probably require widespread use of respiratory protection in the asphalt pavement and roofing products industries. The estimated costs of controls reflect an approach of installing engineering controls to the extent feasible. Feasible controls would include reasonable, practical measures and exclude excessively costly or extravagant measures. The total estimated costs of compliance would be economically feasible.

The overall demand for asphalt products should not be significantly affected. Substitutes, when available, are likely to be of poorer quality or more expensive. The usefulness of and need for asphalt products makes the demand relatively inelastic. Ultimately, the costs of the increased protection of the health of employees working with asphalt would result in slight increases in the costs of roofing and paving work.

Regulatory flexibility analysis. OSHA has preliminarily concluded that this proposed regulation would not have a significant adverse impact on a substantial number of small establishments.

The proposed rule is not expected to have a significant adverse impact on the affected establishments because the costs of compliance would be small in comparison to both revenues and profits. There are no indications that small establishments would be disproportionately burdened by the proposed requirements. The controls necessary to reduce employee exposures are not associated with significant economies of scale, and the need for additional controls is primarily determined by the extent of current efforts to control exposures in the plants rather than by plant size. The proposed regulation would tend to equalize competitiveness among plants by eliminating any economic advantage associated with inadequate engineering controls in some plants.

TABLE V–E8.—COSTS OF COMPLIANCE AS PERCENTAGES OF REVENUES AND PROFITS FOR AFFECTED INDUSTRIES BASED ON A 0.2 MG/M³ PEL FOR ASPHALT FUME

Affected industry	Estimated revenues	Estimated pre-tax profits	Compliance costs as a percent of	
			Revenues	Pre-tax profits
Acoustical Tile	$185,500,000	$6,678,000	0.07	1.96
Special Fine Diameter Fiber	27,000,000	972,000	0.07	1.92

TABLE V-E8.—COSTS OF COMPLIANCE AS PERCENTAGES OF REVENUES AND PROFITS FOR AFFECTED INDUSTRIES BASED ON A 0.2 MG/M ^{3} PEL FOR ASPHALT FUME—Continued

Affected industry	Estimated [illegible]	Estimated pre-tax profits	Compliance costs as a percent of	
			Reve- [illegible]	Pre-tax profits
Independent Fabricators	50,000,000	1,750,000	0.12	3.43
RCF Production	75,000,000	2,700,000	0.03	0.85
Insulation Work	NA	NA	< 0.01	< 0.01
Asphalt Pavement	4,346,200,000	204,271,400	0.13	2.71
Roofing Products	3,402,900,000	85,072,500	0.11	4.41

NA: Not Applicable.
Source: Office of Regulatory Analysis, OSHA; based on CONSAD, 1991 [1].

References

1. CONSAD Research Corporation, Regulatory Analysis of OSHA's Proposed PELs for Occupational Exposures to Mineral (Slag/Rock) Wool and Fibrous Glass Fibers and Petroleum Asphalt Fumes. Prepared for the Office of Regulatory Analysis, OSHA, under contract number J9-F8-0033. January 30, 1991.
2. TIMA Inc., Health and Safety Aspects of Man-Made Vitreous Fibers: Information, Data, Comments, and Recommendations Regarding Occupational Exposure to Man-Made Vitreous Fibers. Stamford, Connecticut, July 10, 1990.
3. U.S. Department of Commerce, Bureau of the Census, Industry Series, Washington, DC, June 1990.

VI. Clearance of Information Collection Requirements

On March 31, 1983, the Office of Management and Budget (OMB) published a new 5 CFR Part 1320, implementing the information collection provisions of the Paperwork Reduction Act (PRA) of 1980, 44 U.S.C. 3501 *et. seq.* (48 FR 13666). Part 1320, which became effective on April 30, 1983, sets forth obtaining OMB clearance not later than the date of publication of the proposal in the **Federal Register** for collection of information requirements contained in proposed rules. It also requires agencies to include a statement in the notice of proposed rulemaking indicating that such information requirements have been submitted to OMB for review under section 3504(h) of the Paperwork Reduction Act.

In addition to the above requirements, applicable federal regulations also provide, 5 CFR 1320 4(a), 1320.5(a), and 1320.5(d), respectively, as follows:

An agency shall not engage in a collection of information without obtaining OMB approval of the collection of information and displaying a currently valid control number and, unless OMB determines it to be inappropriate, an expiration date.

Notwithstanding any other provision of law, no person shall be subject to any penalty for failure to comply with any information collection request if the request does not display a currently valid OMB control number, or, in the case of an information collection request which is submitted to nine or fewer persons, the request fails to state that for this reason it is not subject to OMB review under the Act.

Whenever a member of the public is protected from imposition of a penalty under this section for failure to comply with a collection of information, such penalty may not be imposed by an agency through judicial process, or by any other person through judicial or administrative process.

The proposed PELs update standard for Construction, Maritime, and Agriculture will create no additional recordkeeping requirements.

VII. Summary and Explanation of the Proposed Standard

Part 1926—Construction

Table Z, Construction in the standard includes proposed new permissible exposure limits for approximately 370 substances. Approximately 160 of the 370 are new PELs for substances which were not previously regulated. OSHA is proposing that the Z-Table in construction be identified as Table Z, Construction to indicate both its relation to and difference from Table Z-1-A for general industry. The substances listed on Tables Z-2 and Z-3 of 29 CFR part 1910 will be incorporated into the construction table, resulting in only one table for construction, Table Z, Construction.

Table Z, Construction is divided into two parts. On the left-hand side are the current (or transitional) limits, and on the right-hand side are the proposed limits for construction.

For construction, CFR part 1926.55 regulates exposure to gases, vapors, fumes, dusts, and mists, and references the "Threshold Limit Values of Airborne Contaminants for 1970" of the American Conference of Governmental Industrial Hygienists. These are the limits that currently exist and are enforced in construction.

The scope of many of the substance-specific OSHA health standards found in 29 CFR part 1910 includes coverage of the construction sector. Lead, § 1910.1025, and coke oven emissions, § 1910.1029, are the only substance-specific health standards specifying exposure limits that do not cover construction.

However, lead is covered in construction to the degree that the 1970 TLV for lead is 200 $\mu g/m^3$ TWA. And while the standard for coke oven emissions does not currently apply to construction, the coal tar pitch volatile TLV of 200 $\mu g/m^3$ TWA does apply.

OSHA is proposing, as part of this current rulemaking, a PEL of 50 $\mu g/m^3$ TWA for lead as an 8-hour TWA for construction. This is the level that has existed in general industry since 1978; this is also the level effective in the maritime sector. The ancillary provisions (e.g., medical surveillance, exposure monitoring) are outside the scope of this rulemaking. However, OSHA is currently developing a proposal to address all of the issues in a comprehensive standard covering workplace exposure to lead in the construction industry. OSHA is also proposing that the coke oven limit of 0.15 mg/m^3 (as found in § 1910.1029) apply to construction.

The policy reasons for this proposal are explained in part I of the preamble. The health bases for the new exposure limits are explained in part IV. The feasibility analysis for the proposal is summarized in part V. OSHA's preliminary findings that the proposed limits substantially reduce significant risk and are feasible, are based on these analyses.

In addition to Table Z, Construction, OSHA is proposing to both adopt some new language and to carry over some existing language from § 1926.55. Also, in some cases OSHA is proposing to

directly incorporate by technical amendment language currently applicable to construction by cross reference to the 1970 ACGIH TLVs.

The new language is in ¶ (a)(3), (a)(4), and (a)(5), which refer to compliance with the Final Rule Limits columns and to skin absorption. The language used is consistent with the language used in the General Industry standard. Also new is the language in ¶ (d) on start-up dates and transitional provisions. Comments are requested on all these provisions.

Substantially unchanged from prior provisions are ¶ (b) on Methods of Compliance and ¶ (c) on the Computation Formula. Paragraph (b) is a direct carryover from the existing § 1926.55(b). The Computation Formula is an existing provision already applicable to construction by cross reference to the 1970 ACGIH TLVs. Also ¶ (a)(1) and (a)(2), covering Transitional Limits, are existing requirements applicable to construction by cross reference to the 1970 ACGIH TLVs. Substantive comments are not requested on ¶ (a)(1), (a)(2), (b), and (c), as these changes are technical amendments being made for the convenience of the public. The record is not being opened to consider substantive changes to these provisions. OSHA will consider comments suggestive of style change for the purpose of clarity.

The language in the proposed § 1926.55 (a) sets forth the obligation to protect employees from airborne and skin exposure. Airborne limits may be Time-Weighted Averages (TWA), Short Term Exposure Limits (STEL) and Ceilings (C). These are defined in ¶ (a)(5).

The standard states in § (a)(5):

(3) The following definitions apply to paragraph (a) of this section and Table Z, Construction;

(i) Time-weighted average (TWA) is the average airborne exposure of an employee in any 8-hour work shift of a 40-hour work week which shall not be exceeded.

(ii) Short term exposure limit (STEL) is the employee's 15-minute time-weighted average exposure which shall not be exceeded at any time during a work day. This applies even if the eight hour time-weighted average is within the TWA.

(iii) Ceiling is the employee's exposure which shall not be exceeded during any part of the work day. If instantaneous monitoring is not feasible, then the ceiling shall be assessed by sampling over a 15-minute period as would be done for a STEL, unless a different time period is specifically indicated.

In the current 29 CFR 1926.55(a), OSHA specifies that the exposure levels used in the "Threshold Limit Values of Airborne Contaminants for 1970" shall apply to construction. The 1970 TLVs include the computation formula for mixtures. Therefore, the reference to the 1970 TLVs has applied to construction the same Computation Formula that has applied to general industry as set forth in ¶ (d) of the General Industry Air Contaminants Final Rule. (See section I–H for further discussion.)

The standard provides an explanation of skin notation. In ¶ (a)(4) the standard states:

An employee's skin exposure to materials listed in Table Z, Construction with an "S" notation shall be limited through the use of gloves, coveralls, goggles or other appropriate personal protective equipment or method necessary to prevent possible skin absorption.

Skin notations are used where the substance may be absorbed through the skin in sufficient amounts to cause systemic toxicity. As the standard's language indicates, appropriate personal protective equipment, engineering controls or work practices may also be used to minimize or eliminate skin contact in these situations. No specific hierarchy of controls is mandated for the prevention of skin contact. (See also discussion under VI. Health Effects Discussion, C. 17, Substances for Which OSHA is Adding Skin Notations.)

It should be noted that upon the effective date for the proposed new standard, OSHA is proposing that the Skin Designation in the Final Rule Limits columns becomes applicable. Consequently the Skin designation in the Transitional Limits Columns will no longer have effect after the effective date unless a stay is issued.

OSHA recognizes that it takes time for employers to evaluate exposures and purchase, install and make operable equipment to control such exposures. OSHA believes that 3 months from the date of publication is a reasonable time to evaluate exposures and come into compliance with any reasonable combination of controls. OSHA is proposing 4 years as the time to come into compliance with a preference for engineering controls. Comment on these start-up dates is requested.

The purpose of the Transitional Limits is to state the limits to be achieved by primary reliance on engineering controls, if feasible, during the 4 year transitional period. During the transitional period the new limits may be achieved by any reasonable combination of engineering, work practice and respiratory controls. This priority on compliance with engineering controls as set forth in § 1926.55 (b) for the exposure limits in the Transitional Limit Columns is the existing requirement. The exposure limits in the Transitional Limits columns also would remain in place if any of the new limits are stayed.

OSHA is not proposing to change limits in this rulemaking for 9 substances for which comprehensive rulemakings are in progress, or NPRMs have been published. These substances are cadmium (dust and fume), butadiene, ethylene dibromide, methylene chloride and two glycol ethers and their acetates. As OSHA has made substantial progress towards developing a comprehensive standard for these substances, it would be wasteful of resources to consider these substances in the current proceeding.

Complete and published proposed rules are available for cadmium (dust and fume), butadiene, ethylene dibromide and methylene chloride. For those substances for which a proposal has been published, the proposed PEL is located in the Proposed Final Rule columns in each Z–table. If a proposal has not been published for a specific substance, then the existing limit is reflected in the table. OSHA intends to carry over the PELs established in these separate substance-specific rulemakings to the Z-tables. OSHA wishes to make clear that it is not opening the record on any substance for which separate rulemaking is in progress.

In reference to ethylene dibromide, an NPRM was published in 1983, and a final rule has not been published. As EDB is no longer produced in the U.S., OSHA has given the substance low priority. Therefore, the present limits are listed in the Z-tables.

The language of this proposal follows the format shown to and approved by the Construction Advisory Committee. The style of this proposal differs from that of general industry and maritime. This difference is reflected primarily in the introductions to ¶ 1926.55(a), 1915.1000(a), and 1910.1000. OSHA believes there are advantages in following the same style across all sectors as much as is possible. OSHA is considering changing the format in construction to more closely follow the format in other sectors. OSHA requests comment on this.

Part 1915—Shipyards

OSHA consulted with the Shipyards Advisory Committee on the Air Contaminants Standard for Shipyards at its May and August 1991 meetings. The content and format of the regulatory language in part reflects the result of these consultations. The committee is generally supportive of the proposal.

The Committee believes that the Shipyard regulations in general should become more self-contained with less need for cross-reference to General Industry standards. They also suggested that placing health regulations in their own Subpart Z for shipyards and maintaining consistency of definitions and regulatory language with the 1989 PELs would make these regulations easier to understand. OSHA agrees with these suggestions.

Accordingly, OSHA is proposing that there be a new Subpart Z for Part 1915 Shipyards which contains a § 1915.1000 Air Contaminants. That section includes a Table Z, Shipyards which lists all of the permissible exposure limits (PELs) applicable to Shipyards.

Table Z, Shipyards incorporates PELs from 4 sources. Approximately 370 PELs that are either new or more protective than the old limits are being proposed in Shipyards. Approximately 210 PELs will remain unchanged. Approximately 15 substances are currently covered in Shipyards through cross-reference to single substance standards in 29 CFR 1910.1001–1048. They are listed in the table with the exposure limit and a cross- reference to the ancillary provisions of the single substance standard. Finally, there are two substances (coke oven emissions and cotton dust) that have been applied to general industry but have not been specifically applied to Shipyards. OSHA is proposing that the exposure limits for cotton dust be made applicable to Shipyards. Table I–F lists the substances which OSHA is either newly proposing for shipyards or is proposing more protective limits. OSHA is only opening the record for these substances.

Table Z, Shipyards lists all the substances for which OSHA has either existing or proposed PELs. Table Z, Shipyards is divided into Transitional Limit columns on the left and Proposed Final Rule Limit columns on the right. The Proposed Final Rule Limit columns include all the proposed exposure limits applicable to shipyards and all the existing exposure limits which are not being included in this rulemaking. The Proposed Final Rule Limit has columns which set forth the 8-hour TWA limit, the STEL limit, and the Ceiling limit, if applicable. The last column indicates whether there is skin designation. These terms are defined in § 1915.1000(a).

The Transitional Limit columns state the existing exposure limits which are applicable to ship repairing and shipbreaking based on the 1970 TLVs. As explained in the Legal Authority section, the existing exposure limits for shipbuilding are slightly different since they are based on the 1971 PELs. Since the anomaly is confusing and has no health basis, OSHA is proposing that on the effective date of this standard, the Transitional Limits for shipbuilding be identical with those for ship repairing and shipbreaking. To facilitate this transition, Table Z, Shipyards has already been organized to incorporate this change.

The purpose of the Transitional Limits is to state the limits to be achieved by primary reliance on engineering controls, if feasible, during the 4 year transitional period. During the transitional period the new limits may be achieved by any reasonable combination of engineering, work practice and respiratory controls. This priority on compliance for engineering controls as set forth in § 1915.1000(c) for the exposure limits in the Transitional Limit Columns is the existing requirement. The exposure limits in the Transitional Limits columns also would remain in place if any of the new limits are stayed.

It should be noted that upon the effective date for the proposed new standard, OSHA is proposing that the Skin Designation in the Final Rule Limits columns becomes applicable. Consequently the Skin designation in the Transitional Limits Columns will no longer have effect after the effective date unless there is a stay.

The substantive paragraphs of the proposed § 1915.1000 include the same concepts as the 1989 PELs for General Industry, and the same concepts as proposed for Construction, Longshoring and Marine Terminals. Pursuant to ¶ (a), an employee's exposure to substances listed in Table Z, Shipyards shall be kept below the limits specified in Table Z. It is the employer's responsibility to take steps to keep employee exposure below the limits.

The airborne limits for employee exposure may be an 8 hour Time Weighted Average (TWA), a Short Term Exposure Limit (STEL) or a Ceiling Limit, or in some cases a TWA and a STEL or a Ceiling. Those terms are defined in ¶ (a)(3) as follows:

(i) Time weighted average (TWA) is the employee's average airborne exposure in any 8-hour shift of a 40-hour work week which shall not be exceeded.

(ii) Short term exposure limit (STEL) is the employee's 15-minute time weighted average exposure which shall not be exceeded at any time during a work day unless another time limit is specified in a parenthetical notation below the limit. If another time period is specified, the time weighted average exposure over that time period shall not be exceeded at any time during the working day.

(iii) Ceiling is the employee's exposure which shall not be exceeded during any part of the work day. If instantaneous monitoring is not feasible, then the ceiling shall be assessed as a 15-minute time weighted average exposure which shall not be exceeded at any time over a working day.

When there is an "X" in the Skin designation column, then skin exposure shall be prevented or reduced to prevent skin absorption. Skin notations are used where the substance may be absorbed through the skin in sufficient amounts to cause systemic toxicity. As the standard's language indicates, appropriate personal protective equipment, engineering controls or work practices may also be used to minimize or eliminate skin contact in these situations. No specific hierarchy of controls is mandated for the prevention of skin contact. (See also discussion under VI. Health Effects Discussion, C. 17, Substances for Which OSHA is Adding Skin Notations.)

Paragraph (b) provides the computation formula if an employee is exposed to more than 1 toxic substance at one time. It is applicable if the employee is exposed to toxic substances affecting the same organ or causing the same disease. It permits the computation of maximum exposures in such circumstances when there are simultaneous exposures to several toxic substances. The computation formula is already applicable to ship repair and shipbreaking by their incorporation of the 1970 TLVs and to shipbuilding through the 1971 TLVs. OSHA is not opening the computation formulae for comment. (See Section I–H for further discussion.)

Paragraph (c) sets forth methods of compliance with a preference for feasible engineering and work practice controls. This paragraph states an existing requirement which already applies to Shipyards through the 1970 TLVs or the 1971 PELs. OSHA is not opening methods of compliance for comment in this proceeding as OSHA is in the process of completing a separate rulemaking on methods of compliance.

Paragraph (d) sets forth proposed effective and start-up dates. The proposed effective date is 90 days after publication in the **Federal Register** of the final rule pursuant to statute (section 6(b)4)). The Shipyard Advisory Committee indicated that they were familiar with the new exposure limits and it would save confusion if they were made applicable without delay. Accordingly OSHA is proposing that the new PELs take effect on the effective date with compliance with any reasonable combination of work practices, engineering controls or respirators. OSHA is also proposing, pursuant to the Committee's recommendation that the start-up date to come into compliance with a preference for feasible engineering

controls pursuant to ¶ (c) be 4 years after publication of the final rule in the **Federal Register**. OSHA requests comments on these start-up dates.

Paragraphs (d)(3) covers the Transitional Limits which are discussed above. Paragraph (d)(4) covers stays, if any, issued after the final rule is published. It provides that if a new limit is stayed, the prior (Transitional Limit) remain in effect.

A number of amendments to part 1915 are necessary to maintain consistency with other sectors. It is not OSHA's intention, by these proposed consequential amendments, to change other concepts in the Shipyards standards except to make clear that the Air Contaminants exposure limits are applicable to all employees and operations in the Shipyard industry.

OSHA is accordingly proposing to delete all references to the 1970 TLVs and replace them where necessary with reference to the proposed 29 CFR part 1915, subpart Z. These proposed amendments are made to §§ 1915.5, 1915.12(a)(3), 1915.12(b)(3) and 1915.32(b). However OSHA is not proposing changes to the provisions of § 1915.12 which require in certain circumstances pre-entry inspection prior to entry into holds, compartments and various areas where a build up of toxic substances may occur and various precautions must be taken.

There are nine substances for which OSHA has either published an NPRM or is otherwise engaged in rulemaking. The disposition of the PELs for those substances in regard to this rulemaking is discussed at greater length at the end of the discussion on the Summary and Explanation of the proposed Construction standard. (See VII. Summary and Explanation of the Proposed Standard, part 1926—Construction.) OSHA wishes to make it clear that it is not opening the record for any substance for which comprehensive rulemaking is in progress.

Parts 1917—Marine Terminals, and Part 1918—Longshoring

Table Z, Longshoring and Marine Terminals includes proposed permissible exposure limits (PELs) for approximately 370 new or more protective PELs in the Maritime subsectors of Marine Terminals and Longshoring. OSHA is proposing today a 1917 subpart Z for Marine Terminals (§ 1917.1000) and a 1918 subpart Z for Longshoring (§ 1918.1000).

As both sectors have identical Transitional and Final Rule exposure limits, OSHA is proposing to publish only one Z Table for both subsectors. Table Z, Longshoring & Marine Terminals. OSHA is proposing that the table be printed only once and that it be located in part 1917 after § 1917.1000. It will be cross-referenced in § 1918.1000. OSHA believes that this will not create an inconvenience or confusion to the public or as parts 1917 and 1918 are invariably published together. There is some saving of space and cost to the government by following this approach. Comments are requested on it.

The proposed new Table Z, Longshoring & Marine Terminals, and § 1917.1000 and § 1918.1000 are conceptually similar to the parallel provisions of part 1915. Accordingly, the discussions of those part 1915 provisions are applicable here and will not be repeated. Instead the differences and the different consequential provisions are discussed. See also the Legal Authority section of the preamble for the historical background of these distinctions.

The Final Rule limit columns of Table Z, Marine Terminals and Longshoring are identical to the same columns on Table Z, Shipyards. The Transitional Limits columns are different for approximately 30 substances. This is because the existing limits for Longshoring and Marine Terminals are based on the 1971 OSHA PELs whereas the existing limits for Shipyards are based on the 1970 TLVs. (See the above discussion of Shipyards for a further explanation.)

Some of the provisions of parts 1917 and 1918 are unique due to the nature of the original provisions in these two parts and to the special concern for confined spaces in holds, compartments, vehicles, warehouses etc. Also these sectors often handle intact sealed containers. OSHA's proposed intention is to make the Air Contaminant limits generally applicable to all operations in Marine Terminals and Longshoring with exceptions for intact sealed, containers and carbon monoxide while retaining the existing unique provisions of parts 1917 and 1918 to the extent they are not inconsistent with the air contaminants provisions.

Intact, sealed containers of toxic substances which are often handled in marine terminals and longshore operations, by definition, do not present the hazard of airborne exposure or skin contact with toxic substances. Part 1917 in § 1917.1(a)(2)(ii) currently provides that the air contaminant limits do not apply when a substance or cargo is contained within a sealed, intact means of packaging or containment complying with Department of Transportation or International Maritime Organization requirements. OSHA is proposing to retain this provision and formally incorporate it into part 1918. As long as toxic substances are properly contained there will not be exposure.

However, containers can be damaged in the course of cargo handling, resulting in the possibility of leakage and employee exposure. Consequently, OSHA is proposing language that specifies that should cargo containers become damaged to the extent that the possibility for leakage exists, then the Air Contaminant exposure limits apply. However, as these circumstances will be in the nature of accidents or emergencies, any reasonable combination of engineering controls, work practice and personal protective equipment is permitted as the method of compliance. The proposed language for these provisions is located in § 1917.1000(d) and § 1918.1000(d).

Because of the dangers of death from carbon monoxide poisoning in the confined spaces of holds, vehicles, rail cars and poorly ventilated warehouse spaces where workers in marine terminals and longshoring frequently work, parts 1917 and 1918 have detailed protective provisions located in § 1917.24 and § 1918.93(a). These sections have testing provisions and a requirement that employees be removed when carbon monoxide exposure exceeds 100 ppm in a confined space. Because of the difficulty of observing employees in confined spaces, the difficulty of escaping from them and the unfortunate occurrence of death to persons attempting to rescue persons disabled in confined spaces, these protective provisions are appropriate. OSHA is retaining those provisions but is proposing to lower the 8 hour TWA for carbon monoxide for these sectors from 50 ppm to 35 ppm and apply a 200 ppm ceiling measured over 5 minutes to open spaces. These limits are consistent with OSHA's final determination for general industry. See the health write-up for carbon monoxide.

Paragraph (b) provides the computation formula if an employee is exposed to more than 1 toxic substance at one time. It is applicable if the employee is exposed to toxic substances affecting the same organ or causing the same disease. It permits the computation of maximum exposures in such circumstances when there are simultaneous exposures to several toxic substances. The computation formula is already applicable to longshoring and marine terminals by reference to 1910.1000. OSHA is not opening the computation formulae for comment. (See Section I–H for further discussion.)

Part 1917 has certain unique provisions on hazardous cargo—

§ 1917.22, pesticides—§ 1917.25, menhaden (a species of fish) terminals—1917.73, welding and hot work—1917.152 and spray painting § 1917.153. These are being retained. However, § 1917.23(a) is proposed to be amended to clearly indicate that the air contaminant exposure levels are always applicable.

There are nine substances for which OSHA has either published an NPRM or is otherwise engaged in rulemaking. The disposition of the PELs for those substances in regard to this rulemaking is discussed at greater length at the end of the discussion on the Summary and Explanation of the proposed Construction standard. (See VII. Summary and Explanation of the Proposed Standard, part 1926—Construction.)

OSHA is proposing that the new air contaminant exposure limits take effect 90 days after the publication of the final rule with any reasonable combination of controls and 4 years after publication with a preference for engineering controls for Marine Terminals and Longshoring. OSHA requests comment on these dates.

Part 1928—Agriculture

Table Z, Agriculture includes proposed permissible exposure limits (PELs) for approximately 600 substances that have not previously been regulated in agriculture. OSHA is proposing that the Z–Table in agriculture be identified as Table Z, Agriculture to indicate both its relation to and difference from Table Z–1–A for General Industry. All of the limits that would apply in agriculture are shown in this single table, i.e., in Table Z, Agriculture. There is no transitional limit column in Table Z, Agriculture because OSHA PELs have not previously applied in agriculture.

When OSHA published its Air Contaminants Final Rule for general industry on January 19, 1989 (54 FR 2332 et seq.), the Agency stated:

> OSHA has also determined that it is appropriate to limit this rulemaking to the General Industry sector. Application to the Construction, Maritime & Agriculture segments may require some modifications to this proposed rule because of differences in exposures and work situations in the established PELs for these segments, and differences regarding feasibility for these sectors. OSHA will pursue this as part of second stage rulemaking * * *

The limits being proposed in agriculture today constitute part of that second stage of rulemaking. In the prior rulemaking for general industry, OSHA reduced the PELs for 212 substances and set new PELs for 164 substances previously not regulated by OSHA. The PELs being proposed today in Table Z, Agriculture include the 376 new limits that were promulgated in this earlier rulemaking. In addition, Table Z, Agriculture, proposes PELs for approximately 160 substances which were previously regulated by OSHA in general industry since 1970 but whose limits were not changed in the 1989 rulemaking. These 160 substances are being regulated in agriculture for the first time, and are included in Table 1–F.

There are another 52 or so substances for which OSHA had considered a change in the PEL in the proposal for general industry, but for which none was made in the final rule. Regulation of these 52 substances is also being proposed in agriculture.

To ensure consistency in the protection afforded to workers across sectors, OSHA is also proposing PELs in agriculture for 10 of the 24 substances the Agency has regulated in comprehensive 6(b) rulemaking proceedings (see §§ 1910.1000 to 1910.1047) which have numerical exposure limits as part of the standard. These limits have previously been issued in section 6(b) rulemakings. The health effects and feasibility of OSHA's PELs have been fully considered. These findings are summarized in Section IV.C of this preamble. These ten substances are asbestos, vinyl chloride, inorganic arsenic, lead, benzene, cotton dust, DBCP, acrylonitrile, ethylene oxide and formaldehyde.

OSHA is not proposing to change limits for 9 substances for which the Agency is currently conducting comprehensive section 6(b) rulemakings are in progress because the appropriate PELs for these substances will not be determined until their respective final rules are published. OSHA is not opening the record in the present air contaminants rulemaking for construction, maritime, or agriculture for any substance that is currently undergoing separate rulemaking. Once these rulemakings are completed, OSHA will add the respective PELs to all of the Z tables.

The policy reasons for this proposal are explained in part I of the preamble. The health basis of the new exposure limits are explained in part IV and feasibility analysis for the proposal is summarized in part V. OSHA's preliminary conclusion that the proposed limits noted in Table Z, Agriculture, substantially reduce significant risk among workers in agriculture and are feasible in this sector are based on the analyses summarized in those sections of the preamble.

In addition to the Table Z, Agriculture, OSHA is also proposing to adopt language similar to that in ¶ (a) of § 1910.1000 and include it in part 1928. That language sets forth the obligation of agricultural employers to protect employees from overexposures to airborne contaminants and toxic substances that cause systemic toxicity when absorbed through the skin. Airborne limits may be expressed as Time-Weighted Averages (TWAs), Short Term Exposure Limits (STELs) and Ceilings (Cs). These are defined as follows in paragraph (a)(3) of the standard:

(i) Time Weighted Average (TWA) is the average airborne exposure of an employee in any 8-hour work shift of a 40-hour work week which shall not be exceeded.

(ii) Short term exposure limit (STEL) is the employee's 15-minute time-weighted average exposure which shall not be exceeded at any time during a work day. This applies even if the eight hour time-weighted average is within the TWA.

(iii) Ceiling is the employee's exposure which shall not be exceeded during any part of the work day. If instantaneous monitoring is not feasible, then the ceiling shall be assessed by sampling over a 15-minute period as would be done for a STEL, unless a different time period is specifically indicated.

The standard provides an explanation of skin notation. In ¶ (a)(2) the standard states:

> An employee's skin exposure to materials listed in Table Z, Agriculture, with an "S" Notation shall be limited through the use of gloves, coveralls, goggles or other appropriate personal protective equipment or method necessary to prevent possible skin absorption.

Skin notations are used where the substance may be absorbed through the skin in sufficient amounts to cause systemic toxicity. As the standard's language indicates, appropriate personal protective equipment, engineering controls or work practices may also be used to minimize or eliminate skin contact in these situations. No specific hierarchy of controls is mandated for the prevention of skin contact. (See also discussion under VI. Health Effects Discussion, C. 17, Substances for Which OSHA is Adding Skin Notations.)

OSHA is proposing, in paragraph (b), to apply the Computation (or Mixture) Formula to agriculture. See section I–H for further discussion.

OSHA recognizes that it takes time for employers to evaluate exposures and to purchase, install and make operable equipment to control such exposures. OSHA believes that 1 year from the date of publication is a reasonable time to evaluate exposures and come into

compliance with any combination of respirators, work practices and engineering controls for agriculture. This is longer than OSHA normally provides, but as no exposure limits previously applied in agriculture, more time may be necessary for agricultural employers to become familiar with the regulations, determine employee exposures, and institute controls. Four years are provided for coming into compliance with a preference for engineering controls. Comment on these start-up dates is requested.

With regard to proposed § 1910.1000(c), methods of compliance, OSHA recognizes that engineering controls that are typically used in industrial applications have not been regularly adapted to the agricultural setting. However, simple controls, such as placing lids on containers, have been used in agriculture. Although OSHA recognizes that agriculture may lag behind other sectors with regard to the use of engineering controls, industrial hygiene sampling, etc., the Agency expects that experience with this regulation will parallel OSHA's experience in the past and that use of technology and industrial hygiene approaches will occur over a reasonable period of time.

The four-year period of time before compliance with engineering controls will permit knowledge of appropriate technology to become known to the farming community. OSHA and NIOSH intend to engage in extensive outreach to inform agricultural employers of appropriate technology to control exposures on farms.

The purpose of this approach is to allow OSHA to assure safe and healthy work sites for workers in agriculture, while maintaining a role which includes education as well as enforcement. This approach is consistent with OSHA's activity and goals in agriculture. As literature and training become more widely available, the agricultural employer will be expected to have access to that knowledge.

VIII. Public Participation-Public Hearings

Interested persons are invited to submit written data, views, and arguments with respect to OSHA'S proposed rule. These comments must be postmarked on or before September 25, 1992, and submitted in quadruplicate to the Docket Officer, Docket H–020A, room N–2625, U.S. Department of Labor, Washington, DC 20210, Tel. (202) 523–7894.

This rulemaking covers a large number of substances and industries. Therefore, to permit the public and OSHA to efficiently review the comments, it is necessary to specify the format of the comments in greater detail than normally required for OSHA rulemaking.

Comments on the general concept of the proposal shall be first and shall begin on a new page with the heading "General Comments", the name of the commenter, and the commenter's SIC code or codes if a business.

Comments on individual substances shall follow. The comment on each substance shall start on a new page with a heading identifying the substance with the name and code number used in Table I–G (the HS code number) of the preamble; (not the CAS number) and a second line identifying the comment as on "Health Issues" or on "Feasibility Issues." If there are comments on both they shall begin on separate pages with headings that identify the substances, and its code number and area of the comment. For feasibility comments, the heading should contain a third line identifying the SIC codes (preferably 4 digit) that the comment covers.

In addition the first or second page of each comment is to have a table of contents indicating the page number that the general comments begin and the page number that Health and Feasibility comments for each chemical individually discussed begin. Finally, one of the four sets of each comment received should not be stapled or bound, so that it can be easily copied. Written submissions must clearly identify the specific provisions of the proposal which are addressed and the position taken with respect to each issue.

The data, views and arguments that are submitted will be available for public inspection and copying at the above address. All timely submissions received will be made a part of the record of this proceeding. The exhibits cited in this document will be available for public inspection and copying at the above address.

In addition, the record and the record of the General Industry rulemaking Docket No. H–020 currently contains many data bases of economic and health information. That information is also available for inspection and copying at the Docket Office. (Much of the information is on computer tape. OSHA will supply duplicate tapes for the copying charge.)

Notice of Intention To Appear at the Informal Hearing

Pursuant to section 6(b)(3) of the OSHA Act, informal public hearings will be held on this proposal in Washington, DC from Tuesday, October 20, 1992 through Friday, October 30, 1992; in San Diego from Tuesday, November 17, 1992 through Friday, November 20, 1992; and in Des Moines from Tuesday, December 8, 1992 through Friday, December 11, 1992. In each city if testimony is less extensive the hearing may terminate earlier and if more extensive it may be extended.

The Washington hearing will commence at 9:30 a.m. in the auditorium of the Frances Perkins Building, U.S. Department of Labor, 3d Street and Constitution Avenue NW., Washington, DC 20210.

The hearing in San Diego will be at the Holiday Inn on the Bay, 1355 North Harbor Drive, San Diego, California, 92101, Tel: (619) 232–3861.

The hearing in Des Moines will be at the Holiday Inn Des Moines, 1050 Sixth Ave., Des Moines, Iowa, 50314, Tel: (515) 283–0151.

Persons desiring to participate at the informal public hearing must file a notice of intention to appear by September 11, 1992. The notice of intention to appear must contain the following information:

1. The name, address, and telephone number of each person to appear;
2. The capacity in which the person will appear;
3. The approximate amount of time required for the presentation;
4. The issues and substances that will be addressed;
5. A brief statement of the position that will be taken with respect to each issue and substance addressed; and
6. Whether the party intends to submit documentary evidence and, if so, a brief summary of it.
7. The city or cities where the participant wishes to testify. Repetitive testimony in different cities is not permitted.

The Notice of Intention to Appear shall be mailed to Mr. Thomas Hall, OSHA Division of Consumer Affairs, Docket No. H–020, U.S. Department of Labor, room N–3647, 200 Constitution Avenue, NW., Washington, DC 20210, telephone (202) 523–8615, and shall be postmarked no later than September 11, 1992.

A notice of intention to appear also may be transmitted by facsimile to (202) 523–5986 or (for FTS) to 8–523–5986, by the same date provided the original and 3 copies are sent to the same address and postmarked no later than 3 days later.

Filing of Testimony and Evidence Before the Hearing

Any party requesting more than ten (10) minutes for presentation at the informal public hearing, or who intends

to submit documentary evidence, must provide in quadruplicate the testimony and evidence to be presented at the informal public hearing. The documentary evidence and testimony shall follow the format and include the headings and index required for comments. One copy shall not be stapled or bound and be suitable for copying. These materials must be provided to Mr. Thomas Hall, OSHA Division of Consumer Affairs at the address above and be postmarked no later than September 25, 1992.

Each submission will be reviewed in light of the amount of time requested in the Notice of Intention to Appear. In instances where the information contained in the submission does not justify the amount of time requested, a more appropriate amount of time will be allocated and the participant will be notified of that fact prior to the informal public hearing.

Any party who has not substantially complied with the above requirement may be limited to a ten-minute presentation and may be requested to return for questioning at a later time.

Any party who has not filed a notice of intention to appear may be allowed to testify for no more than 10 minutes as time permits, at the discretion of the Administrative Law Judge, but will not be allowed to question witnesses.

Notice of intention to appear, testimony and evidence will be available for inspection and copying at the Docket Office at the address above.

Conduct and Nature of Hearings

The hearings will commence at 9:30 a.m. on the first day in each city. At that time, any procedural matters relating to the proceeding will be resolved.

The nature of an informal rulemaking hearing is established in the legislative history of section 6 of the OSH Act and is reflected by OSHA's rules of procedure for hearings (29 CFR 1911.15(a)). Although the presiding officer is an Administrative Law Judge and questioning by interested persons is allowed on crucial issues, the proceeding is informal and legislative in type. The Agency's intent, in essence, is to provide interested persons with an opportunity to make effective oral presentations which can proceed expeditiously in the absence of procedural restraints which impede or protract the rulemaking process.

Additionally, since the hearing is primarily for information gathering and clarification, it is an informal administrative proceeding rather than an adjudicative one. The technical rules of evidence, for example do not apply. The regulations that govern hearings and the pre-hearing guidelines to be issued for this hearing will ensure fairness and due process and also facilitate the development of a clear, accurate and complete record. Those rules and guidelines will be interpreted in a manner that furthers that development. Thus, questions of relevance, procedure and participation generally will be decided so as to favor development of the record.

The hearing will be conducted in accordance with 29 CFR part 1911. It should be noted that § 1911.4 specifies the Assistant Secretary may upon reasonable notice issue alternatives procedures to expedite proceedings or for other good cause. The hearing will be presided over by an Administrative Law Judge who makes no decision or recommendation on the merits of OSHA's proposal. The responsibility of the Administrative Law Judge is to ensure that the hearing proceeds at a reasonable pace and in an orderly manner. The Administrative Law Judge, therefore, will have all the powers necessary and appropriate to conduct a full and fair informal hearing as provided in 29 CFR part 1911 including the powers:

1. To regulate the course of the proceedings;
2. To dispose of procedural requests, objections and comparable matters;
3. To confine the presentations to the matters pertinent to the issues raised;
4. To regulate the conduct of those present at the hearing by appropriate means;
5. In the Judge's discretion, to question and permit the questioning of any witness and to limit the time for questioning; and
6. In the Judge's discretion, to keep the record open for a reasonable, stated time (known as the post hearing comment period) to receive written information and additional data, views and arguments from any person who has participated in the oral proceedings.

State Plan Applicability

The 25 states with their own OSHA-approved occupational safety and health plans must adopt a comparable standard within six months of the publication date of a final standard. These States include: Alaska, Arizona, California, Connecticut (for State and local government employees only), Hawaii, Indiana, Iowa, Kentucky, Maryland, Michigan, Minnesota, Nevada, New Mexico, New York (for State and local government employees only), North Carolina, Oregon, Puerto Rico, South Carolina, Tennessee, Utah, Vermont, Virginia, Virgin Islands, Washington, Wyoming. Until such time as a State standard is promulgated, Federal OSHA will provide interim enforcement assistance, as appropriate.

List of Subjects

29 CFR Part 1910

Air contaminants, Occupational safety and health. Permissible exposure limits, Health, Risk assessment, Construction, Maritime, Shipyards, Longshoring, Marine terminals, Agriculture.

29 CFR Part 1915

Air contaminants, Hazardous substances, Longshore and harbor workers, Occupational safety and health, Permissible exposure limits, Vessels.

29 CFR Part 1917

Air contaminants, Hazardous substances, Longshore and harbor workers, Occupational safety and health, Permissible exposure limits.

29 CFR Part 1918

Air contaminants, Freight, Hazardous substances, Longshore and harbor workers, Occupational safety and health, Permissible exposure limits, Vessels.

29 CFR Part 1926

Air contaminants, Construction industry, Hazardous substances, Occupational safety and health, Permissible exposure limits.

29 CFR Part 1928

Air contaminants, Agriculture, Occupational safety and health, Permissible exposure limits.

IX. Authority

This document has been prepared under the direction of Dorothy L. Strunk, Acting Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor, 200 Constitution Avenue NW., Washington, DC 20210. Pursuant to section 6 of the Occupational Safety and Health Act of 1970 (29 U.S.C. 655), section 4 of the Administrative Procedures Act (5 U.S.C. 551), 29 CFR part 1911 and Secretary of Labor's Orde: No. 1–90 (55 FR 9033), it is proposed to amend 29 CFR parts 1910, 1915, 1917, 1918, 1926 and 1928 as set forth below.

Signed at Washington, DC., this 29 day of May, 1992.

Dorothy L. Strunk,

Acting Assistant Secretary of Labor.

X. Standard

OSHA proposes to amend 29 CFR part 1910, subpart Z as follows:

PART 1910—OCCUPATIONAL SAFETY AND HEALTH STANDARDS

1. The authority citation for Subpart Z continues to read in part as follows:

Authority: Secs. 6, 8 Occupational Safety and Health Act, 29 U.S.C. 655, 657; Secretary of Labor's Orders 12–71 (36 FR 8754), 8–76 (41 FR 25059), 9–83 (48 FR 35736) or 1–90 (55 FR 9033), as applicable; and 29 CFR part 1911.

All of subpart Z issued under section 6(b) of the Occupational Safety and Health Act, 29 U.S.C. 655(b) except those substances listed in the Final Rule Limits columns of Table Z–1–A, which have identical limits listed in the Transitional Limits columns of Table Z–1–A, Table Z–2 or Table Z–3. The latter were issued under section 6(a) (29 U.S.C. 655(a)).

Section 1910.1000, the Transitional Limits columns of Table Z–1–A, Table Z–2 and Table Z–3 also issued under 5 U.S.C. 553. § 1910.1000, the Transitional Limits columns of Table Z–1–A, Table Z–2 and Table Z–3 not issued under 29 CFR part 1911 except for the arsenic, benzene, cotton dust, and formaldehyde listings.

* * * * *

2. It is proposed to amend § 1910.1000 by adding a new paragraph (f)(5) to read as follows:

§ 1910.1000 Air contaminants.

* * * * *

(f) Effective dates, start-up dates and transitional provisions

* * * * *

(5) For the exposure limits for asphalt, fiberglass and mineral wool the effective date shall be [90 days after the date of publication of final rule in the **Federal Register**], the start-up date for compliance through any reasonable combination of engineering controls work practices and personal protective equipment shall be (6 months after the date of publication of the final rule in the **Federal Register**) and the start-up date for compliance using the methods of compliance specified in paragraph (e) of this section shall be (2 years after the date of publication in the **Federal Register**).

3. It is proposed to amend § 1910.1000, Table Z–1–A by inserting new entries as follows:

(a) After the entry for Asbestos insert into the Substance column the entry "Asphalt fumes (total particulate)", insert in the CAS No. column "8052–42–4" and insert in the Final rule limits, TWA, mg/m^3 column the entry "5".

(b) After the entry for Ferrovanadium dust, insert into the Substance column the entry "Fibrous glass" and insert into the Final rule limits, TWA, columns the entry "1 f/cc".

(c) After the entry for Mica, insert into the Substance column the entry "Mineral wool" and insert into the Final rule limits, TWA, columns the entry "1 f/cc".

OSHA proposes to amend 29 CFR parts 1915, 1917, 1918, 1926 and 1928 as follows:

I. OSHA proposes to amend 29 CFR part 1915 as follows:

PART 1915—SHIPYARDS

1. It is proposed to revise the authority citation for part 1915 to read as follows:

Authority: Sec. 41, Longshore and Harbor Workers Compensation Act (33 U.S.C. 941); secs 4, 6, 8, Occupational Safety and Health Act of 1970 (29 U.S.C. 653, 655, 657); Secretary of Labor's Order No. 12–71 (36 FR 8754), 8–76 (41 FR 25059), 9–83 (48 FR 35736) or 1–90 (55 FR 9033), as applicable; 29 CFR Part 1911.

Section 1915.99 also issued under 5 U.S.C. 553.

§ 1915.5 [Amended]

2. It is proposed to amend § 1915.5 by removing the paragraph which reads:

Threshold Limit Values, 1970, American Conference of Governmental Industrial Hygienists, 1014 Broadway, Cincinnati, Ohio 45202, Subpart B, § 1915.12 (a)(3) and (b)(3); Subpart C, § 1915.32(b).

3. It is proposed to amend § 1915.12 by revising paragraphs (a)(3) and (b)(3) to read as follows:

§ 1915.12 Precautions before entering.

(a) Flammable atmospheres and residues.

* * * * *

(3) If the atmosphere in the space to be entered is found to contain a concentration of flammable vapor or gas below the level immediately dangerous to life as defined in § 1915.152(b)(1), but above the permissible exposure limits specified in 29 CFR 1915, subpart Z, employees shall be protected in accordance with the requirements of 29 CFR 1915, subpart Z.

(b) Toxic atmospheres and residues.

* * * * *

(3) If the atmosphere in the space to be entered is found to contain a concentration of toxic contaminants below the level immediately dangerous to life as defined in § 1915.152(b)(1), but above the permissible exposure limits specified in 29 CFR 1915, subpart Z, employees shall be protected in accordance with the requirements of 29 CFR 1915, subpart Z.

* * * * *

4. It is proposed to amend § 1915.32 by revising paragraph (b) to read as follows:

§ 1915.32 Toxic cleaning solvents.

* * * * *

(b) The requirements of 29 CFR 1915, subpart Z shall be followed.

* * * * *

Subparts M–Y—[Reserved]

5. It is proposed to reserve subparts M–Y and to add a new subpart Z consisting of § 1915.1000 to read as follows:

Subpart Z—Toxic and Hazardous Substances

§ 1915.1000 Air contaminants.

(a) *Exposure limits.* An employee's exposure to any substance listed in Table Z, Shipyards shall be limited in accordance with the requirements of the following paragraphs of this section:

(1) Final Rule Limits Columns. An employee's exposure to any substance listed in Table Z, Shipyards shall not exceed the Time Weighted Average (TWA), Short Term Exposure Limit (STEL) and Ceiling Limit specified for that substance in Table Z under the Final Limits columns.

(2) Skin Designation. To prevent or reduce skin absorption, an employee's skin exposure to substances listed in Table Z, Shipyards with an "X" in one or both of the Skin Designation columns following the substance name shall be prevented or reduced to the extent necessary in the circumstances through the use of gloves, coveralls, goggles, or other appropriate personal protective equipment, engineering controls or work practices.

(3) Definitions. The following definitions are applicable to Table Z, Shipyards:

(i) *Time weighted average (TWA)* is the employee's average airborne exposure in any 8-hour work shift of a 40-hour work week which shall not be exceeded.

(ii) *Short term exposure limit (STEL)* is the employee's 15-minute time weighted average exposure which shall not be exceeded at any time during a work day unless another time limit is specified in a parenthetical notation below the limit. If another time period is specified, the time weighted average exposure over that time period shall not be exceeded at any time during the working day.

(iii) *Ceiling* is the employee's exposure which shall not be exceeded during any part of the work day. If instantaneous monitoring is not feasible, then the ceiling shall be assessed as a 15-minute time weighted average

exposure which shall not be exceeded at any time over a working day.

(b) *Computation formulae.* The computation formula which shall apply to employee exposure to more than one substance for which 8-hour time weighted averages are listed in subpart Z of 29 CFR Part 1915 in order to determine whether an employee is exposed over the regulatory limit is as follows:

(1)(i) The cumulative exposure for an 8-hour work shift shall be computed as follows:

$$E=(C_aT_a+C_bT_b+\ldots C_nT_n)\div 8$$

Where:

E is the equivalent exposure for the working shift.

C is the concentration during any period of time T where the concentration remains constant.

T is the duration in hours of the exposure at the concentration C.

The value of E shall not exceed the 8-hour time weighted average specified in subpart Z or 29 CFR part 1915 for the material involved.

(ii) To illustrate the formula prescribed in paragraph (d)(1)(i) of this section, assume that Substance A has an 8-hour time weighted average limit of 100 ppm noted in Table Z. Assume that an employee is subject to the following exposure:

Two hours exposure at 150 ppm
Two hours exposure at 75 ppm
Four hours exposure at 50 ppm

Substituting this information in the formula, we have

$$(2\times 150+2\times 75+4\times 50)\div 8=81.25 \text{ ppm}$$

Since 81.25 ppm is less than 100 ppm, the 8-hour time weighted average limit, the exposure is acceptable.

(2)(i) In case of a mixture of air contaminants an employer shall compute the equivalent exposure as follows:

$$E_m=(C_1\div L_1+C_2\div L_2)+\ldots (C_n\div L_n)$$

Where:

E_m is the equivalent exposure for the mixture.

C is the concentration of a particular contaminant.

L is the exposure limit for that substance specified in subpart Z of 29 CFR part 1915.

The value of E_m shall not exceed unity (1).

(ii) To illustrate the formula prescribed in paragraph (d)(2)(i) of this section, consider the following exposures:

Substance	Actual concentration of 8 hr. exposure (ppm)	8 hr. TWA PEL (ppm)
B	500	1000
C	45	200
D	40	200

Substituting in the formula we have:

$Em=500\div 1.000+45\div 200+40\div 200$
$Em=0.500+0.225+0.200$
$Em=0.925$

Since Em is less than unity (1), the exposure combination is within acceptable limits.

(c) *Methods of compliance.* To achieve compliance with paragraphs (a) and (b) of this section administrative or engineering controls must first be implemented whenever feasible. When such controls are not feasible to achieve full compliance, protective equipment or other protective measures shall be used to keep the exposure of employees to air contaminants within the limits prescribed in this section. Any equipment and technical measures used for this purpose must first be approved for each particular use by a competent industrial hygienist or other technically qualified person.

(d) *Effective and start-up dates*—(1) Effective date. The effective date for the permissible exposure limits specified in the Final Rule Limits columns of Table Z, Shipyards is [90 days after date of publication in the **Federal Register**].

(2) Start-up dates. (i) The permissible exposure limits specified in the Final Rule Limits columns of Table Z, Shipyards shall be achieved by any reasonable combination of engineering controls, work practices and personal protective equipment from the effective date through [4 years after date of publication in the **Federal Register**].

(ii) The permissible exposure limits specified in the Final Rule Limits columns of Table Z, Shipyards shall be achieved by the method of compliance specified in paragraph (c) of this section commencing [4 years after date of publication in the **Federal Register**].

(iii) The skin designation in the Final Rule Limits columns shall be complied with commencing on the effective date.

(3) Transitional provision. The permissible exposure limits specified in the Transitional Limits columns of Table Z, Shipyards (which are a Time Weighted Average unless proceeded by a "C" in which case they are a ceiling) shall be achieved by the methods of compliance specified in paragraph (c) of this section from the effective date through [4 years after date of publication in the **Federal Register**].

(4) Stays. If any new or amended provisions or new or revised limits for any substance or substances are either administratively stayed or judicially stayed or vacated, then the existing provisions or limits for those substances specified in the Transitional Limits columns of Table Z, Shipyards shall remain in effect until such stay is lifted, or indefinitely if the limit is vacated.

TABLE Z.—S

Substance	CAS No.[4]	Transitional Limits[a] ppm[a]	(1970 TLVs) mg/m³[b]	Skin Designation
Abate; see Temephos				
Acetaldehyde	75-07-0	200	360	—
Acetic acid	64-19-7	10	25	—
Acetic anhydride	108-24-7	5	20	—
Acetone	67-64-1	1000	2400	—
Acetonitrile	75-05-8	40	70	—
2-Acetylaminofluorine; see 1910.1014	53-96-3			
Acetylene	74-86-2	E		
Acetylene dichloride; see 1,2-Dichloroethylene				
Acetylene tetrabromide	79-27-6	1	14	—
Acetylsalicylic acid (Aspirin)	50-78-2	—	—	—
Acrolein	107-02-8	0.1	0.25	—
Acrylamide	79-06-1	—	0.3	X
Acrylic acid	79-10-7	—	—	—
Acrylonitrile [f]; see 1910.1045	107-13-1	(1)	(1)	(1)
Aldrin	309-00-2	—	0.25	X
Allyl alcohol	107-18-6	2	5	X
Allyl chloride	107-05-1	1	3	—
Allyl glycidyl ether (AGE)	106-92-3	(C)10	(C)45	—
Allyl propyl disulfide	2179-59-1	2	12	—
alpha-Alumina	1344-28-1			
Total dust		—	15	—
Respirable fraction		—	5	—
Aluminum (as Al)	7429-90-5			
Metal				
Total dust		—	15	—
Respirable fraction		—	5	—
Pyro powders		—	—	—
Welding fumes[g]		—	—	—
Soluble salts		—	—	—
Alkyls		—	—	—
Alundum; see alpha-Alumina				
4-Aminodiphenyl; see 1910.1011	92-67-1			
2-Aminoethanol; see Ethanolamine				
2-Aminopyridine	504-29-0	0.5	2	—
Amitrole	61-82-5	—	—	—
Ammonia	7664-41-7	50	35	—
Ammonium chloride fume	12125-02-9	—	—	—
Ammonium sulfamate	7773-06-0			
Total dust		—	15	—
Respirable fraction		—	5	—
n-Amyl acetate	628-63-7	100	525	—
sec-Amyl acetate	626-38-0	125	650	—
Aniline and homologs	62-53-3	5	19	X
Anisidine (o-, p- isomers)	29191-52-4	—	0.5	X
Antimony and compounds (as Sb)	7440-36-0	—	0.5	—
ANTU (alpha Naphthylthiourea)	86-88-4	—	0.3	—
Argon	7440-37-1	E		
Arsenic, inorganic compounds (as As) [f]; see 1910.1018	7440-38-2	—	0.01	—
Arsenic, organic compounds (as As)	7440-38-2	—	0.5	—
Arsine	7784-42-1	0.05	0.2	—
Asbestos [f]; see 1926.58	Varies	(1)	(1)	(1)
Asphalt (Petroleum) fumes	8052-42-4	—	—	—
Atrazine	1912-24-9	—	—	—
Azinphos-methyl	86-50-0	—	0.2	X

Z.—Shipyards

Skin Designation	Proposed Final Rule Limits						Skin Designation
	TWA		STEL [c]		CEILING		
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
—	100	180	150	270	—	—	—
—	10	25	—	—	—	—	—
—	—	—	—	—	5	20	—
—	750	1800	1000	2400	—	—	—
—	40	70	60	105	—	—	—
—	1	14	—	—	—	—	—
—	—	5	—	—	—	—	—
—	0.1	0.25	0.3	0.6	—	—	—
X	—	0.03	—	—	—	—	X
—	10	30	—	—	—	—	X
(1)	2	—	10	—	—	—	X
X	—	0.25	—	—	—	—	X
X	2	5	4	10	—	—	X
—	1	3	2	6	—	—	—
—	5	22	10	44	—	—	—
—	2	12	3	18	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	2	—	—	—	—	—
—	—	2	—	—	—	—	—
—	0.5	2	—	—	—	—	—
—	—	0.2	—	—	—	—	—
—	—	—	35	27	—	—	—
—	—	10	—	20	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	100	525	—	—	—	—	—
—	125	650	—	—	—	—	—
X	2	8	—	—	—	—	X
X	—	0.5	—	—	—	—	X
—	—	0.5	—	—	—	—	—
—	—	0.3	—	—	—	—	—
—	—	0.01	—	—	—	—	—
—	—	0.5	—	—	—	—	—
—	0.05	0.2	—	—	—	—	—
(1)	—	0.2 f/cc	—	1 f/cc (30 min)	—	—	—
—	—	5	—	—	—	—	—
—	—	5	—	—	—	—	—
X	—	0.2	—	—	—	—	X

Substance	Transitional Limits[a] CAS No.[d]	Transitional Limits[a] ppm[e]	(1970 TLVs) mg/m³[b]	Skin Designation
Barium, soluble compounds (as Ba)	7440-39-3	—	0.5	—
Barium sulfate	7727-43-7			
Total dust		—	15	—
Respirable fraction		—	5	—
Benomyl	17804-35-2			
Total dust		—	15	—
Respirable fraction		—	5	—
Benzene [f,g]; see 1910.1028	71-43-2	(1)	(1)	(1)
Benzidine; see 1910.1010	92-87-5		A1	
p-Benzoquinone; see Quinone				
Benzo(a)pyrene; see Coal tar pitch volatiles				
Benzoyl peroxide	94-36-0	—	5	—
Benzyl chloride	100-44-7	1	5	—
Beryllium and beryllium compounds (as Be)	7440-41-7	—	0.002	—
Biphenyl; see Diphenyl				
Bismuth telluride, Undoped	1304-82-1			
Total dust		—	15	—
Respirable fraction		—	5	—
Bismuth telluride, Se-doped	1304-82-1	—	—	—
Bisphenol A; see Diglycidyl ether				
Borates, tetra, sodium salts				
Anhydrous	1330-43-4	—	—	—
Decahydrate	1303-96-4	—	—	—
Pentahydrate	12179-04-3	—	—	—
Boron oxide	1303-86-2			
Total dust		—	15	—
Boron tribromide	10294-33-4	1	10	—
Boron trifluoride	7637-07-2	(C)1	(C)3	—
Bromacil	314-40-9	—	—	—
Bromine	7726-95-6	0.1	0.7	—
Bromine pentafluoride	7789-30-2	0.1	0.7	—
Bromoform	75-25-2	0.5	5	X
Butadiene (1,3-Butadiene) [b]; see 55 FR 32736	106-99-0	1000	2200	—
Butane	106-97-8	—	—	—
Butanethiol; see Butyl mercaptan				
2-Butanone (Methyl ethyl ketone)	78-93-3	200	590	—
2-Butoxyethanol	111-76-2	50	240	X
n-Butyl-acetate	123-86-4	150	710	—
sec-Butyl acetate	105-46-4	200	950	—
tert-Butyl acetate	540-88-5	200	950	—
Butyl acrylate	141-32-2	—	—	—
n-Butyl alcohol	71-36-3	100	300	—
sec-Butyl alcohol	78-92-2	150	450	—
tert-Butyl alcohol	75-65-0	100	300	—
Butylamine	109-73-9	(C)5	(C)15	X
tert-Butyl chromate (as CrO_3)	1189-85-1	—	(C)0.1	X
n-Butyl glycidyl ether (BGE)	2426-08-6	50	270	—
n-Butyl lactate	138-22-7	—	—	—
Butyl mercaptan	109-79-5	0.5	1.5	—
o-sec-Butylphenol	89-72-5	—	—	—
p-tert-Butyltoluene	98-51-1	10	60	—
Cadmium fume (as Cd) [b]; see 55 FR 4052	7440-43-9	—	(C)0.1	—

Shipyards—Continued

Skin Designation	Proposed Final Rule Limits						
	TWA		STEL [c]		CEILING		Skin Designation
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
—	—	0.5	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
(1)	1	—	5	—	—	—	—
—	—	5	—	—	—	—	—
—	1	5	—	—	—	—	—
—	—	0.002	—	0.025 (30 min.)	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	—	—	—	—	—	—
—	1	10	—	—	1	10	—
—	0.1	0.7	0.3	2	1	3	—
X	0.1	0.7	—	—	—	—	—
—	0.5	5	—	—	—	—	—
—	2	—	10	—	—	—	X
—	800	1900	—	—	—	—	—
—	200	590	300	885	—	—	—
X	25	120	—	—	—	—	X
—	150	710	200	950	—	—	—
—	200	950	—	—	—	—	—
—	200	950	—	—	—	—	—
—	10	55	—	—	—	—	—
—	—	—	—	—	—	—	—
—	100	305	—	—	50	150	—
—	100	300	150	450	—	—	X
—	—	—	—	—	5	15	—
—	25	135	—	—	—	0.1	X
—	5	25	—	—	—	—	X
—	0.5	1.5	—	—	—	—	—
—	5	30	—	—	—	—	—
—	10	60	20	120	—	—	X
—	—	0.001 or 0.005	—	0.005 or 0.025	—	—	—

Cadmium dust (as Cd) [h]; see 55 FR 4052	7440-43-9	—	0.2	—	
Calcium carbonate	1317-65-3	—			
Total dust		—	15	—	
Respirable fraction		—	5	—	
Calcium cyanamide	156-62-7	—	—	—	
Calcium hydroxide	1305-62-0				
Total dust		—	15	—	
Respirable fraction		—	5	—	
Calcium oxide	1305-78-8	—	5	—	
Calcium silicate	1344-95-2				
Total dust		—	15	—	
Respirable fraction		—	5	—	
Calcium sulfate	7778-18-9				
Total dust		—	15	—	
Respirable fraction		—	5	—	
Camphor, synthetic	76-22-2	—	2	—	
Caprolactam	105-60-2				
Dust		—	—	—	
Vapor		—	—	—	
Captafol (Difolatan)	2425-06-1	—	—	—	
Captan	133-06-2	—	—	—	
Carbaryl (Sevin)	63-25-2	—	5	—	
Carbofuran (Furadan)	1563-66-2	—	—	—	
Carbon black	1333-86-4	—	3.5	—	
Carbon dioxide	124-38-9	5000	30000	—	10
Carbon disulfide	75-15-0	20	30	X	
Carbon monoxide	630-08-0	50	55	—	
Carbon tetrabromide	558-13-4	—	—	—	
Carbon tetrachloride	56-23-5	10	25	X	
Carbonyl fluoride	353-50-4	—	—	—	
Catechol (Pyrocatechol)	120-80-9	—	—	—	
Cellulose	9004-34-6				
Total dust		—	15	—	
Respirable fraction		—	5	—	
Cesium hydroxide	21351-79-1	—	—	—	
Chlordane	57-74-9	—	0.5	X	
Chlorinated camphene	8001-35-2	—	0.5	X	
Chlorinated diphenyl oxide	55720-99-5	—	0.5	—	
Chlorine	7782-50-5	1	3	—	
Chlorine dioxide	10049-04-4	0.1	0.3	—	
Chlorine trifluoride	7790-91-2	(C)0.1	(C)0.4	—	
Chloroacetaldehyde	107-20-0	(C)1	(C)3	—	
a-Chloroacetophenone (Phenacyl chloride)	532-27-4	0.05	0.3	—	
Chloroacetyl chloride	79-04-9	—	—	—	
Chlorobenzene	108-90-7	75	350	—	
o-Chlorobenzylidene malononitrile	2698-41-1	0.05	0.4	—	
Chlorobromomethane	74-97-5	200	1050	—	
2-Chloro-1,3-butadiene; see beta-Chloroprene					
Chlorodifluoromethane	75-45-6	—	—	—	
Chlorodiphenyl (42% Chlorine) (PCB)	53469-21-9	—	1	X	
Chlorodiphenyl (54% Chlorine) (PCB)	11097-69-1	—	0.5	X	
1-Chloro,2,3-epoxypropane; see Epichlorohydrin					
2-Chloroethanol; see Ethylene chlorohydrin					
Chloroethylene; see Vinyl chloride					
Chloroform (Trichloromethane)	67-66-3	50	240	—	
bis(Chloromethyl) ether; see 1910.1008	542-88-1				
Chloromethyl methyl ether; see 1910.1006	107-30-2				
1-Chloro-1-nitropropane	600-25-9	20	100	—	
Chloropentafluoroethane	76-15-3	—	—	—	
Chloropicrin	76-06-2	0.1	0.7	—	
beta-Chloroprene	126-99-8	25	90	X	
o-Chlorostyrene	2039-87-4	—	—	—	
o-Chlorotoluene	95-49-8	—	—	—	

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TABLE Z.—Shipyards

Substance	CAS No.[d]	Transitional Limits[c] ppm[a]	Transitional Limits[c] mg/m³[b]	(1970 TLVs) ppm[a]	(1970 TLVs) mg/m³[b]	Skin Designation
2-Chloro-6-(trichloromethyl) pyridine	1929-82-4					
Total dust		—	15	—	15	—
Respirable fraction		—	5	—	5	—
Chlorpyrifos	2921-88-2	—	—	—	—	—
Chromic acid and chromates (as CrO_3)	Varies with compound	—	0.1	—	0.1	—
Chromium (II) compounds (as Cr)	7440-47-3	—	0.5	—	0.5	—
Chromium (III) compounds (as Cr)	7440-47-3	—	0.5	—	0.5	—
Chromium metal and insol. salts (as Cr)	7440-47-3	—	1	—	1	—
Chrysene; see Coal tar pitch volatiles						
Clopidol	2971-90-6					
Total dust		—	15	—	15	—
Respirable fraction		—	5	—	5	—
Coal dust (less than 5% SiO_2), Respirable fraction		—	—	—	—	—
Coal dust (greater than or equal to 5% SiO_2), Respirable quartz fraction		—	—	—	—	—
Coal tar pitch volatiles (benzene soluble fraction), anthracene, BaP, phenanthrene, acridine, chrysene, pyrene	65996-93-2	—	0.2	—	0.2	—
Cobalt metal, dust, and fume (as Co)	7440-48-4	—	0.1	—	0.1	—
Cobalt carbonyl (as Co)	10210-68-1	—	—	—	—	—
Cobalt hydrocarbonyl (as Co)	16842-03-8	—	—	—	—	—
Coke oven emissions; see 1910.1029						
Copper	7440-50-8					
Fume (as Cu)		—	0.1	—	0.1	—
Dusts and mists (as Cu)		—	1	—	1	—
Corundum; see Emery						
Cotton dust[f]; see 1910.1043		—	1	—	1	—
This 8-hour TWA applies to respirable dust as measured by a vertical elutriator cotton dust sampler or equ respirable dust personal sampler may be used. In these circumstances the exposure limit is 1 mg/m³ respirab						
Crag herbicide (Sesone)	136-78-7					
Total dust		—	15	—	15	—
Respirable fraction		—	5	—	5	—
Cresol, all isomers	1319-77-3	5	22	5	22	X
Crotonaldehyde	123-73-9; 4170-30-3	2	6	2	6	—
Crufomate	299-86-5	—	—	—	—	—
Cumene	98-82-8	50	245	50	245	X
Cyanamide	420-04-2	—	—	—	—	—
Cyanides (as CN)	Varies with compound	—	5	—	5	—
Cyanogen	460-19-5	10	—	10	—	—
Cyanogen chloride	506-77-4	—	—	—	—	—
Cyclohexane	110-82-7	300	1050	300	1050	—
Cyclohexanol	108-93-0	50	200	50	200	—
Cyclohexanone	108-94-1	50	200	50	200	—
Cyclohexene	110-83-8	300	1015	300	1015	—
Cyclohexylamine	108-91-8	—	—	—	—	—
Cyclonite	121-82-4	—	1.5	—	1.5	X
Cyclopentadiene	542-92-7	75	200	75	200	—
Cyclopentane	287-92-3	—	—	—	—	—
Cyhexatin	13121-70-5	—	—	—	—	—
2,4-D-(Dichlorophenoxyacetic acid)	94-75-7	—	10	—	10	—
Decaborane	17702-41-9	0.05	0.3	0.05	0.3	X
Demeton (Systox)	8065-48-3	—	0.1	—	0.1	X

yards—Continued

in ig- on	Proposed Final Rule Limits						Skin Desig-nation
	TWA		STEL [c]		CEILING		
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	0.2	—	—	—	—	X
—	—	—	—	—	—	0.1	—
—	—	0.5	—	—	—	—	—
—	—	0.5	—	—	—	—	—
—	—	1	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	2	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	0.2	—	—	—	—	—
—	—	0.05	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	1	—	—	—	—	—
—	—	0.5	—	—	—	—	—

or equivalent instrument. For the Transitional Limit and where it is not feasible to use a vertical elutriator, a espirable dust, personal sampler.

—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
X	5	[illegible]	—	—	—	—	X
—	2	[illegible]	—	—	—	—	—
—	—	5	—	—	—	—	—
X	50	245	—	—	—	—	X
—	—	2	—	—	—	—	—
—	—	5	—	—	—	—	—
—	10	20	—	—	—	—	—
—	—	—	—	—	0.3	0.6	—
—	300	1050	—	—	—	—	—
—	50	200	—	—	—	—	X
—	25	100	—	—	—	—	X
—	300	1015	—	—	—	—	—
—	10	40	—	—	—	—	—
X	—	1.5	—	—	—	—	X
—	75	300	—	—	—	—	—
—	500	1720	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	10	—	—	—	—	—
X	0.05	0.3	0.15	0.9	—	—	X
X	—	0.1	—	—	—	—	X

Diacetone alcohol (4-Hydroxy-4-methyl-2-pentanone)	123-42-2	50	240	—
1,2-Diaminoethane; see Ethylenediamine				
Diazinon	333-41-5	—	—	—
Diazomethane	334-88-3	0.2	0.4	—
Diborane	19287-45-7	0.1	0.1	—
1,2-Dibromo-3-chloropropane (DBCP)[5]; see 1910.1044	96-12-8	0.001	—	X
1,2-Dibromoethane; see Ethylene dibromide				
2-N-Dibutylaminoethanol	102-81-8	—	—	—
Dibutyl phosphate	107-66-4	1	5	—
Dibutyl phthalate	84-74-2	—	5	—
Dichloroacetylene	7572-29-4	(C)0.1	(C)0.4	—
o-Dichlorobenzene	95-50-1	(C)50	(C)300	—
p-Dichlorobenzene	106-46-7	75	450	—
3,3'-Dichlorobenzidine; see 1910.1007	91-94-1			
Dichlorodifluoromethane	75-71-8	1000	4950	—
1,3-Dichloro-5,5-dimethyl hydantoin	118-52-5	—	0.2	—
Dichlorodiphenyltrichloroethane (DDT)	50-29-3	—	1	X
1,1-Dichloroethane	75-34-3	100	400	—
1,2-Dichloroethane; see Ethylene dichloride				
1,2-Dichloroethylene	540-59-0	200	790	—
Dichloroethyl ether	111-44-4	(C)15	(C)90	X
Dichloromethane; see Methylene chloride				
Dichloromonofluoromethane	75-43-4	1000	4200	—
1,1-Dichloro-1-nitroethane	594-72-9	(C)10	(C)60	—
1,2-Dichloropropane; see Propylene dichloride				
1,3-Dichloropropene	542-75-6	—	—	—
2,2-Dichloropropionic acid	75-99-0	—	—	—
Dichlorotetrafluoroethane	76-14-2	1000	7000	—
Dichlorvos (DDVP)	62-73-7	—	1	X
Dicrotophos	141-66-2	—	—	—
Dicyclopentadiene	77-73-6	—	—	—
Dicyclopentadienyl iron	102-54-5			
Total dust		—	15	—
Respirable fraction		—	5	—
Dieldrin	60-57-1	—	0.25	X
Diethanolamine	111-42-2	—	—	—
Diethylamine	109-89-7	25	75	—
2-Diethylaminoethanol	100-37-8	10	50	X
Diethylene triamine	111-40-0	(C)10	(C)42	X
Diethyl ether; see Ethyl ether				
Diethyl ketone	96-22-0	—	—	—
Diethyl phthalate	84-66-2	—	—	—
Difluorodibromomethane	75-61-6	100	860	—
Diglycidyl ether (DGE)	2238-07-5	(C)0.5	(C)2.8	—
Dihydroxybenzene; see Hydroquinone				
Diisobutyl ketone	108-83-8	50	290	—
Diisopropylamine	108-18-9	5	20	X
4-Dimethylaminoazobenzene; see 1910.1015	60-11-7			
Dimethoxymethane; see Methylal				
Dimethyl acetamide	127-19-5	10	35	X
Dimethylamine	124-40-3	10	18	—
Dimethylaminobenzene; see Xylidine				
Dimethylaniline (N,N-Dimethylaniline)	121-69-7	5	25	X
Dimethylbenzene; see Xylene				
Dimethyl-1,2-dibromo- 2,2-dichloroethyl phosphate	300-76-5	—	3	—
Dimethylformamide	68-12-2	10	30	X
2,6-Dimethyl-4-heptanone; see Diisobutyl ketone				
1,1-Dimethylhydrazine	57-14-7	0.5	1	X
Dimethylphthalate	131-11-3	—	5	—
Dimethyl sulfate	77-78-1	1	5	X
Dinitolmide (3,5-Dinitro-o-toluamide)	148-01-6	—	—	—
Dinitrobenzene (all isomers)			1	X
(ortho)	528-29-0			
(meta)	99-65-0			

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TABLE Z.—Shipyards

Substance	CAS No.[4]	Transitional Limits[5] (1970 TLVs) ppm[5]	mg/m³[5]	Skin Designation
(para)	100-25-4			
Dinitro-o-cresol	534-52-1	—	0.2	X
Dinitrotoluene	25321-14-6	—	1.5	X
Dioxane (Diethylene dioxide)	123-91-1	100	360	X
Dioxathion (Delnav)	78-34-2	—	—	—
Diphenyl (Biphenyl)	92-52-4	0.2	1	—
Diphenylamine	122-39-4	—	10	—
Diphenylmethane diisocyanate; see Methylene bisphenyl isocyanate				
Dipropylene glycol methyl ether	34590-94-8	100	600	X
Dipropyl ketone	123-19-3	—	—	—
Diquat	85-00-7	—	—	—
Di-sec octyl phthalate (Di-(2-ethylhexyl) phthalate)	117-81-7	—	5	—
Disulfiram	97-77-8	—	—	—
Disulfoton	298-04-4	—	—	—
2,6-Di-tert-butyl-p-cresol	128-37-0	—	—	—
Diuron	330-54-1	—	—	—
Divinyl benzene	1321-74-0	—	—	—
Emery	12415-34-8	—		
Total dust		—	15	—
Respirable fraction		—	5	—
Endosulfan	115-29-7	—	0.1	X
Endrin	72-20-8	—	0.1	X
Epichlorohydrin	106-89-8	5	19	X
EPN	2104-64-5	—	0.5	X
1,2-Epoxypropane; see Propylene oxide				
2,3-Epoxy-1-propanol; see Glycidol				
Ethane	74-84-0	E		
Ethanethiol; see Ethyl mercaptan				
Ethanolamine	141-43-5	3	6	—
Ethion	563-12-2	—	—	—
2-Ethoxyethanol (Cellosolve)	110-80-5	200	740	X
2-Ethoxyethyl acetate (Cellosolve acetate)	111-15-9	100	540	X
Ethyl acetate	141-78-6	400	1400	—
Ethyl acrylate	140-88-5	25	100	X
Ethyl alcohol (Ethanol)	64-17-5	1000	1900	—
Ethylamine	75-04-7	10	18	—
Ethyl amyl ketone (5-Methyl-3-heptanone)	541-85-5	25	130	—
Ethyl benzene	100-41-4	100	435	—
Ethyl bromide	74-96-4	200	890	—
Ethyl butyl ketone (3-Heptanone)	106-35-4	50	230	—
Ethyl chloride	75-00-3	1000	2600	—
Ethyl ether	60-29-7	400	1200	—
Ethyl formate	109-94-4	100	300	—
Ethyl mercaptan	75-08-1	0.5	1	—
Ethyl silicate	78-10-4	100	850	—
Ethylene	74-85-1	E		
Ethylene chlorohydrin	107-07-3	5	16	X
Ethylenediamine	107-15-3	10	25	—
Ethylene dibromide	106-93-4	(C)25	(C)190	X
Ethylene dichloride (1,2-Dichloroethane)	107-06-2	50	200	
Ethylene glycol	107-21-1	—	—	—
Ethylene glycol dinitrate	628-96-6	(C)0.2	(C)1	X
Ethylene glycol methyl acetate; see Methyl cellosolve acetate				
Ethyleneimine; see 1910.1012	151-56-4	0.5	1	X
Ethylene oxide[6]; see 1910.1047	75-21-8	(1)	(1)	(1)

oyards—Continued

Skin Designation	Proposed Final Rule Limits						
	TWA		STEL [c]		CEILING		Skin Designation
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
X	—	0.2	—	—	—	—	
X	—	1.5	—	—	—	—	X
X	25	90	—	—	—	—	X
—	—	0.2	—	—	—	—	X
—	0.2	1	—	—	—	—	X
—	—	10	—	—	—	—	—
X	100	600	150	900	—	—	—
—	50	235	—	—	—	—	X
—	—	0.5	—	—	—	—	—
—	—	5	—	10	—	—	—
—	—	2	—	—	—	—	—
—	—	0.1	—	—	—	—	X
—	—	10	—	—	—	—	—
—	10	50	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
X	—	0.1	—	—	—	—	X
X	—	0.1	—	—	—	—	X
X	2	8	—	—	—	—	X
X	—	0.5	—	—	—	—	X
—	3	8	6	15	—	—	—
—	—	0.4	—	—	—	—	—
X	200	740	—	—	—	—	X
X	100	540	—	—	—	—	X
—	400	1400	—	—	—	—	X
X	5	20	25	100	—	—	X
—	1000	1900	—	—	—	—	—
—	10	18	—	—	—	—	—
—	25	130	—	—	—	—	—
—	100	435	125	545	—	—	—
—	200	890	250	1110	—	—	—
—	50	230	—	—	—	—	—
—	1000	2600	—	—	—	—	—
—	400	1200	500	1500	—	—	—
—	100	300	—	—	—	—	—
—	0.5	1	—	—	—	—	—
—	10	85	—	—	—	—	—
X	—	—	—	—	—	—	—
X	10	25	—	—	1	3	X
—	—	—	—	—	—	—	—
—	1	4	2	8	25	190	X
X	—	—	—	—	—	—	—
—	—	—	—	0.1	50	125	X
X	—	—	—	—	—	—	—
)	1	—	5	—	—	—	—

Ethylidene chloride; see 1,1-Dichloroethane							
Ethylidene norbornene	16219-75-3	—	—				
N-Ethylmorpholine	100-74-3	20	94				X
Fenamiphos	22224-92-6	—	—				
Fensulfothion (Dasanit)	115-90-2	—	—				
Fenthion	55-38-9	—	—				
Ferbam	14484-64-1						
Total dust		—	15				
Ferrovanadium dust	12604-58-9	—	1				
Fibreus Glass							
Total dust			15				
Respirable fraction			5				
Fluorides (as F)	Varies with compound	—	2.5				
Fluorine	7782-41-4	0.1	0.2				
Fluorotrichloromethane (Trichlorofluoromethane)	75-69-4	1000	5600				
Fonofos	944-22-9	—	—				
Formaldehyde [4,b]; see 1910.1048; and 56 FR 32302	50-00-0	1	(2 ppm STEL)				
Formamide	75-12-7	—	—				
Formic acid	64-18-6	5	9				
Furfural	98-01-1	5	20				X
Furfuryl alcohol	98-00-0	50	200				
Gasoline	8006-61-9		A[a]				
Germanium tetrahydride	7782-65-2	—	—				
Glutaraldehyde	111-30-8	—	—				
Glycerin (mist)	56-81-5						
Total dust		—	15				
Respirable fraction		—	5				
Glycidol	556-52-5	50	150				
Glycol monoethyl ether; see 2-Ethoxyethanol							
Grain dust (oat, wheat, barley)		—	—				
Graphite, natural, respirable dust	7782-42-5	(3)	(3)				(3)
Graphite, synthetic							
Total dust		—	15				
Respirable fraction		—	5				
Guthion; see Azinphos methyl							
Gypsum	13397-24-5						
Total dust		—	15				
Respirable fraction		—	5				
Hafnium	7440-58-6	—	0.5				
Helium	7440-59-7	E					
Heptachlor	76-44-8	—	0.5				X
Heptane (n-Heptane)	142-82-5	500	2000				
Hexachlorobutadiene	87-68-3	—	—				
Hexachlorocyclopentadiene	77-47-4	—	—				
Hexachloroethane	67-72-1	1	10				X
Hexachloronaphthalene	1335-87-1	—	0.2				X
Hexafluoroacetone	684-16-2	—	—				
n-Hexane	110-54-3	500	1800				
Hexane isomers	Varies with compound	—	—				
2-Hexanone (Methyl n-butyl ketone)	591-78-6	100	410				
Hexone (Methyl isobutyl ketone)	108-10-1	100	410				
sec-Hexyl acetate	108-84-9	50	300				
Hexylene glycol	107-41-5	—	—				
Hydrazine	302-01-2	1	1.3				X
Hydrogen	1333-74-0	E					
Hydrogenated terphenyls	61788-32-7	—	—				
Hydrogen bromide	10035-10-6	3	10				
Hydrogen chloride	7647-01-0	(C)5	(C)7				
Hydrogen cyanide	74-90-8	10	11				X
Hydrogen fluoride (as F)	7664-39-3	3	2				
Hydrogen peroxide	7722-84-1	1	1.4				
Hydrogen selenide (as Se)	7783-07-5	0.05	0.2				

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Substance	CAS No.[d]	Transitional Limits[c] ppm[e]	(1970 TLVs) mg/m³[b]	Skin Designation
Hydrogen sulfide	7783-06-4	10	15	—
Hydroquinone	123-31-9	—	2	—
2-Hydroxypropyl acrylate	999-61-1	—	—	—
Indene	95-13-6	10	45	—
Indium and compounds (as In)	7440-74-6	—	0.1	—
Iodine	7553-56-2	(C)0.1	(C)1	—
Iodoform	75-47-8	—	—	—
Iron oxide fume	1309-37-1	—	10	—
Iron pentacarbonyl (as Fe)	13463-40-6	—	—	[g] —
Iron salts (soluble) (as Fe)	Varies with compound	—	1	—
Isoamyl acetate	123-92-2	100	525	—
Isoamyl alcohol (primary and secondary)	123-51-3	100	360	—
Isobutyl acetate	110-19-0	150	700	—
Isobutyl alcohol	78-83-1	100	300	—
Isooctyl alcohol	26952-21-6	—	—	—
Isophorone	78-59-1	25	140	—
Isophorone diisocyanate	4098-71-9	—	—	—
2-Isopropoxyethanol	109-59-1	—	—	—
Isopropyl acetate	108-21-4	250	950	—
Isopropyl alcohol	67-63-0	400	980	—
Isopropylamine	75-31-0	5	12	—
N-Isopropylaniline	768-52-5	—	—	—
Isopropyl ether	108-20-3	500	2100	—
Isopropyl glycidyl ether (IGE)	4016-14-2	50	240	—
Kaolin	1332-58-7			
Total dust		—	15	—
Respirable fraction		—	5	—
Ketene	463-51-4	0.5	0.9	—
Lead, inorganic (as Pb) [f]; see 1910.1025	7439-92-1	—	0.05	—
Limestone	1317-65-3	—		
Total dust		—	15	—
Respirable fraction		—	5	—
Lindane	58-89-9	—	0.5	X
Lithium hydride	7580-67-8	—	0.025	—
L.P.G. (Liquefied petroleum gas)	68476-85-7	1000	1800	—
Magnesite	546-93-0			
Total dust		—	15	—
Respirable fraction		—	5	—
Magnesium oxide fume	1309-48-4			
Total particulate			15	—
Malathion	121-75-5			
Total dust		—	15	X
Maleic anhydride	108-31-6	0.25	1	—
Manganese compounds (as Mn)	7439-96-5	—	(C)5	—
Manganese fume (as Mn)	7439-96-5	—	(C)5	—
Manganese cyclopentadienyl tricarbonyl (as Mn)	12079-65-1	—	—	—
Manganese tetroxide (as Mn)	1317-35-7	—	—	—
Marble	1317-65-3	—		
Total dust		—	15	—
Respirable fraction		—	5	—
Mercury (aryl and inorganic)(as Hg)	7439-97-6	—	0.1	X
Mercury (organo) alkyl compounds (as Hg)	7439-97-6	—	0.01	X
Mercury (vapor) (as Hg)	7439-97-6	—	0.1	X
Mesityl oxide	141-79-7	25	100	—
Methacrylic acid	79-41-4	—	—	—
Methane	74-82-8	E		

yards—Continued

n g- on	Proposed Final Rule Limits						
	TWA		STEL [c]		CEILING		Skin Desig- nation
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
—	10	14	15	21	—	—	—
—	—	2	—	—	—	—	—
—	0.5	3	—	—	—	—	X
—	10	45	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	—	—	—	0.1	1	—
—	0.6	10	—	—	—	—	—
—	—	10	—	—	—	—	—
—	0.1	0.8	0.2	1.6	—	—	—
—	—	1	—	—	—	—	—
—	100	525	—	—	—	—	—
—	100	360	125	450	—	—	—
—	150	700	—	—	—	—	—
—	50	150	—	—	—	—	—
—	50	270	—	—	—	—	X
—	4	23	—	—	—	—	—
—	0.005	—	0.02	—	—	—	X
—	25	105	—	—	—	—	—
—	250	950	310	1185	—	—	—
—	400	980	500	1225	—	—	—
—	5	12	10	24	—	—	—
—	2	10	—	—	—	—	X
—	500	2100	—	—	—	—	—
—	50	240	75	360	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	0.5	0.9	1.5	3	—	—	—
—	—	0.05	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
X	—	0.5	—	—	—	—	X
—	—	0.025	—	—	—	—	—
—	1000	1800	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	10	—	—	—	—	—
X	—	10	—	—	—	—	X
—	0.25	1	—	—	—	—	—
—	—	—	—	—	—	5	—
—	—	1	—	3	—	—	—
—	—	0.1	—	—	—	—	X
—	—	1	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
X	—	—	—	—	—	0.1	X
X	—	0.01	—	0.03	—	—	X
X	—	0.05	—	—	—	—	X
—	15	60	25	100	—	—	—
—	20	70	—	—	—	—	X

Methanethiol; see Methyl mercaptan				
Methomyl (Lannate)	16752-77-5	—	—	—
Methoxychlor	72-43-5			
Total dust		—	15	—
2-Methoxyethanol (Methyl cellosolve)	109-86-4	25	80	X
2-Methoxyethanol acetate (Methyl cellosolve acetate)	110-49-6	25	120	X
4-Methoxyphenol	150-76-5	—	—	—
Methyl acetate	79-20-9	200	610	—
Methyl acetylene (Propyne)	74-99-7	1000	1650	—
Methyl acetylene-propadiene mixture (MAPP)		1000	1800	—
Methyl acrylate	96-33-3	10	35	X
Methylacrylonitrile	126-98-7	—	—	—
Methylal (Dimethoxy-methane)	109-87-5	1000	3100	—
Methyl alcohol	67-56-1	200	260	—
Methylamine	74-89-5	10	12	—
Methyl amyl alcohol; see Methyl isobutyl carbinol				
Methyl n-amyl ketone	110-43-0	100	465	—
Methyl bromide	74-83-9	(C)20	(C)80	X
Methyl butyl ketone; see 2-Hexanone				
Methyl cellosolve; see 2-Methoxyethanol				
Methyl cellosolve acetate; see 2-Methoxyethyl acetate				
Methyl chloride	74-87-3	100	210	—
Methyl chloroform (1,1,1-Trichloroethane)	71-55-6	350	1900	—
Methyl 2-cyanoacrylate	137-05-3	—	—	—
Methylcyclohexane	108-87-2	500	2000	—
Methylcyclohexanol	25639-42-3	100	470	—
o-Methylcyclohexanone	583-60-8	100	460	X
Methylcyclopentadienyl manganese tricarbonyl (as Mn)	12108-13-3	—	—	—
Methyl demeton	8022-00-2	—	—	—
4,4'-Methylene bis (2-chloroaniline) (MBOCA)	101-14-4	—	—	—
Methylene bis(4-cyclohexylisocyanate)	5124-30-1	—	—	—
Methylene chloride [b]; see 56 FR 57036	75-09-2	500	1740	—
Methyl ethyl ketone (MEK); see 2-Butanone				
Methyl ethyl ketone peroxide (MEKP)	1338-23-4	—	—	—
Methyl formate	107-31-3	100	250	—
Methyl hydrazine (Monomethyl hydrazine)	60-34-4	(C)0.2	(C)0.35	X
Methyl iodide	74-88-4	5	28	X
Methyl isoamyl ketone	110-12-3	100	475	—
Methyl isobutyl carbinol	108-11-2	25	100	X
Methyl isobutyl ketone; see Hexone				
Methyl isocyanate	624-83-9	0.02	0.05	X
Methyl isopropyl ketone	563-80-4	—	—	—
Methyl mercaptan	74-93-1	0.5	1	—
Methyl methacrylate	80-62-6	100	410	100
Methyl parathion	298-00-0	—	—	—
Methyl propyl ketone; see 2-Pentanone				
Methyl silicate	681-84-5	5	30	—
alpha-Methyl styrene	98-83-9	(C)100	(C)480	—
Methylene bisphenyl isocyanate (MDI)	101-68-8	(C)0.02	(C)0.2	—
Metribuzin	21087-64-9	—	—	—
Mica; see Silicates				
Mineral wool				
Total dust		—	15	—
Respirable dust		—	5	—
Molybdenum (as Mo)	7439-98-7			
Soluble compounds		—	5	—
Insoluble compounds				
Total dust		—	15	—
Monocrotophos (Azodrin)	6923-22-4	—	—	—
Monomethyl aniline	100-61-8	2	9	X
Monomethyl hydrazine; see Methyl hydrazine				
Morpholine	110-91-8	20	70	X
Naphtha (Coal tar)	8030-30-6	100	400	—

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TABLE Z.—Shipyards-

Substance	CAS No.[4]	Transitional Limits[a] ppm[a]	(1970 TLVs) mg/m³[b]	Skin Designation
Naphthalene	91-20-3	10	50	—
alpha-Naphthylamine; see 1910.1004	134-32-7			
beta-Naphthylamine; see 1910.1009	91-59-8		A[1]	
Neon	7440-01-9	E		
Nickel carbonyl (as Ni)	13463-39-3	0.001	0.007	—
Nickel, metal and insoluble compounds (as Ni)	7440-02-0	—	1	—
Nickel, soluble compounds (as Ni)	7440-02-0	—	1	—
Nicotine	54-11-5	—	0.5	X
Nitric acid	7697-37-2	2	5	—
Nitric oxide	10102-43-9	25	30	—
p-Nitroaniline	100-01-6	1	6	X
Nitrobenzene	98-95-3	1	5	X
p-Nitrochlorobenzene	100-00-5	—	1	X
4-Nitrodiphenyl; see 1910.1003	92-93-3			
Nitroethane	79-24-3	100	310	—
Nitrogen	7727-37-9	E		
Nitrogen dioxide	10102-44-0	(C)5	(C)9	—
Nitrogen trifluoride	7783-54-2	10	29	—
Nitroglycerin	55-63-0	(C)0.2	(C)2	X
Nitromethane	75-52-5	100	250	—
1-Nitropropane	108-03-2	25	90	—
2-Nitropropane	79-46-9	25	90	—
N-Nitrosodimethylamine; see 1910.1016	62-79-9	A1		
Nitrotoluene (all isomers)		5	30	X
o-isomer	88-72-2;			
m-isomer	99-08-1;			
p-isomer	99-99-0			
Nitrotrichloromethane; see Chloropicrin				
Nitrous oxide	10024-97-2	E		
Nonane	111-84-2	—	—	—
Octachloronaphthalene	2234-13-1	—	0.1	X
Octane	111-65-9	400	1900	—
Oil mist, mineral	8012-95-1	—	5	—
Osmium tetroxide (as Os)	20816-12-0	—	0.002	—
Oxalic acid	144-62-7	—	1	—
Oxygen difluoride	7783-41-7	0.05	0.1	—
Ozone	10028-15-6	0.1	0.2	—
Paraffin wax fume	8002-74-2	—	—	—
Paraquat, respirable dust	4685-14-7; 1910-42-5; 2074-50-2	—	0.5	X
Parathion	56-38-2	—	0.1	X
Particulates not otherwise regulated				
Total dust		—	15	—
Respirable fraction		—	5	—
PCB; See chlorodiphenyl (42% and 54% chlorine)				
Pentaborane	19624-22-7	0.005	0.01	—
Pentachloronaphthalene	1321-64-8	—	0.5	X
Pentachlorophenol	87-86-5	—	0.5	X
Pentaerythritol	115-77-5	—		
Total dust		—	15	—
Respirable fraction		—	5	—
Pentane	109-66-0	500	1500	—
2-Pentanone (Methyl propyl ketone)	107-87-9	200	700	—
Perchloroethylene (Tetrachloroethylene)	127-18-4	100	670	—
Perchloromethyl mercaptan	594-42-3	0.1	0.8	—
Perchloryl fluoride	7616-94-6	3	13.5	—

ards—Continued

	Proposed Final Rule Limits						
	TWA		STEL [c]		CEILING		Skin Designation
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
—	10	50	15	75	—	—	—
—	0.001	0.007	—	—	—	—	—
—	—	1	—	—	—	—	—
—	—	0.1	—	—	—	—	—
X	—	0.5	—	—	—	—	X
—	2	5	4	10	—	—	—
—	25	30	—	—	—	—	—
X	—	3	—	—	—	—	X
X	1	5	—	—	—	—	X
X	—	1	—	—	—	—	X
—	100	310	—	—	—	—	—
—	—	—	1	1.0	—	—	—
—	10	29	—	—	—	—	—
X	—	—	—	0.1	—	—	X
—	100	250	—	—	—	—	—
—	25	90	—	—	—	—	—
—	10	35	—	—	—	—	—
X	2	11	—	—	—	—	X
—	200	1050	—	—	—	—	—
X	—	0.1	—	0.3	—	—	X
—	300	1450	375	1800	—	—	—
—	—	5	—	—	—	—	—
—	0.0002	0.002	0.0006	0.006	—	—	—
—	—	1	—	2	—	—	—
—	—	—	—	—	0.05	0.1	—
—	0.1	0.2	0.3	0.6	—	—	—
—	—	2	—	—	—	—	—
X	—	0.1	—	—	—	—	X
X	—	0.1	—	—	—	—	X
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	0.005	0.01	0.015	0.03	—	—	—
X	—	0.5	—	—	—	—	X
X	—	0.5	—	—	—	—	X
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	500	1900	750	2250	—	—	—
—	200	700	250	875	—	—	—
—	25	170	—	—	—	—	—
—	0.1	0.8	—	—	—	—	—
—	3	14	6	28	—	—	—

Perlite	93763-70-3			
Total dust		—	15	
Respirable fraction		—	5	
Petroleum distillates (Naphtha)(Rubber Solvent)			A[4]	
Phenol	108-95-2	5	19	X
Phenothiazine	92-84-2	—	—	
p-Phenylene diamine	106-50-3	—	0.1	X
Phenyl ether, vapor	101-84-8	1	7	
Phenyl ether-biphenyl mixture, vapor		1	7	
Phenylethylene; see Styrene				
Phenyl glycidyl ether (PGE)	122-60-1	10	60	
Phenylhydrazine	100-63-0	5	22	X
Phenyl mercaptan	108-98-5	—	—	
Phenylphosphine	638-21-1	—	—	
Phorate	298-02-2	—	—	
Phosdrin (Mevinphos)	7786-34-7	—	0.1	X
Phosgene (Carbonyl chloride)	75-44-5	0.1	0.4	
Phosphine	7803-51-2	0.3	0.4	
Phosphoric acid	7664-38-2	—	1	
Phosphorus (yellow)	7723-14-0	—	0.1	
Phosphorus oxychloride	10025-87-3	—	—	
Phosphorus pentachloride	10026-13-8	—	1	
Phosphorus pentasulfide	1314-80-3	—	1	
Phosphorus trichloride	7719-12-2	0.5	3	
Phthalic anhydride	85-44-9	2	12	
m-Phthalodinitrile	626-17-5	—	—	
Picloram	1918-02-1			
Total dust		—	15	
Respirable fraction		—	5	
Picric acid	88-89-1	—	0.1	X
Piperazine dihydrochloride	142-64-3	—	—	
Pindone (2-Pivalyl-1,3-indandione)	83-26-1	—	0.1	
Plaster of Paris	26499-65-0			
Total dust		—	15	
Respirable fraction		—	5	
Platinum (as Pt)	7440-06-4			
Metal		—	—	
Soluble salts		—	0.002	
Polytetrafluoroethylene decomposition products			A[4]	
Portland cement	65997-15-1			
Total dust		15	—	10
Respirable fraction		5	—	
Potassium hydroxide	1310-58-3	—	—	
Propargyl alcohol	107-19-7	1	—	X
beta-Propriolactone; see 1910.1013	57-57-8		A[1]	
Propionic acid	79-09-4	—	—	
Propoxur (Baygon)	114-26-1	—	—	
n-Propyl acetate	109-60-4	200	840	
n-Propyl alcohol	71-23-8	200	500	
n-Propyl nitrate	627-13-4	25	110	
Propylene dichloride	78-87-5	75	350	
Propylene glycol dinitrate	6423-43-4	—	—	
Propylene glycol monomethyl ether	107-98-2	—	—	
Propylene imine	75-55-8	2	5	X
Propylene oxide	75-56-9	100	240	
Propyne; see Methyl acetylene				
Pyrethrum	8003-34-7	—	5	
Pyridine	110-86-1	5	15	
Quinone	106-51-4	0.1	0.4	
RDX; see Cyclonite				
Resorcinol	108-46-3	—	—	
Rhodium (as Rh), metal fume and insoluble compounds	7440-16-6	—	0.1	
Rhodium (as Rh), soluble compounds	7440-16-6	—	0.001	
Ronnel	299-84-3	—	10	

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Substance	CAS No.[4]	Transitional Limits[5] (1970 TLVs) ppm[6]	mg/m³[6b]	Skin Designation
Rosin core solder pyrolysis products, as formaldehyde		—	—	—
Rotenone	83-79-4	—	5	—
Rouge		—		
Total dust		—	15	—
Respirable fraction		—	5	—
Selenium compounds (as Se)	7782-49-2	—	0.2	—
Selenium hexafluoride (as Se)	7783-79-1	0.05	0.4	—
Silica, amorphous, precipitated and gel	112926-00-8	[3]	[3]	[3]
Silica, amorphous, diatomaceous earth, containing less than 1% crystalline silica	61790-53-2	[3]	[3]	[3]
Silica, crystalline cristobalite, respirable dust	14464-46-1	[3]	[3]	[3]
Silica, crystalline quartz, respirable dust	14808-60-7	[3]	[3]	[3]
Silica, crystalline tripoli (as quartz), respirable dust	1317-95-9	[3]	[3]	[3]
Silica, crystalline tridymite, respirable dust	15468-32-3	[3]	[3]	[3]
Silica, fused, respirable dust	60676-86-0	[3]	[3]	[3]
Silicates (less than 1% crystalline silica)				
Mica (respirable dust)	12001-26-2	[3]	[3]	[3]
Soapstone, total dust		[3]	[3]	[3]
Soapstone, respirable dust		[3]	[3]	[3]
Talc (containing asbestos): use asbestos limit 1910.1001		[3]	[3]	[3]
Talc (containing no asbestos), respirable dust	14807-96-6	[3]	[3]	[3]
Tremolite		[1]	[1]	[1]
Silicon	7440-21-3			
Total dust		—	15	—
Respirable fraction		—	5	—
Silicon carbide	409-21-2			
Total dust		—	15	—
Respirable fraction		—	5	—
Silicon tetrahydride	7803-62-5	—	—	—
Silver, metal and soluble compounds (as Ag)	7440-22-4	—	0.01	—
Soapstone; see Silicates				
Sodium azide	26628-22-8			
(as HN_3)		—	—	—
(as NaN_3)		—	—	—
Sodium bisulfite	7631-90-5	—	—	—
Sodium fluoroacetate	62-74-8	—	0.05	X
Sodium hydroxide	1310-73-2	—	2	—
Sodium metabisulfite	7681-57-4	—	—	—
Starch	9005-25-8	—		
Total dust		—	15	—
Respirable fraction		—	5	—
Stibine	7803-52-3	0.1	0.5	—
Stoddard solvent	8052-41-3	200	1150	—
Strychnine	57-24-9	—	0.15	—
Styrene	100-42-5	100	420	50
Subtilisins (Proteolytic enzymes)	9014-01-1	—	—	—
Sucrose	57-50-1			
Total dust		—	15	—
Respirable fraction		—	5	—
Sulfur dioxide	7446-09-5	5	13	—
Sulfur hexafluoride	2551-62-4	1000	6000	—
Sulfuric acid	7664-93-9	—	1	—
Sulfur monochloride	10025-67-9	1	6	—

yards—Continued

n g- on	Proposed Final Rule Limits						Skin Desig-nation
	TWA		STEL [c]		CEILING		
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
—	—	0.1	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	0.2	—	—	—	—	—
—	0.05	0.4	—	—	—	—	—
(3)	—	6	—	—	—	—	—
(3)	—	6	—	—	—	—	—
(3)	—	0.05	—	—	—	—	—
(3)	—	0.1	—	—	—	—	—
(3)	—	0.1	—	—	—	—	—
(3)	—	0.05	—	—	—	—	—
(3)	—	0.1	—	—	—	—	—
(3)	—	3	—	—	—	—	—
(3)	—	6	—	—	—	—	—
(3)	—	3	—	—	—	—	—
(3)	—	0.2 f/cc	—	1 f/cc (30 min)	—	—	—
(3)	—	2	—	—	—	—	—
(1)	(4)	(4)	(4)	(4)	(4)	(4)	(4)
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	5	7	—	—	—	—	—
—	—	0.01	—	—	—	—	—
—	—	—	—	0.1	—	X	
—	—	—	—	—	0.3	X	
—	—	5	—	—	—	—	—
X	—	0.05	—	0.15	—	—	X
—	—	—	—	—	—	2	—
—	—	5	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	0.1	0.5	—	—	—	—	—
—	100	525	—	—	—	—	—
—	—	0.15	—	—	—	—	—
50	215	100	425	—	—	—	—
—	—	—	—	0.00006 [e] (60 min)	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	2	5	5	13	—	—	—
—	1000	6000	—	—	—	—	—
—	—	1	—	—	—	—	—
—	—	—	—	—	1	6	—

Sulfur pentafluoride	5714-22-7	0.025	0.25	—
Sulfur tetrafluoride	7783-60-0	—	—	—
Sulfuryl fluoride	2699-79-8	5	20	—
Sulprofos	35400-43-2	—	—	—
Systox, see Demeton				
2,4,5-T (2,4,5-Trichlorophenoxyacetic acid)	93-76-5	—	10	—
Talc; see Silicates				
Tantalum, metal and oxide dust	7440-25-7	—	5	—
TEDP (Sulfotep)	3689-24-5	—	0.2	X
Teflon decomposition products			A[a]	
Tellurium and compounds (as Te)	13494-80-9	—	0.1	—
Tellurium hexafluoride (as Te)	7783-80-4	0.02	0.2	—
Temephos	3383-96-8			
Total dust		—	15	—
Respirable fraction		—	5	—
TEPP (Tetraethyl pyrophosphate)	107-49-3	—	0.05	X
Terphenyls	26140-60-3	(C)1	(C)9	—
1,1,1,2-Tetrachloro-2,2-difluoroethane	76-11-9	500	4170	—
1,1,2,2-Tetrachloro-1,2-difluoroethane	76-12-0	500	4170	—
1,1,2,2-Tetrachloroethane	79-34-5	5	35	X
Tetrachloroethylene; see Perchloroethylene				
Tetrachloromethane; see Carbon tetrachloride				
Tetrachloronaphthalene	1335-88-2	—	2	X
Tetraethyl lead (as Pb)	78-00-2	—	0.1	X
Tetrahydrofuran	109-99-9	200	590	—
Tetramethyl lead, (as Pb)	75-74-1	—	0.15	X
Tetramethyl succinonitrile	3333-52-6	0.5	2	X
Tetranitromethane	509-14-8	1	8	—
Tetrasodium pyrophosphate	7722-88-5	—	—	—
Tetryl (2,4,6-Trinitrophenylmethylnitramine)	479-45-8	—	1.5	X
Thallium, soluble compounds (as Tl)	7440-28-0	—	0.1	X
4,4'-Thiobis (6-tert, Butyl-m-cresol)	96-69-5			
Total dust		—	15	—
Respirable fraction		—	2	—
Thioglycolic acid	68-11-1	—	—	—
Thionyl chloride	7719-09-7	—	—	—
Thiram	137-26-8	—	5	—
Tin, inorganic compounds (except oxides) (as Sn)	7440-31-5	—	2	—
Tin, organic compounds (as Sn)	7440-31-5	—	0.1	—
Tin oxide (as Sn)	21651-19-4	—	—	—
Total dust		—	15	—
Respirable fraction		—	5	—
Titanium dioxide	13463-67-7			
Total dust		—	15	—
Toluene	108-88-3	200	750	100
Toluene-2,4-diisocyanate (TDI)	584-84-9	(C)0.02	(C)0.14	—
m-Toluidine	108-44-1	—	—	—
o-Toluidine	95-53-4	5	22	X
p-Toluidine	106-49-0	—	—	—
Toxaphene; see Chlorinated camphene				
Tremolite; see Silicates				
Tributyl phosphate	126-73-8	—	5	—
Trichloroacetic acid	76-03-9	—	—	—
1,2,4-Trichlorobenzene	120-82-1	—	—	—
1,1,1-Trichloroethane; see Methyl chloroform				
1,1,2-Trichloroethane	79-00-5	10	45	X
Trichloroethylene	79-01-6	100	535	—
Trichloromethane; see Chloroform				
Trichloronaphthalene	1321-65-9	—	5	X
1,2,3-Trichloropropane	96-18-4	50	300	—
1,1,2-Trichloro-1,2,2-trifluoroethane	76-13-1	1000	7600	—
Triethylamine	121-44-8	25	100	—
Trifluorobromomethane	75-63-8	1000	6100	—
Trimellitic anhydride	552-30-7	—	—	—
Trimethylamine	75-50-3	—	—	—
Trimethyl benzene	25551-13-7	25	120	—

| | | |

TABLE Z.—Shipyards

Substance	CAS No.[4]	Transitional Limits* (1970 TLVs) ppm[6]	mg/m[3] **	Skin Designation
Trimethyl phosphite	121-45-9	—	—	—
2,4,6-Trinitrophenyl; see Picric acid				
2,4,6-Trinitrophenylmethylnitramine; see Tetryl				
2,4,6-Trinitrotoluene (TNT)	118-96-7	—	1.5	X
Triorthocresyl phosphate	78-30-8	—	0.1	—
Triphenyl amine	603-34-9	—	—	—
Triphenyl phosphate	115-86-6	—	5	—
Tungsten (as W)	7440-33-7			
Insoluble compounds		—	5	—
Soluble compounds		—	1	—
Turpentine	8006-64-2	100	560	—
Uranium (as U)	7440-61-1			
Soluble compounds		—	0.2	—
Insoluble compounds		—	0.2	—
n-Valeraldehyde	110-62-3	—	—	—
Vanadium	1314-62-1			
Respirable dust (as V_2O_5)		—	(C)0.5	—
Fume (as V_2O_5)		—	(C)0.1	—
Vegetable oil mist				
Total dust		—	15	—
Respirable fraction		—	5	—
Vinyl acetate	108-05-4	—	—	—
Vinyl benzene; see Styrene				
Vinyl bromide	593-60-2	—	—	—
Vinyl chloride[5]; see 1910.1017	75-01-4	(5)	(5)	(5)
Vinyl cyanide; see Acrylonitrile				
Vinyl cyclohexene dioxide	106-87-6	—	—	—
Vinylidene chloride (1,1-Dichloroethylene)	75-35-4	—	—	—
Vinyl toluene	25013-15-4	100	480	—
VM & P Naphtha	8032-32-4	—	—	—
Warfarin	81-81-2	—	0.1	—
Welding fumes (total particulate)**		—	—	—
Wood dust, all soft and hard woods, except Western red cedar		—	—	—
Wood dust, Western red cedar		—	—	—
Xylenes (o-, m-, p- isomers)	1330-20-7	100	435	—
m-Xylene alpha, alpha, diamine	1477-55-0	—	—	—
Xylidine	1300-73-8	5	25	X
Yttrium	7440-65-5	—	1	—
Zinc chloride fume	7646-85-7	—	1	—
Zinc chromate (as CrO_3) Varies with Compound	13530-65-9	—	—	—
Zinc oxide fume	1314-13-2	—	5	—
Zinc oxide	1314-13-2			
Total dust		—	15	—
Respirable fraction		—	5	—
Zinc stearate	557-05-1			
Total dust		—	15	—
Respirable fraction		—	5	—
Zirconium compounds (as Zr)	7440-67-7	—	5	—

Footnotes:
[1] Same as Final Rule Limits.
[2] See Mineral Dusts Table.
[3] Use Asbestos Limit 1910.1001.
[4] See 1910.1001 for asbestiform; for non-asbestiform see 1910.1101.
* The transitional PELs are 8-hour TWAs unless otherwise noted; a (C) denotes a ceiling limit.
** As determined from breathing-zone air samples.
[6] Parts of vapor or gas per million parts of contaminated air by volume at 25°C and 760 torr.

ards—Continued

	Proposed Final Rule Limits						
	TWA		STEL [c]		CEILING		Skin Designation
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
	2	10	—	—	—	—	—
X	—	0.5	—	—	—	—	X
—	—	0.1	—	—	—	—	X
—	—	5	—	—	—	—	—
—	—	3	—	—	—	—	—
—	—	5	—	10	—	—	—
—	—	1	—	3	—	—	—
—	100	560	—	—	—	—	—
—	—	0.05	—	—	—	—	—
—	—	0.2	—	0.6	—	—	—
—	50	175	—	—	—	—	—
—	—	0.05	—	—	—	—	—
—	—	0.05	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	10	30	20	60	—	—	—
—	5	20	—	—	—	—	—
C	1	—	5	—	—	—	—
—	10	60	—	—	—	—	X
—	1	4	—	—	—	—	—
—	100	480	—	—	—	—	—
—	300	1350	400	1800	—	—	—
—	—	0.1	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	5	—	10	—	—	—
—	—	2.5	—	—	—	—	—
—	100	435	150	655	—	—	—
—	—	—	—	—	—	0.1	X
X	2	10	—	—	—	—	X
—	—	1	—	—	—	—	—
—	—	1	—	2	—	—	—
—	0.1	—	—	—	—	—	—
—	—	5	—	10	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	5	—	10	—	—	—

[b] Milligrams of substance per cubic meter of air. When entry is in this column only, the value is exact; when listed v
[c] Duration is for 15 minutes, unless otherwise noted.
[d] The CAS number is for information only. Enforcement is based on the substance name. For an entry covering m
not CAS numbers for the individual compounds.
[e] Compliance with the subtilisins PEL is assessed by sampling with a high volume sampler (600–800 liters per minu
[f] For those substances for which a full standard exists, exposure limits are provided in the table for informatio
[g] For sectors excluded from 1910.1028 the limit is 10 ppm and see 1910.1000, Table Z–2 for ceiling limits.
[h] Where OSHA has published a proposal for a substance separately from this rulemaking but has not issued a
columns. The proposed new limit is published in the Final Rule Limits columns. However, the proposed new limit
rulemaking.

The 1970 TLVs use letter designations instead of a numerical value as follows:

A[1] Because of the high incidence of cancer, either in humans or in animals, no exposure or contact by any route, r
A[2] Polytetrafluoroethylene decomposition products. Because these products decompose in part by hydrolysis in
exposure. No TLV is recommended pending determination of the toxicity of the products, but air concentrations shou
A[3] Gasoline and/or Petroleum Distillates. The composition of these materials varies greatly and thus a single TLV
and additives should be determined to arrive at the appropriate TLV.
E Simple asphyxiant.

isted with a ppm entry, it is approximate.

ring more than one metal compound, measured as the metal, the CAS number for the metal is given--

r minute) for at least 60 minutes.
rmational purposes, and the binding requirements are as set forth in the single-substance standard.

ed a final rule, the proposal is referenced and the existing limit is published in the Transitional Limits
limit is presented just for information purposes, and the substances are not being considered in this

oute, respiratory, oral or skin should be permitted.
sis in alkaline solution, they can be quantitatively determined in air as fluoride to provide an index of
should be minimal.
e TLV for all types of these materials is no longer applicable. The content of benzene, other aromatics

II. OSHA proposes to amend 29 CFR Part 1917 as follows:

PART 1917—MARINE TERMINALS

1. It is proposed to revise the authority citation for part 1917 to read as follows:

Authority: Sec. 41, Longshore and Harbor Workers Compensation Act (33 U.S.C. 941); secs. 4, 6, 8, Occupational Safety and Health Act of 1970 (29 U.S.C. 653, 655, 657); Secretary of Labor's Order No. 12–71 (36 FR 8754), 8–76 (41 FR 25059), 9–83 (48 FR 35736) or 1–90 (55 FR 9033), as applicable; 29 CFR Part 1911.

Section 1917.28 also issued under 5 U.S.C. 553.

2. It is proposed to amend § 1917.1 by revising paragraph (a)(2)(ii) as follows:

§ 1917.1 Scope and applicability.

(a) * * *
(2) * * *
(i) * * *

(ii) Toxic and hazardous substances. 29 CFR 1917, Subpart Z applies generally except when a substance or cargo is contained within a sealed intact means of packaging or containment complying with Department of Transportation or International Maritime Organization requirements; [1]

* * * * *

3. It is proposed to amend § 1917.2 by revising paragraph (p)(1) as follows:

§ 1917.2 Definitions.

* * * * *

(p) *Hazardous cargo, material, substance or atmosphere* means:

(1) Any substance listed in 29 CFR 1917, subpart Z. A hazardous atmosphere, or hazardous level of a hazardous cargo, material, substance or atmosphere or a hazardous vapor concentration exists for any substance covered by 29 CFR 1917 or subpart Z when the exposure level in the marine terminal workplace is above the permissible exposure limits specified in 29 CFR 1917, subpart Z;

* * * * *

4. It is proposed to amend § 1917.23 by revising paragraph (a) to read as follows:

§ 1917.23 Hazardous atmospheres and substances. (See § 1917.2 (p)).

(a) *Purpose and scope.* This section covers areas in which the employer is aware that a hazardous atmosphere or substance may exist except that 29 CFR 1917, subpart Z always applies pursuant to its terms, and except that paragraphs (b) through (e) of this section do not apply where one or more of the following sections apply: Section 1917.22 *Hazardous cargo;* § 1917.24 *Carbon monoxide;* § 1917.25 *Fumigants, pesticides, insecticides and hazardous preservatives;* § 1917.73 *Menhaden terminals;* § 1917.152 *Welding, cutting, and heating (hot work);* and § 1917.153 *Spray painting.*

* * * * *

5. It is proposed to amend § 1917.24 by revising paragraph (a) as follows:

§ 1917.24 Carbon monoxide.

(a) Exposure limits. The carbon monoxide content of the atmosphere in a compartment room, building, vehicle, railcar or any enclosed space shall be maintained at not more than 35 ppm (0.0035%) as an 8-hour time weighted average and employees shall be removed from the enclosed space if the carbon monoxide concentration exceeds 100 parts per million (0.01%). The short term exposure limit in outdoors non-enclosed spaces shall be 200 ppm (0.02%) measured over a 5 minute period.

* * * * *

Subparts H–Y [Reserved]

6. It is proposed to reserve Subparts H–Y and to add a new Subpart Z consisting of § 1917.1000 to read as follows:

Subpart Z—Toxic and Hazardous Substances

§ 1917.1000 Air Contaminants.

(a) *Exposure limits.* An employee's exposure to any substance listed in Table Z, Longshoring and Marine Terminals shall be limited in accordance with the requirements of the following paragraphs of this section:

(1) Final Rule Limits Columns. An employee's exposure to any substance listed in Table Z, Longshoring and Marine Terminals shall not exceed the Time Weighted Average (TWA), Short Term Exposure Limit (STEL) and Ceiling Limit specified for that substance in Table Z, Longshoring and Marine Terminals under the Final Rule Limits columns.

(2) Skin Designation. To prevent or reduce skin absorption, an employee's skin exposure to substances listed in Table Z, Longshoring and Marine Terminals with an "X" in one or both of the Skin Designation columns following the substance name shall be prevented or reduced to the extent necessary in the circumstances through the use of gloves, coveralls, goggles, or other appropriate personal protective equipment, engineering controls or work practices.

(3) Definitions. The following definitions are applicable to Table Z, Longshoring and Marine Terminals:

(i) *Time weighted average (TWA)* is the employee's average airborne exposure in any 8-hour work shift of a 40-hour work week which shall not be exceeded.

(ii) *Short term exposure limit (STEL)* is the employee's 15-minute time weighted average exposure which shall not be exceeded at any time during a work day unless another time limit is specified in a parenthetical notation below the limit. If another time period is specified, the time weighted average exposure over that time period shall not be exceeded at any time during the working day.

(iii) *Ceiling* is the employee's exposure which shall not be exceeded during any part of the work day. If instantaneous monitoring is not feasible, then the ceiling shall be assessed as a 15-minute time weighted average exposure which shall not be exceeded at any time over a working day.

(b) *Computation formulae.* The computation formula which shall apply to employee exposure to more than one substance for which 8-hour time weighted averages are listed in subpart Z in order to determine whether an employee is exposed over the regulatory limit is as follows:

(1)(i) The cumulative exposure for an 8-hour work shift shall be computed as follows:

$$E=(C_aT_a+C_bT_b+\ldots C_nT_n)\div 8$$

Where:

E is the equivalent exposure for the working shift.

C is the concentration during any period of time T where the concentration remains constant.

T is the duration in hours of the exposure at the concentration C.

The value of E shall not exceed the 8-hour time weighted average specified in subpart Z or 29 CFR part 1917 for the material involved.

(ii) To illustrate the formula prescribed in paragraph (d)(1)(i) of this section, assume that Substance A has an 8-hour time weighted average limit of 100 ppm noted in Table Z. Assume that an employee is subject to the following exposure:

Two hours exposure at 150 ppm
Two hours exposure at 75 ppm
Four hours exposure at 50 ppm

Substituting this information in the formula, we have

$$\{2\times 150+2\times 75+4\times 50\}\div 8=81.25 \text{ ppm}$$

Since 81.25 ppm is less than 100 ppm, the 8-hour time weighted average limit, the exposure is acceptable.

[1] The International Maritime Organization publishes the International Maritime Dangerous Goods Code to aid compliance with the International legal requirements of the International Convention for the Safety of Life at Sea, 1960.

(2)(i) In case of a mixture of air contaminants an employer shall compute the equivalent exposure as follows:

$E_m = (C_A \div L_A + C_B \div L_B) + \ldots (C_n \div L_n)$

Where:

E_m is the equivalent exposure for the mixture.

C is the concentration of a particular contaminant.

L is the exposure limit for that substance specified in subpart Z of 29 CFR part 1917.

The value of E_m shall not exceed unity (1).

(ii) To illustrate the formula prescribed in paragraph (d)(2)(i) of this section, consider the following exposures:

Substance	Actual concentration of 8-hour exposure (ppm)	8-hr. TWA PEL (ppm)
B	500	1,000
C	45	200
D	40	200

Substituting in the formula we have:

$E_m = 500 \div 1,000 + 45 \div 200 + 40 \div 200$

$E_m = 0.500 + 0.225 + 0.200$

$E_m = 0.925$

Since E_m is less than unity (1), the exposure combination is within acceptable limits.

(c) *Methods of compliance.* To achieve compliance with paragraph (a) of this section administrative or engineering controls must first be implemented whenever feasible. When such controls are not feasible to achieve full compliance, protective equipment or other protective measures shall be used to keep the exposure of employees to air contaminants within the limits prescribed in this section. Any equipment and technical measures used for this purpose must first be approved for each particular use by a competent industrial hygienist or other technically qualified person. Whenever respirators are used, their use shall comply with § 1917.92 which cross references 29 CFR 1910.134.

(d) *Sealed containers.* (1) When a substance or cargo is contained within a sealed, intact means of packaging or containment complying with Department of Transportation or International Maritime Organization requirements, then the rest of this section is not applicable.

(2) When sealed, intact means of packaging complying with Department of Transportation or International Maritime Organization requirements containing substances or cargo break, leak or are damaged so that there is a reasonable possibility of leakage, the exposure limits specified in 29 CFR Part 1917, Subpart Z are applicable. These exposure limits shall be met by any reasonable combination of engineering controls work practices and personal protective equipment.

(e) *Effective and start-up dates.*—(1) *effective date.* The effective date for the permissible exposure limits specified in the Final Rule Limits columns of Table Z, Longshoring and Marine Terminals is [90 days after date of Publication in the **Federal Register**].

(2) Start-up dates. (i) The permissible exposure limits specified in the Final Rule Limits columns of Table Z, Longshoring and Marine Terminals shall be achieved by any reasonable combination of engineering controls, work practices and personal protective equipment from the effective date through [4 years after date of publication in the **Federal Register**].

(ii) The permissible exposure limits specified in the Final Rule Limits columns of Table Z, Longshoring and Marine Terminals shall be achieved by the method of compliance specified in paragraph (c) of this section commencing [4 years after date of publication in the **Federal Register**].

(iii) The skin designation in the Final Rule Limits columns shall be complied with commencing on the effective date.

(3) Transitional provision. The permissible exposure limits specified in the Transitional Limits columns of Table Z, Longshoring and Marine Terminals (which are a Time Weighted Average unless preceded by a "C" in which case they are a Ceiling) shall continue to be achieved by the methods of compliance specified in paragraph (c) of this section from the effective date through [4 years after date of publication in the **Federal Register**].

(4) If any new or amended provisions or new or revised limits for any substance or substances are either administratively stayed or judicially stayed or vacated, then the existing provisions or limits for those substances specified in the Transitional Limits columns of Table Z, Longshoring and Marine Terminals shall remain in effect until such stay is lifted, or indefinitely if the limit is vacated.

TABLE Z.—Longshoring &

Substance	CAS No.[4]	Transitional Limits PEL[5]		Skin Designation
		ppm[6]	mg/m[3][6]	
Acetaldehyde	75-07-0	200	360	—
Acetic acid	64-19-7	10	25	—
Acetic anhydride	108-24-7	5	20	—
Acetone	67-64-1	1000	2400	—
Acetonitrile	75-05-8	40	70	—
2-Acetylaminofluorine; see 1910.1014	53-96-3			
Acetylene dichloride; see 1,2-Dichloroethylene				
Acetylene tetrabromide	79-27-6	1	14	—
Acetylsalicylic acid (Aspirin)	50-78-2	—	—	—
Acrolein	107-02-8	0.1	0.25	—
Acrylamide	79-06-1	—	0.3	X
Acrylic acid	79-10-7	—	—	—
Acrylonitrile[7]; see 1910.1045	107-13-1	(7)	(7)	(7)
Aldrin	309-00-2	—	0.25	X
Allyl alcohol	107-18-6	2	5	X
Allyl chloride	107-05-1	1	3	—
Allyl glycidyl ether (AGE)	106-92-3	(C)10	(C)45	—
Allyl propyl disulfide	2179-59-1	2	12	—
alpha-Alumina	1344-28-1			
Total dust		—	15	—
Respirable fraction		—	5	—
Aluminum (as Al)	7429-90-5			
Metal				
Total dust		—	15	—
Respirable fraction		—	5	—
Pyro powders		—	—	—
Welding fumes**		—	—	—
Soluble salts		—	—	—
Alkyls		—	—	—
4-Aminodiphenyl; see 1910.1011	92-67-1			
2-Aminoethanol; see Ethanolamine				
2-Aminopyridine	504-29-0	0.5	2	—
Amitrole	61-82-5	—	—	—
Ammonia	7664-41-7	50	35	—
Ammonium chloride fume	12125-02-9	—	—	—
Ammonium sulfamate	7773-06-0			
Total dust		—	15	—
Respirable fraction		—	5	—
n-Amyl acetate	628-63-7	100	525	—
sec-Amyl acetate	626-38-0	125	650	—
Aniline and homologs	62-53-3	5	19	X
Anisidine (o-, p-isomers)	29191-52-4	—	0.5	X
Antimony and compounds (as Sb)	7440-36-0	—	0.5	—
ANTU (alpha Naphthylthiourea)	86-88-4	—	0.3	—
Arsenic, inorganic compounds (as As)[7]; see 1910.1018	7440-38-2	—	0.01	—
Arsenic, organic compounds (as As)	7440-38-2	—	0.5	—
Arsine	7784-42-1	0.05	0.2	—
Asbestos[7]; see 1910.1001 and 1910.1101	Varies	(7)	(7)	(7)
Asphalt (Petroleum) Fumes	8052-42-4	—	—	—
Atrazine	1912-24-9	—	—	—
Azinphos-methyl	86-50-0	—	0.2	X
Barium, soluble compounds (as Ba)	7440-39-3	—	0.5	—
Barium sulfate	7727-43-7			
Total dust		—	15	—

ng & Marine Terminals

	Proposed Final Rule Limits						
	TWA		STEL [c]		CEILING		Skin Designation
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
	100	180	150	270			
	10	25					
	—	—			5	20	
	750	1800	1000	2400			
	40	70	60	105			
	1	14					
	—	5					
	0.1	0.25	0.3	0.8			
X	—	0.03					X
	10	30					X
(3)	2	—	10				X
X	—	0.25					X
X	2	5	4	10			X
	1	3	2	6			
	5	22	10	44			
	2	12	3	18			
		10					
		5					
		15					
		5					
		5					
		5					
		2					
		2					
	0.5	2					
	—	0.2					
		—	35	27			
		10		20			
		10					
		5					
	100	525					
	125	650					
X	2	8					X
X	—	0.5					
	—	0.5					
	—	0.3					
	—	0.01					
	—	0.5					
	0.05	0.2					
(7)	—	0.2 f/cc	—	1 f/cc (30 min.)	—	—	
	—	5	—	—	—	—	
X	—	5	—	—	—	—	X
	—	0.2	—	—	—	—	
	—	0.5	—	—	—	—	
	—	10	—	—	—	—	

Respirable fraction		—	5	—
Benomyl	17804-35-2			
Total dust		—	15	—
Respirable fraction		—	5	—
Benzene [f,g]; see 1910.1028	71-43-2	(*)	(*)	(*)
Benzidine; see 1910.1010	92-87-5			
p-Benzoquinone; see Quinone				
Benzo(a)pyrene; see Coal tar pitch volatiles				
Benzoyl peroxide	94-36-0	—	5	—
Benzyl chloride	100-44-7	1	5	—
Beryllium and beryllium compounds (as Be)	7440-41-7	(*)	(*)	(*)
Biphenyl; see Diphenyl				
Bismuth telluride, Undoped	1304-82-1			
Total dust		—	15	—
Respirable fraction		—	5	—
Bismuth telluride, Se-doped	1304-82-1	—	—	—
Borates, tetra, sodium salts				
Anhydrous	1330-43-4	—	—	—
Decahydrate	1303-96-4	—	—	—
Pentahydrate	12179-04-3	—	—	—
Boron oxide	1303-86-2			
Total dust		—	15	—
Boron tribromide	10294-33-4	—	—	—
Boron trifluoride	7637-07-2	(C)1	(C)3	—
Bromacil	314-40-9	—	—	—
Bromine	7726-95-6	0.1	0.7	—
Bromine pentafluoride	7789-30-2	—	—	—
Bromoform	75-25-2	0.5	5	X
Butadiene (1,3-Butadiene) [h]; see 55 FR 32736	106-99-0	1000	2200	—
Butane	106-97-8	—	—	—
Butanethiol; see Butyl mercaptan				
2-Butanone (Methyl ethyl ketone)	78-93-3	200	590	—
2-Butoxyethanol	111-76-2	50	240	X
n-Butyl-acetate	123-86-4	150	710	—
sec-Butyl acetate	105-46-4	200	950	—
tert-Butyl acetate	540-88-5	200	950	—
Butyl acrylate	141-32-2	—	—	—
n-Butyl alcohol	71-36-3	100	300	—
sec-Butyl alcohol	78-92-2	150	450	—
tert-Butyl alcohol	75-65-0	100	300	—
Butylamine	109-73-9	(C)5	(C)15	X
tert-Butyl chromate (as CrO_3)	1189-85-1	—	(C)0.1	X
n-Butyl glycidyl ether (BGE)	2426-08-6	50	270	—
n-Butyl lactate	138-22-7	—	—	—
Butyl mercaptan	109-79-5	10	35	—
o-sec-Butylphenol	89-72-5	—	—	—
p-tert-Butyltoluene	98-51-1	10	60	—
Cadmium fume (as Cd) [i]; see 55 FR 4052	7440-43-9	(*)	(*)	(*)
Cadmium dust (as Cd) [i]; see 55 FR 4052	7440-43-9	(*)	(*)	(*)
Calcium carbonate	1317-65-3			
Total dust		—	15	—
Respirable fraction		—	5	—
Calcium cyanamide	156-62-7	—	—	—
Calcium hydroxide	1305-62-0			
Total dust		—	15	—
Respirable fraction		—	5	—

| | | |

TABLE Z.—Longshoring & Marin

Substance	CAS No.[4]	Transitional Limits PEL[5]		Skin Designation
		ppm [6]	mg/m [3] [6]	
Calcium oxide	1305-78-8	—	5	—
Calcium silicate	1344-95-2			
Total dust		—	15	—
Respirable fraction		—	5	—
Calcium sulfate	7778-18-9			
Total dust		—	15	—
Respirable fraction		—	5	—
Camphor, synthetic	76-22-2	—	2	—
Caprolactam	105-60-2			
Dust		—	—	—
Vapor		—	—	—
Captafol (Difolatan)	2425-06-1	—	—	—
Captan	133-06-2	—	—	—
Carbaryl (Sevin)	63-25-2	—	5	—
Carbofuran (Furadan)	1563-66-2	—	—	—
Carbon black	1333-86-4	—	3.5	—
Carbon dioxide	124-38-9	5000	9000	—
Carbon disulfide	75-15-0	(7)	(7)	(7)
Carbon monoxide	630-08-0	50	55	—
Carbon tetrabromide	558-13-4	—	—	—
Carbon tetrachloride	56-23-5	(7)	(7)	(7)
Carbonyl fluoride	353-50-4	—	—	—
Catechol (Pyrocatechol)	120-80-9	—	—	—
Cellulose	9004-34-6			
Total dust		—	15	—
Respirable fraction		—	5	—
Cesium hydroxide	21351-79-1	—	—	—
Chlordane	57-74-9	—	0.5	X
Chlorinated camphene	8001-35-2	—	0.5	X
Chlorinated diphenyl oxide	55720-99-5	—	0.5	—
Chlorine	7782-50-5	(C)1	(C)3	—
Chlorine dioxide	10049-04-4	0.1	0.3	—
Chlorine trifluoride	7790-91-2	(C)0.1	(C)0.4	—
Chloroacetaldehyde	107-20-0	(C)1	(C)3	—
a-Chloroacetophenone (Phenacyl chloride)	532-27-4	0.05	0.3	—
Chloroacetyl chloride	79-04-9	—	—	—
Chlorobenzene	108-90-7	75	350	—
o-Chlorobenzylidene malononitrile	2698-41-1	0.05	0.4	—
Chlorobromomethane	74-97-5	200	1050	—
2-Chloro-1,3-butadiene; see beta-Chloroprene				
Chlorodifluoromethane	75-45-6	—	—	—
Chlorodiphenyl (42% Chlorine) (PCB)	53469-21-9	—	1	X
Chlorodiphenyl (54% Chlorine) (PCB)	11097-69-1	—	0.5	X
1-Chloro,2,3-epoxypropane; see Epichlorohydrin				
2-Chloroethanol; see Ethylene chlorohydrin				
Chloroethylene; see Vinyl chloride				
Chloroform (Trichloromethane)	67-66-3	(C)50	(C)240	—
bis(Chloromethyl) ether; see 1910.1008	542-88-1			
Chloromethyl methyl ether; see 1910.1006	107-30-2			
1-Chloro-1-nitropropane	600-25-9	20	100	—
Chloropentafluoroethane	76-15-3	—	—	—
Chloropicrin	76-06-2	0.1	0.7	—
beta-Chloroprene	126-99-8	25	90	X
o-Chlorostyrene	2039-87-4	—	—	—
o-Chlorotoluene	95-49-8	—	—	—
2-Chloro-6-(trichloromethyl) pyridine	1929-82-4			
Total dust		—	15	—

Marine Terminals—Continued

n ig- on	Proposed Final Rule Limits						Skin Desig- nation
	TWA		STEL [c]		CEILING		
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
	—	5	—	—	—	—	—
	—	15	—	—	—	—	—
	—	5	—	—	—	—	—
	—	15	—	—	—	—	—
	—	5	—	—	—	—	—
	—	2	—	—	—	—	—
	—	1	—	3	—	—	—
	5	20	10	40	—	—	—
	—	0.1	—	—	—	—	—
	—	5	—	—	—	—	—
	—	5	—	—	—	—	—
	—	0.1	—	—	—	—	—
	—	3.5	—	—	—	—	—
	10,000	18,000	30,000	54,000	—	—	—
[3]	4	12	12	36	—	—	X
	35	40	—	—	200	229	—
	0.1	1.4	0.3	4	—	—	—
[3]	2	12.6	—	—	—	—	—
	2	5	5	15	—	—	—
	5	20	—	—	—	—	X
	—	15	—	—	—	—	—
	—	5	—	—	—	—	—
	—	2	—	—	—	—	—
X	—	0.5	—	—	—	—	X
X	—	0.5	—	1	—	—	X
	—	0.5	—	—	—	—	—
	0.5	1.5	1	3	—	—	—
	0.1	0.3	0.3	0.9	—	—	—
	—	—	—	—	0.1	0.4	—
	—	—	—	—	1	3	—
	0.05	0.3	—	—	—	—	—
	0.05	0.2	—	—	—	—	—
	75	350	—	—	—	—	—
	—	—	—	—	0.05	0.4	X
	200	1050	—	—	—	—	—
	1000	3500	—	—	—	—	—
X	—	1	—	—	—	—	X
X	—	0.5	—	—	—	—	X
	2	9.78	—	—	—	—	—
	2	10	—	—	—	—	—
	1000	5320	—	—	—	—	—
	0.1	0.7	—	—	—	—	—
X	10	35	—	—	—	—	X
	50	265	75	428	—	—	—
	50	250	—	—	—	—	—
	—	15	—	—	—	—	—

Respirable fraction		—	5	—
Chlorpyrifos	2921-88-2	—	—	—
Chromic acid and chromates (as CrO_3)	Varies with compound	—	(C)0.1	—
Chromium (II) compounds (as Cr)	7440-47-3	—	0.5	—
Chromium (III) compounds (as Cr)	7440-47-3	—	0.5	—
Chromium metal (as Cr)	7440-47-3	—	1	—
Chrysene; see Coal tar pitch volatiles				
Clopidol	2971-90-6			
Total dust		—	15	—
Respirable fraction		—	5	—
Coal dust (less than 5% SiO_2), Respirable fraction		(1)	(1)	(1)
Coal dust (greater than or equal to 5% SiO_2), Respirable quartz fraction		(1)	(1)	(1)
Coal tar pitch volatiles (benzene-soluble fraction), anthracene BaP, phenanthrene, acridine, chrysene, pyrene	[illegible]	—	0.2	—
Cobalt metal, dust, and fume (as Co)	7440-48-4	—	0.1	—
Cobalt carbonyl (as Co)	10210-68-1	—	—	—
Cobalt hydrocarbonyl (as Co)	16842-03-8	—	—	—
Coke oven emissions [2]; see 1910.1029				
Copper	7440-50-8			
Fume (as Cu)		—	0.1	—
Dusts and mists (as Cu)		—	1	—
Cotton dust [3]; see 1910.1043		—	1	—
This 8-hour TWA applies to respirable dust as measured by a vertical elutriator cotton dust sampler or equiv respirable dust personal sampler may be used. In these circumstances the exposure limit is 1mg/m³ respirable				
Crag herbicide (Sesone)	136-78-7			
Total dust		—	15	—
Respirable fraction		—	5	—
Cresol, all isomers	1319-77-3	5	22	X
Crotonaldehyde	123-73-9; 4170-30-3	2	6	—
Crufomate	299-86-5	—	—	—
Cumene	98-82-8	50	245	X
Cyanamide	420-04-2	—	—	—
Cyanides (as CN)	Varies with Compound	—	5	—
Cyanogen	460-19-5	—	—	—
Cyanogen chloride	506-77-4	—	—	—
Cyclohexane	110-82-7	300	1050	—
Cyclohexanol	108-93-0	50	200	—
Cyclohexanone	108-94-1	50	200	—
Cyclohexene	110-83-8	300	1015	—
Cyclohexylamine	108-91-8	—	—	—
Cyclonite	121-82-4	—	—	—
Cyclopentadiene	542-92-7	75	200	—
Cyclopentane	287-92-3	—	—	—
Cyhexatin	13121-70-5	—	—	—
2,4-D (Dichlorophenoxyacetic acid)	94-75-7	—	10	—
Decaborane	17702-41-9	0.05	0.2	X
Demeton (Systox)	8065-48-3	—	0.1	X
Diacetone alcohol (4-Hydroxy-4-methyl-2-pentanone)	123-42-2	50	240	—
1,2-Diaminoethane; see Ethylenediamine				
Diazinon	333-41-5	—	—	—
Diazomethane	334-88-3	0.2	0.4	—
Diborane	19287-45-7	0.1	0.1	—
1,2-Dibromo-3-chloropropane (DBCP) [4]; see 1910.1044	96-12-8	0.001	—	X
1,2-Dibromoethane; see Ethylene dibromide				
2-N-Dibutylaminoethanol	102-81-8	—	—	—
Dibutyl phosphate	107-66-4	1	5	—
Dibutyl phthalate	84-74-2	—	5	—
Dichloroacetylene	7572-29-4	—	—	—
o-Dichlorobenzene	95-50-1	(C)50	(C)300	—

—	—	5	—	—	—	—	—
—	—	0.2	—	—	—	—	—
—	—	—	—	—	—	0.1	—
—	—	0.5	—	—	—	—	—
—	—	0.5	—	—	—	—	—
—	—	1	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
(1)	—	2	—	—	—	—	—
(2)	—	0.1	—	—	—	—	—
—	—	0.2	—	—	—	—	—
—	—	0.05	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	1	—	—	—	—	—
—	—	0.5	—	—	—	—	—

equivalent instrument. For the Transitional Limit and where it is not feasible to use a vertical elutriator, a irable dust, personal sampler.

—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
X	5	22	—	—	—	X	—
—	2	6	—	—	—	—	—
—	—	5	—	—	—	—	—
X	50	245	—	—	—	X	—
—	—	2	—	—	—	—	—
—	—	5	—	—	—	—	—
—	10	20	—	—	—	—	—
—	—	—	—	—	0.3	0.6	—
—	300	1050	—	—	—	—	—
—	50	200	—	—	—	X	—
—	25	100	—	—	—	X	—
—	300	1015	—	—	—	—	—
—	10	40	—	—	—	—	—
—	—	1.5	—	—	—	X	—
—	75	200	—	—	—	—	—
—	600	1720	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	10	—	—	—	—	—
X	0.05	0.3	0.15	0.9	—	X	—
X	—	0.1	—	—	—	X	—
—	50	240	—	—	—	—	—
—	—	0.1	—	—	—	X	—
—	0.2	0.4	—	—	—	—	—
—	0.1	0.1	—	—	—	—	—
X	0.001	—	—	—	—	—	X
—	2	14	—	10	—	—	—
—	1	5	2	—	—	—	—
—	—	5	—	—	0.1	0.4	—
—	—	—	—	—	50	300	—

Substance	CAS No.[d]	Transitional Limits PEL[c]		Skin Designation
		ppm [a]	mg/m [a b]	
p-Dichlorobenzene	106-46-7	75	450	—
3,3'-Dichlorobenzidine; see 1910.1007	91-94-1			
Dichlorodifluoromethane	75-71-8	1000	4950	—
1,3-Dichloro-5,5-dimethyl hydantoin	118-52-5	—	0.2	—
Dichlorodiphenyltrichloroethane (DDT)	50-29-3	—	1	X
1,1-Dichloroethane	75-34-3	100	400	—
1,2-Dichloroethylene	540-59-0	200	790	—
Dichloroethyl ether	111-44-4	(C)15	(C)90	X
Dichloromethane; see Methylene chloride				
Dichloromonofluoromethane	75-43-4	1000	4200	—
1,1-Dichloro-1-nitroethane	594-72-9	(C)10	(C)60	—
1,2-Dichloropropane; see Propylene dichloride				
1,3-Dichloropropene	542-75-6	—	—	—
2,2-Dichloropropionic acid	75-99-0	—	—	—
Dichlorotetrafluoroethane	76-14-2	1000	7000	—
Dichlorvos (DDVP)	62-73-7	—	1	X
Dicrotophos	141-66-2	—	—	—
Dicyclopentadiene	77-73-6	—	—	—
Dicyclopentadienyl iron	102-54-5			
Total dust		—	15	—
Respirable fraction		—	5	—
Dieldrin	60-57-1	—	0.25	X
Diethanolamine	111-42-2	—	—	—
Diethylamine	109-89-7	25	75	—
2-Diethylaminoethanol	100-37-8	10	50	X
Diethylene triamine	111-40-0	—	—	—
Diethyl ether; see Ethyl ether				
Diethyl ketone	96-22-0	—	—	—
Diethyl phthalate	84-66-2	—	—	—
Difluorodibromomethane	75-61-6	100	860	—
Diglycidyl ether (DGE)	2238-07-5	(C)0.5	(C)2.8	—
Dihydroxybenzene; see Hydroquinone				
Diisobutyl ketone	108-83-8	50	290	—
Diisopropylamine	108-18-9	5	20	X
4-Dimethylaminoazobenzene; see 1910.1015	60-11-7			
Dimethoxymethane; see Methylal				
Dimethyl acetamide	127-19-5	10	35	X
Dimethylamine	124-40-3	10	18	—
Dimethylaminobenzene; see Xylidine				
Dimethylaniline (N-Dimethylaniline)	121-69-7	5	25	X
Dimethylbenzene; see Xylene				
Dimethyl-1,2-dibromo-2,2-dichloroethyl phosphate	300-76-5	—	5	—
Dimethylformamide	68-12-2	10	30	X
2,6-Dimethyl-4-heptanone; see Diisobutyl ketone				
1,1-Dimethylhydrazine	57-14-7	0.5	1	X
Dimethylphthalate	131-11-3	—	5	—
Dimethyl sulfate	77-78-1	1	5	X
Dinitolmide (3,5-Dinitro-o-toluamide)	148-01-6	—	—	—
Dinitrobenzene (all isomers)		—	1	X
(ortho)	528-29-0			
(meta)	99-65-0			
(para)	100-25-4			
Dinitro-o-cresol	534-52-1	—	0.2	X
Dinitrotoluene	25321-14-6	—	1.5	X
Dioxane (Diethylene dioxide)	123-91-1	100	360	X
Dioxathion (Delnav)	78-34-2	—	—	—
Diphenyl (Biphenyl)	92-52-4	0.2	1	—

Marine Terminals—Continued

Skin Designation	Proposed Final Rule Limits						Skin Designation
	TWA		STEL [c]		CEILING		
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
—	75	450	110	675	—	—	—
—	1000	4950	—	—	—	—	—
—	—	0.2	—	0.4	—	—	—
X	—	1	—	—	—	—	X
—	100	400	—	—	—	—	—
—	200	790	—	—	—	—	—
X	5	30	10	60	—	—	X
—	10	40	—	—	—	—	—
—	2	10	—	—	—	—	—
—	1	5	—	—	—	—	X
—	1	6	—	—	—	—	—
—	1000	7000	—	—	—	—	—
X	—	1	—	—	—	—	X
—	—	0.25	—	—	—	—	X
—	5	30	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
X	—	0.25	—	—	—	—	X
—	3	15	—	—	—	—	—
—	10	30	25	75	—	—	—
X	10	50	—	—	—	—	X
—	1	4	—	—	—	—	—
—	200	705	—	—	—	—	—
—	—	5	—	—	—	—	—
—	100	860	—	—	—	—	—
—	0.1	0.5	—	—	—	—	—
—	25	150	—	—	—	—	—
X	5	20	—	—	—	—	X
X	10	35	—	—	—	—	X
—	10	18	—	—	—	—	—
X	5	25	10	50	—	—	X
—	—	3	—	—	—	—	X
X	10	30	—	—	—	—	X
X	0.5	1	—	—	—	—	X
—	—	5	—	—	—	—	—
X	0.1	0.5	—	—	—	—	X
—	—	5	—	—	—	—	—
X	—	1	—	—	—	—	X
X	—	0.2	—	—	—	—	X
X	—	1.5	—	—	—	—	X
X	25	90	—	—	—	—	X
—	—	0.2	—	—	—	—	—
—	0.2	1	—	—	—	—	—

Diphenylamine	122-39-4	—	—	—
Diphenylmethane diisocyanate; see Methylene bisphenyl isocyanate				
Dipropylene glycol methyl ether	34590-94-8	100	600	X
Dipropyl ketone	123-19-3	—	—	—
Diquat	85-00-7	—	—	—
Di-sec octyl phthalate (Di-(2-ethylhexyl) phthalate	117-81-7	—	5	—
Disulfiram	97-77-8	—	—	—
Disulfoton	298-04-4	—	—	X
2,6-Di-tert-butyl-p-cresol	128-37-0	—	—	—
Diuron	330-54-1	—	—	—
Divinyl benzene	1321-74-0	—	—	—
Emery	12415-34-8			
Total dust		—	15	—
Respirable fraction		—	5	—
Endosulfan	115-29-7	—	—	—
Endrin	72-20-8	—	0.1	X
Epichlorohydrin	106-89-8	5	19	X
EPN	2104-64-5	—	0.5	X
1,2-Epoxypropane; see Propylene oxide				
2,3-Epoxy-1-propanol; see Glycidol				
Ethanethiol; see Ethyl mercaptan				
Ethanolamine	141-43-5	3	6	—
Ethion	563-12-2	—	—	—
2-Ethoxyethanol (Cellosolve)	110-80-5	200	740	X
2-Ethoxyethyl acetate (Cellosolve acetate)	111-15-9	100	540	X
Ethyl acetate	141-78-6	400	1400	—
Ethyl acrylate	140-88-5	25	100	X
Ethyl alcohol (Ethanol)	64-17-5	1000	1900	—
Ethylamine	75-04-7	10	18	—
Ethyl amyl ketone (5-Methyl-3-heptanone)	541-85-5	25	130	—
Ethyl benzene	100-41-4	100	435	—
Ethyl bromide	74-96-4	200	890	—
Ethyl butyl ketone (3-Heptanone)	106-35-4	50	230	—
Ethyl chloride	75-00-3	1000	2600	—
Ethyl ether	60-29-7	400	1200	—
Ethyl formate	109-94-4	100	300	—
Ethyl mercaptan	75-08-1	(C)10	(C)25	—
Ethyl silicate	78-10-4	100	850	—
Ethylene chlorohydrin	107-07-3	5	16	X
Ethylenediamine	107-15-3	10	25	—
Ethylene dibromide	106-93-4	(1)	(1)	(1)
Ethylene dichloride	107-06-2	(2)	(2)	(2)
Ethylene glycol	107-21-1	—	—	—
Ethylene glycol dinitrate	628-96-6	(C)0.2	(C)1	X
Ethylene glycol methyl acetate; see Methyl cellosolve acetate				
Ethylenelmine; see 1910.1012	151-56-4			
Ethylene oxide [4]; see 1910.1047	75-21-8	(1)	(1)	(1)
Ethylidene chloride; see 1,1-Dichloroethane				
Ethylidene norbornene	16219-75-3	—	—	—
N-Ethylmorpholine	100-74-3	20	94	X
Fenamiphos	22224-92-6	—	—	—
Fensulfothion (Dasanit)	115-90-2	—	—	—
Fenthion	55-38-9	—	—	—
Ferbam	14484-64-1			
Total dust		—	15	—
Ferrovanadium dust	12604-58-9	—	1	—
Fibrous glass				
Total dust		—	15	—
Respirable fraction		—	5	—
Fluorides (as F)	Varies with compound	—	2.5	—
Fluorine	7782-41-4	0.1	0.2	—
Fluorotrichloromethane (Trichlorofluoromethane)	75-69-4	1000	5600	—

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TABLE Z.—Longshoring & Marin

Substance	CAS No.[4]	Transitional Limits PEL[a]		Skin Designation
		ppm[a]	mg/m[3][b]	
Fonofos	944-22-9	—	—	—
Formaldehyde[c,b]; see 1910.1048; 56 FR 3202	50-00-0	1	(2 ppm STEL)	
Formamide	75-12-7	—	—	—
Formic acid	64-18-6	5	9	—
Furfural	98-01-1	5	20	X
Furfuryl alcohol	98-00-0	50	200	—
Gasoline	8006-61-9	—	—	—
Germanium tetrahydride	7782-65-2	—	—	—
Glutaraldehyde	111-30-8	—	—	—
Glycerin (mist)	56-81-5			
Total dust		—	15	—
Respirable fraction		—	5	—
Glycidol	556-52-5	50	150	—
Glycol monoethyl ether; see 2-Ethoxyethanol				
Grain dust (oat, wheat, barley)		—	—	—
Graphite, natural, respirable dust	7782-42-5	([3])	([3])	([3])
Graphite, synthetic				
Total dust		—	15	—
Respirable fraction		—	5	—
Guthion; see Azinphos methyl				
Gypsum	13397-24-5			
Total dust		—	15	—
Respirable fraction		—	5	—
Hafnium	7440-58-6	—	0.5	—
Heptachlor	76-44-8	—	0.5	X
Heptane (n-Heptane)	142-82-5	500	2000	—
Hexachlorobutadiene	87-68-3	—	—	—
Hexachlorocyclopentadiene	77-47-4	—	—	—
Hexachloroethane	67-72-1	1	10	X
Hexachloronaphthalene	1335-87-1	—	0.2	X
Hexafluoroacetone	684-16-2	—	—	—
n-Hexane	110-54-3	500	1800	—
Hexane isomers	Varies with compound	—	—	—
2-Hexanone (Methyl n-butyl ketone)	591-78-6	100	410	—
Hexone (Methyl isobutyl ketone)	108-10-1	100	410	—
sec-Hexyl acetate	108-84-9	50	300	—
Hexylene glycol	107-41-5	—	—	—
Hydrazine	302-01-2	1	1.3	X
Hydrogenated terphenyls	61788-32-7	—	—	—
Hydrogen bromide	10035-10-6	3	10	—
Hydrogen chloride	7647-01-0	(C)5	(C)7	—
Hydrogen cyanide	74-90-8	10	11	—
Hydrogen fluoride (as F)	7664-39-3	([3])	([3])	([3])
Hydrogen peroxide	7722-84-1	1	1.4	—
Hydrogen selenide (as Se)	7783-07-5	0.05	0.2	—
Hydrogen sulfide	7783-06-4	([3])	([3])	([3])
Hydroquinone	123-31-9	—	2	—
2-Hydroxypropyl acrylate	999-61-1	—	—	—
Indene	95-13-6	—	—	—
Indium and compounds (as In)	7440-74-6	—	—	—
Iodine	7553-56-2	(C)0.1	(C)1	—
Iodoform	75-47-8	—	—	—
Iron oxide fume	1309-37-1	—	10	—
Iron pentacarbonyl (as Fe)	13463-40-6	—	—	—

g & Marine Terminals—Continued

Skin Designation	Proposed Final Rule Limits						Skin Designation
	TWA		STEL [c]		CEILING		
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
—	—	0.1	—	—	—	—	
	0.75	—	2	—	—	—	X
—	20	30	30	45	—	—	—
—	5	9	—	—	—	—	—
X	2	8	—	—	—	—	—
—	10	40	15	60	—	—	X
—	300	900	500	1500	—	—	X
—	0.2	0.6	—	—	—	—	—
—	—	—	—	—	0.2	0.8	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	25	75	—	—	—	—	—
—	—	10	—	—	—	—	—
(3)	—	2.5	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
X	—	0.5	—	—	—	—	—
—	—	0.5	—	—	—	—	—
—	400	1600	500	2000	—	—	X
—	0.02	0.24	—	—	—	—	—
X	0.01	0.1	—	—	—	—	—
X	1	10	—	—	—	—	—
—	—	0.2	—	—	—	—	X
—	0.1	0.7	—	—	—	—	X
—	50	180	—	—	—	—	X
—	500	1800	1000	3600	—	—	—
—	5	20	—	—	—	—	—
—	50	205	75	300	—	—	—
—	50	300	—	—	—	—	—
X	—	—	—	—	—	—	—
—	0.1	0.1	—	—	25	125	—
—	0.5	5	—	—	—	—	X
—	—	—	—	—	3	10	—
X	—	—	—	—	5	7	—
(3)	3	—	4.7	5	—	—	X
—	1	1.4	6	—	—	—	—
—	0.05	0.2	—	—	—	—	—
(3)	10	14	15	21	—	—	—
—	—	2	—	—	—	—	—
—	0.5	3	—	—	—	—	—
—	10	45	—	—	—	—	X
—	—	0.1	—	—	—	—	—
—	—	—	—	—	—	—	—
—	0.6	10	—	—	0.1	1	—
—	—	10	—	—	—	—	—
—	0.1	0.8	0.2	1.6	—	—	—

Iron salts (soluble)(as Fe)	Varies with compound	—	—	—
Isoamyl acetate	123-92-2	100	525	—
Isoamyl alcohol (primary and secondary)	123-51-3	100	360	—
Isobutyl acetate	110-19-0	150	700	—
Isobutyl alcohol	78-83-1	100	300	—
Isooctyl alcohol	26952-21-6	—	—	—
Isophorone	78-59-1	25	140	—
Isophorone diisocyanate	4098-71-9	—	—	—
2-Isopropoxyethanol	109-59-1	—	—	—
Isopropyl acetate	108-21-4	250	950	—
Isopropyl alcohol	67-63-0	400	980	—
Isopropylamine	75-31-0	5	12	—
N-Isopropylaniline	768-52-5	—	—	—
Isopropyl ether	108-20-3	500	2100	—
Isopropyl glycidyl ether (IGE)	4016-14-2	50	240	—
Kaolin	1332-58-7			
Total dust		—	15	—
Respirable fraction		—	5	—
Ketene	463-51-4	0.5	0.9	—
Lead inorganic (as Pb)[7]; see 1910.1025	7439-92-1	—	0.05	—
Limestone	1317-65-3			
Total dust		—	15	—
Respirable fraction		—	5	—
Lindane	58-89-9	—	0.5	X
Lithium hydride	7580-67-8	—	0.025	—
L.P.G. (Liquefied petroleum gas)	68476-85-7	1000	1800	—
Magnesite	546-93-0			
Total dust		—	15	—
Respirable fraction		—	5	—
Magnesium oxide fume	1309-48-4			
Total particulate		—	15	—
Malathion	121-75-5			
Total dust		—	15	X
Maleic anhydride	108-31-6	0.25	1	—
Manganese compounds (as Mn)	7439-96-5	—	(C)5	—
Manganese fume (as Mn)	7439-96-5	—	(C)5	—
Manganese cyclopentadienyl tricarbonyl (as Mn)	12079-65-1	—	—	—
Manganese tetroxide (as Mn)	1317-35-7	—	—	—
Marble	1317-65-3			
Total dust		—	15	—
Respirable fraction		—	5	—
Mercury (aryl and inorganic)(as Hg)	7439-97-6	([2])	([2])	([2])
Mercury (organo) alkyl compounds (as Hg)	7439-97-6	([2])	([2])	([2])
Mercury (vapor) (as Hg)	7439-97-6	([2])	([2])	([2])
Mesityl oxide	141-79-7	25	100	—
Methacrylic acid	79-41-4	—	—	—
Methanethiol; see Methyl mercaptan				
Methomyl (Lannate)	16752-77-5	—	—	—
Methoxychlor	72-43-5			
Total dust		—	15	—
2-Methoxyethanol (Methyl cellosolve)	109-86-4	25	80	X
2-Methoxyethyl acetate (Methyl cellosolve acetate)	110-49-6	25	120	X
4-Methoxyphenol	150-76-5	—	—	—
Methyl acetate	79-20-9	200	610	—
Methyl acetylene (Propyne)	74-99-7	1000	1650	—
Methyl acetylenepropadiene mixture (MAPP)	—	1000	1800	—
Methyl acrylate	96-33-3	10	35	X
Methylacrylonitrile	126-98-7	—	—	—
Methylal (Dimethoxymethane)	109-87-5	1000	3100	—
Methyl alcohol	67-56-1	200	260	—
Methylamine	74-89-5	10	12	—
Methyl amyl alcohol; see Methyl isobutyl carbinol				
Methyl n-amyl ketone	110-43-0	100	465	—
Methyl bromide	74-83-9	(C)20	(C)80	X
Methyl butyl ketone; see 2-Hexanone				

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TABLE Z.—Longshoring & Marine

Substance	CAS No.[c]	Transitional Limits PEL[a] ppm[a]	Transitional Limits PEL[a] mg/m³[b]	Skin Designation
Methyl cellosolve; see 2-Methoxyethanol				
Methyl cellosolve acetate; see 2-Methoxyethyl acetate				
Methyl chloride	74-87-3	(*)	(*)	(*)
Methyl chloroform (1,1,1-Trichloroethane)	71-55-6	350	1900	—
Methyl 2-cyanoacrylate	137-05-3	—	—	—
Methylcyclohexane	108-87-2	500	2000	—
Methylcyclohexanol	25639-42-3	100	470	—
o-Methylcyclohexanone	583-60-8	100	460	X
Methylcyclopentadienyl manganese tricarbonyl (as Mn)	12108-13-3	—	—	—
Methyl demeton	8022-00-2	—	—	—
4,4'-Methylene bis (2-chloroaniline) (MBOCA)	101-14-4	—	—	—
Methylene bis(4-cyclohexylisocyanate)	5124-30-1	—	—	—
Methylene chloride[h]; see 56 FR 57036	75-09-2	(*)	(*)	(*)
Methyl ethyl ketone (MEK); see 2-Butanone				
Methyl ethyl ketone peroxide (MEKP)	1338-23-4	—	—	—
Methyl formate	107-31-3	100	250	—
Methyl hydrazine (Mono- methyl hydrazine)	60-34-4	(C)0.2	(C)0.35	X
Methyl iodide	74-88-4	5	28	X
Methyl isoamyl ketone	110-12-3	—	—	—
Methyl isobutyl carbinol	108-11-2	25	100	X
Methyl isobutyl ketone; see Hexone				
Methyl isocyanate	624-83-9	0.02	0.05	X
Methyl isopropyl ketone	563-80-4	—	—	—
Methyl mercaptan	74-93-1	(C)10	(C)20	—
Methyl methacrylate	80-62-6	100	410	—
Methyl parathion	298-00-0	—	—	—
Methyl propyl ketone; see 2-Pentanone				
Methyl silicate	681-84-5	—	—	—
alpha-Methyl styrene	98-83-9	(C)100	(C)480	—
Methylene bisphenyl isocyanate (MDI)	101-68-8	(C)0.02	(C)0.2	—
Metribuzin	21087-64-9	—	—	—
Mica; see Silicates				
Molybdenum (as Mo)	7439-98-7			
Soluble compounds		—	5	—
Insoluble compounds				
Total dust		—	15	—
Monocrotophos (Azodrin)	6923-22-4	—	—	—
Monomethyl aniline	100-61-8	2	9	X
Morpholine	110-91-8	20	70	X
Naphtha (Coal tar)	8030-30-6	100	400	—
Naphthalene	91-20-3	10	50	—
alpha-Naphthylamine; see 1910.1004	134-32-7			
beta-Naphthylamine; see 1910.1009	91-59-8			
Nickel carbonyl (as Ni)	13463-39-3	0.001	0.007	—
Nickel, metal and insoluble compounds (as Ni)	7440-02-0	—	1	—
Nickel, soluble compounds (as Ni)	7440-02-0	—	1	—
Nicotine	54-11-5	—	0.5	X
Nitric acid	7697-37-2	2	5	—
Nitric oxide	10102-43-9	25	30	—
p-Nitroaniline	100-01-6	1	6	X
Nitrobenzene	98-95-3	1	5	X
p-Nitrochlorobenzene	100-00-5	—	1	X
4-Nitrodiphenyl; see 1910.1003	92-93-3			
Nitroethane	79-24-3	100	310	—
Nitrogen dioxide	10102-44-0	(C)5	(C)9	—

& Marine Terminals—Continued

Skin Designation	Proposed Final Rule Limits						
	TWA		STEL [c]		CEILING		Skin Designation
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
(*)	50	105	100	210	—	—	—
—	350	1900	450	2450	—	—	—
—	2	8	4	16	—	—	—
—	400	1600	—	—	—	—	—
—	50	235	—	—	—	—	—
X	50	230	75	345	—	—	—
—	—	0.2	—	—	—	—	X
—	—	0.5	—	—	—	—	—
—	0.02	0.22	—	—	—	—	X
—	—	—	—	—	—	—	X
(*)	25	—	125	—	0.01	0.11	X
—	—	—	—	—	—	—	—
—	100	250	150	375	0.7	5	—
X	—	—	—	—	—	—	—
X	2	10	—	—	0.2	0.35	—
—	50	240	—	—	—	—	X
X	25	100	40	165	—	—	X
—	0.02	0.05	—	—	—	—	—
X	200	705	—	—	—	—	X
—	0.5	1	—	—	—	—	—
—	100	410	—	—	—	—	—
—	—	0.2	—	—	—	—	—
—	1	6	—	—	—	—	—
—	50	240	100	485	—	—	—
—	—	—	—	—	—	—	—
—	—	5	—	—	0.02	0.2	—
—	—	5	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	0.25	—	—	—	—	—
—	0.5	2	—	—	—	—	—
X	20	70	30	105	—	—	X
—	100	400	—	—	—	—	X
—	10	50	15	75	—	—	—
—	0.001	0.007	—	—	—	—	—
—	—	1	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	0.5	—	—	—	—	—
—	2	5	4	10	—	—	X
—	25	30	—	—	—	—	—
—	—	3	—	—	—	—	—
—	1	5	—	—	—	—	X
—	—	1	—	—	—	—	X
—	—	—	—	—	—	—	X
—	100	310	—	—	—	—	—
—	—	—	1	1.8	—	—	—

Nitrogen trifluoride	7783-54-2	10	29	—
Nitroglycerin	55-63-0	(C)0.2	(C)2	X
Nitromethane	75-52-5	100	250	—
1-Nitropropane	108-03-2	25	90	—
2-Nitropropane	79-46-9	25	90	—
N-Nitrosodimethylamine; see 1910.1016	62-79-0			
Nitrotoluene (all isomers)		5	30	X
o-isomer	88-72-2;			
m-isomer	99-08-1;			
p-isomer	99-99-0			
Nitrotrichloromethane; see Chloropicrin				
Nonane	111-84-2	—	—	—
Octachloronaphthalene	2234-13-1	—	0.1	X
Octane	111-65-9	500	2350	—
Oil mist, mineral	8012-95-1	—	5	—
Osmium tetroxide (as Os)	20816-12-0	—	0.002	—
Oxalic acid	144-62-7	—	1	—
Oxygen difluoride	7783-41-7	0.05	0.1	—
Ozone	10028-15-6	0.1	0.2	—
Paraffin wax fume	8002-74-2	—	—	—
Paraquat, respirable dust	4685-14-7; 1910-42-5; 2074-50-2	—	0.5	X
Parathion	56-38-2	—	0.1	X
Particulates not otherwise regulated				
Total dust		—	15	—
Respirable fraction		—	5	—
PCB; see Chlorodiphenyl (42% and 54% chlorine)				
Pentaborane	19624-22-7	0.005	0.01	—
Pentachloronaphthalene	1321-64-8	—	0.5	X
Pentachlorophenol	87-86-5	—	0.5	X
Pentaerythritol	115-77-5			
Total dust		—	15	—
Respirable fraction		—	5	—
Pentane	109-66-0	1000	2950	—
2-Pentanone (Methyl propyl ketone)	107-87-9	200	700	—
Perchloroethylene (Tetrachloroethylene)	127-18-4	(*)	(*)	(*)
Perchloromethyl mercaptan	594-42-3	0.1	0.8	—
Perchloryl fluoride	7616-94-6	3	13.5	—
Perlite	93763-70-3			
Total dust		—	15	—
Respirable fraction		—	5	—
Petroleum distillates (Naphtha)		500	2000	—
Phenol	108-95-2	5	19	X
Phenothiazine	92-84-2	—	—	—
p-Phenylene diamine	106-50-3	—	0.1	X
Phenyl ether, vapor	101-84-8	1	7	—
Phenyl ether-biphenyl mixture, vapor		1	7	—
Phenylethylene; see Styrene				
Phenyl glycidyl ether (PGE)	122-60-1	10	60	—
Phenylhydrazine	100-63-0	5	22	X
Phenyl mercaptan	108-98-5	—	—	—
Phenylphosphine	638-21-1	—	—	—
Phorate	298-02-2	—	—	—
Phosdrin (Mevinphos)	7786-34-7	—	0.1	X
Phosgene (Carbonyl chloride)	75-44-5	0.1	0.4	—
Phosphine	7803-51-2	0.3	0.4	—
Phosphoric acid	7664-38-2	—	1	—
Phosphorus (yellow)	7723-14-0	—	0.1	—
Phosphorus oxychloride	10025-87-3	—	—	—
Phosphorus pentachloride	10026-13-8	—	1	—
Phosphorus pentasulfide	1314-80-3	—	1	—
Phosphorus trichloride	7719-12-2	0.5	3	—
Phthalic anhydride	85-44-9	2	12	—
m-Phthalodinitrile	626-17-5	—	—	—
Picloram	1918-02-1			

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TABLE Z.—Longshoring & Marine

Substance	CAS No.[4]	Transitional Limits PEL[5] ppm[6]	Transitional Limits PEL[5] mg/m³[6b]	Skin Designation[7]
Total dust		—	15	—
Respirable fraction		—	5	—
Picric acid	88-89-1	—	0.1	X
Piperazine dihydrochloride	142-64-3	—	—	—
Pindone (2-Pivalyl-1,3-indandione)	83-26-1	—	0.1	—
Plaster of Paris	26499-65-0			
Total dust		—	15	—
Respirable fraction		—	5	—
Platinum (as Pt)	7440-06-4			
Metal		—	—	—
Soluble salts		—	0.002	—
Portland cement	65997-15-1			
Total dust		(8)	(8)	(8)
Respirable fraction		(8)	(8)	(8)
Potassium hydroxide	1310-58-3	—	—	—
Propane	74-98-6	1000	1800	—
Propargyl alcohol	107-19-7	—	—	—
beta-Propriolactone; see 1910.1013	57-57-8			
Propionic acid	79-09-4	—	—	—
Propoxur (Baygon)	114-26-1	—	—	—
n-Propyl acetate	109-60-4	200	840	—
n-Propyl alcohol	71-23-8	200	500	—
n-Propyl nitrate	627-13-4	25	110	—
Propylene dichloride	78-87-5	75	350	—
Propylene glycol dinitrate	6423-43-4	—	—	—
Propylene glycol monomethyl ether	107-98-2	—	—	—
Propylene imine	75-55-8	2	5	X
Propylene oxide	75-56-9	100	240	—
Propyne; see Methyl acetylene				
Pyrethrum	8003-34-7	—	5	—
Pyridine	110-86-1	5	15	—
Quinone	106-51-4	0.1	0.4	—
Resorcinol	108-46-3	—	—	—
Rhodium (as Rh), metal fume and insoluble compounds	7440-16-6	—	0.1	—
Rhodium (as Rh), soluble compounds	7440-16-6	—	0.001	—
Ronnel	299-84-3	—	15	—
Rosin core solder pyrolysis products, as formaldehyde		—	—	—
Rotenone	83-79-4	—	5	—
Rouge				
Total dust		—	15	—
Respirable fraction		—	5	—
Selenium compounds (as Se)	7782-49-2	—	0.2	—
Selenium hexafluoride (as Se)	7783-79-1	0.05	0.4	—
Silica, amorphous, precipitated and gel	112926-00-8	(8)	(8)	(8)
Silica, amorphous, diatomaceous earth, containing less than 1% crystalline silica	61790-53-2	(8)	(8)	(8)
Silica, crystalline cristobalite, respirable dust	14464-46-1	(8)	(8)	(8)
Silica, crystalline quartz, respirable dust	14808-60-7	(8)	(8)	(8)
Silica, crystalline tripoli (as quartz), respirable dust	1317-95-9	(8)	(8)	(8)
Silica, crystalline tridymite, respirable dust	15468-32-3	(8)	(8)	(8)
Silica, fused, respirable dust	60676-86-0	(8)	(8)	(8)
Silicates (less than 1% crystalline silica)		(8)	(8)	(8)
Mica (respirable dust)	12001-26-2	(8)	(8)	(8)
Soapstone, total dust		(8)	(8)	(8)
Soapstone, respirable dust		(8)	(8)	(8)

Marine Terminals—Continued

	Proposed Final Rule Limits						
	TWA		STEL [c]		CEILING		Skin Designation
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
X	—	0.1	—	—	—	—	X
—	—	5	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	1	—	—	—	—	—
—	—	0.002	—	—	—	—	—
(d)	—	10	—	—	—	—	—
(d)	—	5	—	—	—	—	—
—	—	—	—	—	—	2	—
—	(f)	(f)	(f)	(f)	(f)	(f)	—
—	1	2	—	—	—	—	X
—	10	30	—	—	—	—	—
—	—	0.5	—	—	—	—	—
—	200	840	250	1050	—	—	—
—	200	500	250	625	—	—	—
—	25	105	40	170	—	—	—
—	75	350	110	510	—	—	—
—	0.05	0.3	—	—	—	—	—
—	100	380	150	540	—	—	—
X	2	5	—	—	—	—	X
—	20	50	—	—	—	—	—
—	—	5	—	—	—	—	—
—	5	15	—	—	—	—	—
—	0.1	0.4	—	—	—	—	—
—	10	45	20	90	—	—	—
—	—	0.1	—	—	—	—	—
—	—	0.001	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	0.2	—	—	—	—	—
—	0.05	0.4	—	—	—	—	—
(e)	—	6	—	—	—	—	—
(e)	—	6	—	—	—	—	—
(e)	—	0.05	—	—	—	—	—
(e)	—	0.1	—	—	—	—	—
(e)	—	0.1	—	—	—	—	—
(e)	—	0.05	—	—	—	—	—
(e)	—	0.1	—	—	—	—	—
(e)	—	3	—	—	—	—	—
(e)	—	6	—	—	—	—	—
(e)	—	3	—	—	—	—	—

Talc (containing asbestos)		(*)	(*)	(*)
Talc (containing no asbestos), respirable dust	14807-96-6	(*)	(*)	(*)
Tremolite		(*)	(*)	(*)
Silicon	7440-21-3			
Total dust		—	15	—
Respirable fraction		—	5	—
Silicon carbide	409-21-2			
Total dust		—	15	—
Respirable fraction		—	5	—
Silicon tetrahydride	7803-62-5	—	—	—
Silver, metal and soluble compounds (as Ag)	7440-22-4	—	0.01	—
Soapstone; see Silicates				
Sodium azide				
(as HN_3)	26628-22-8	—	—	—
(as NaN_3)		—	—	—
Sodium bisulfite	7631-90-5	—	—	—
Sodium fluoroacetate	62-74-8	—	0.05	X
Sodium hydroxide	1310-73-2	—	2	—
Sodium metabisulfite	7681-57-4	—	—	—
Starch	9005-25-8			
Total dust		—	15	—
Respirable fraction		—	5	—
Stibine	7803-52-3	0.1	0.5	—
Stoddard solvent	8052-41-3	500	2900	—
Strychnine	57-24-9	—	0.15	—
Styrene	100-42-5	(*)	(*)	—
Subtilisins (Proteolytic enzymes)	9014-01-1	—	—	—
Sucrose	57-50-1			
Total dust		—	15	—
Respirable fraction		—	5	—
Sulfur dioxide	7446-09-5	5	13	—
Sulfur hexafluoride	2551-62-4	1000	6000	—
Sulfuric acid	7664-93-9	—	1	—
Sulfur monochloride	10025-67-9	1	6	—
Sulfur pentafluoride	5714-22-7	0.025	0.25	—
Sulfur tetrafluoride	7783-60-0	—	—	—
Sulfuryl fluoride	2699-79-8	5	20	—
Sulprofos	35400-43-2	—	—	—
Systox, see Demeton				
2,4,5-T (2,4,5-Trichlorophenoxyacetic acid)	93-76-5	—	10	—
Talc; see Silicates				
Tantalum, metal and oxide dust	7440-25-7	—	5	—
TEDP (Sulfotep)	3689-24-5	—	0.2	X
Tellurium and compounds (as Te)	13494-80-9	—	0.1	—
Tellurium hexafluoride (as Te)	7783-80-4	0.02	0.2	—
Temephos	3383-96-8			
Total dust		—	15	—
Respirable fraction		—	5	—
TEPP (Tetraethyl pyrophosphate)	107-49-3	—	0.05	X
Terphenyls	26140-60-3	(C)1	(C)9	—
1,1,1,2-Tetrachloro- 2,2-difluoroethane	76-11-9	500	4170	—
1,1,2,2-Tetrachloro- 1,2-difluoroethane	76-12-0	500	4170	—
1,1,2,2-Tetrachloroethane	79-34-5	5	35	X
Tetrachloroethylene; see Perchloroethylene				
Tetrachloromethane; see Carbon tetrachloride				
Tetrachloronaphthalene	1335-88-2	—	2	X
Tetraethyl lead (as Pb)	78-00-2	—	0.075	X
Tetrahydrofuran	109-99-9	200	590	—
Tetramethyl lead, (as Pb)	75-74-1	—	0.075	X
Tetramethyl succinonitrile	3333-52-6	0.5	3	X
Tetranitromethane	509-14-8	1	8	—
Tetrasodium pyrophosphate	7722-88-5	—	—	—

| | | |

TABLE Z.—Longshoring & Marine

Substance	CAS No.[4]	Transitional Limits PEL[5] ppm[6]	Transitional Limits PEL[5] mg/m[3 b]	Skin Designation
Tetryl (2,4,6-Trinitrophenylmethylnitramine)	479-45-8	—	1.5	X
Thallium, soluble compounds (as Tl)	7440-28-0	—	0.1	X
4,4'-Thiobis (6-tert, Butyl-m-cresol)	96-69-5			
Total dust		—	15	—
Respirable fraction		—	5	—
Thioglycolic acid	68-11-1	—	—	—
Thionyl chloride	7719-09-7	—	—	—
Thiram	137-26-8	—	5	—
Tin, inorganic compounds (except oxides) (as Sn)	7440-31-5	—	2	—
Tin, organic compounds (as Sn)	7440-31-5	—	0.1	—
Tin oxide (as Sn)	21651-19-4	—	—	—
Titanium dioxide	13463-67-7			
Total dust		—	15	—
Toluene	108-88-3	[2]	[2]	[2]
Toluene-2,4-diisocyanate (TDI)	584-84-9	(C)0.02	(C)0.14	—
m-Toluidine	108-44-1	—	—	—
o-Toluidine	95-53-4	5	22	X
p-Toluidine	106-49-0	—	—	—
Toxaphene; see Chlorinated camphene				
Tremolite; see Silicates				
Tributyl phosphate	126-73-8	—	5	—
Trichloroacetic acid	76-03-9	—	—	—
1,2,4-Trichlorobenzene	120-82-1	—	—	—
1,1,1-Trichloroethane; see Methyl chloroform				
1,1,2-Trichloroethane	79-00-5	10	45	X
Trichloroethylene	79-01-6	[2]	[2]	—
Trichloromethane; see Chloroform				
Trichloronaphthalene	1321-65-9	—	5	X
1,2,3-Trichloropropane	96-18-4	50	300	—
1,1,2-Trichloro-1,2,2-trifluoroethane	76-13-1	1000	7600	—
Triethylamine	121-44-8	25	100	—
Trifluorobromomethane	75-63-8	1000	6100	—
Trimellitic anhydride	552-30-7	—	—	—
Trimethylamine	75-50-3	—	—	—
Trimethyl benzene	25551-13-7	—	—	—
Trimethyl phosphite	121-45-9	—	—	—
2,4,6-Trinitrophenyl; see Picric acid				
2,4,6-Trinitrophenylmethylnitramine; see Tetryl				
2,4,6-Trinitrotoluene (TNT)	118-96-7	—	1.5	X
Triorthocresyl phosphate	78-30-8	—	0.1	—
Triphenyl amine	603-34-9	—	—	—
Triphenyl phosphate	115-86-6	—	3	—
Tungsten (as W)	7440-33-7			
Insoluble compounds		—	—	—
Soluble compounds		—	—	—
Turpentine	8006-64-2	100	560	—
Uranium (as U)	7440-61-1			
Soluble compounds		—	0.05	—
Insoluble compounds		—	0.25	—
n-Valeraldehyde	110-62-3	—	—	—
Vanadium	1314-62-1			
Respirable dust (as V_2O_5)		—	(C)0.5	—
Fume (as V_2O_5)		—	(C)0.1	—
Vegetable oil mist				
Total dust		—	15	—
Respirable fraction		—	5	—
Vinyl acetate	108-05-4	—	—	—

arine Terminals—Continued

	Proposed Final Rule Limits						
	TWA		STEL [c]		CEILING		Skin Designation
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
X	—	1.5	—	—	—	—	X
X	—	0.1	—	—	—	—	X
	—	—	—	—	—	—	—
	—	10	—	—	—	—	—
	—	5	—	—	—	—	—
	1	4	—	—	—	—	X
	—	—	—	—	1	5	—
	—	5	—	—	—	—	—
	—	2	—	—	—	—	—
	—	0.1	—	—	—	—	X
	—	2	—	—	—	—	—
	—	10	—	—	—	—	—
[d]	100	375	150	560	—	—	—
	0.005	0.04	0.02	0.15	—	—	—
	2	9	—	—	—	—	X
X	5	22	—	—	—	—	X
	2	9	—	—	—	—	X
	—	—	—	—	—	—	—
	0.2	2.5	—	—	—	—	—
	1	7	—	—	—	—	—
	—	—	—	—	5	40	—
X	10	45	—	—	—	—	X
	50	270	200	1080	—	—	—
X	—	5	—	—	—	—	X
	10	60	—	—	—	—	—
	1000	7600	1250	9500	—	—	—
	10	40	15	60	—	—	—
	1000	6100	—	—	—	—	—
	0.005	0.04	—	—	—	—	—
	10	24	15	36	—	—	—
	25	125	—	—	—	—	—
	2	10	—	—	—	—	—
X	—	0.5	—	—	—	—	X
	—	0.1	—	—	—	—	X
	—	5	—	—	—	—	—
	—	3	—	—	—	—	—
	—	5	—	10	—	—	—
	—	1	—	3	—	—	—
	100	560	—	—	—	—	—
	—	0.05	—	—	—	—	—
	—	0.2	—	0.6	—	—	—
	50	175	—	—	—	—	—
	—	0.05	—	—	—	—	—
	—	0.05	—	—	—	—	—
	—	15	—	—	—	—	—
	—	5	—	—	—	—	—
	10	30	20	60	—	—	—

Vinyl benzene; see Styrene				
Vinyl bromide	593-60-2	—	—	—
Vinyl chloride [f]; see 1910.1017	75-01-4	[1]	[1]	[1]
Vinyl cyanide; see Acrylonitrile				
Vinyl cyclohexene dioxide	106-87-6	—	—	—
Vinylidene chloride (1,1-Dichloroethylene)	75-35-4	—	—	—
Vinyl toluene	26013-15-4	100	480	—
VM & P Naphtha	8032-32-4	—	—	—
Warfarin	81-81-2	—	0.1	—
Welding fumes (total particulate)**		—	—	—
Wood dust, all soft and hard woods, except Western red cedar		—	—	—
Wood dust, Western red cedar		—	—	—
Xylenes (o-, m-, p-isomers)	1330-20-7	100	435	—
m-Xylene alpha, alpha'diamine	1477-55-0	—	—	—
Xylidine	1300-73-8	5	25	X
Yttrium	7440-65-5	—	1	—
Zinc chloride fume	7646-85-7	—	1	—
Zinc chromate (as CrO_3)	13530-65-9	[3]	[3]	[3]
Zinc oxide fume	1314-13-2	—	5	—
Zinc oxide	1314-13-2			
Total dust		—	15	—
Respirable fraction		—	5	—
Zinc stearate	557-05-1			
Total dust		—	15	—
Respirable fraction		—	5	—
Zirconium compounds (as Zr)	7440-67-7	—	5	—

Footnotes:
[1] Same as Final Rule Limits.
[2] 1910.1000, Table Z-2 or Table Z-3.
[3] See discussion.
[4] See 1910.1001 for asbestiform; for non-asbestiform see 1910.1101.
[5] Use asbestos limit 1910.1001.
* The transitional PELs are 8-hour TWAs unless otherwise noted; a (C) designation denotes a ceiling limit.
** As determined from breathing-zone air samples.
[a] Parts of vapor or gas per million parts of contaminated air by volume at 25°C and 760 torr.
[b] Milligrams of substance per cubic meter of air. When entry is in this column only, the value is exact; when listi
[c] Duration is for 15 minutes, unless otherwise noted.
[d] The CAS number is for information only. Enforcement is based on the substance name. For an entry coverin
not CAS numbers for the individual compounds.
[e] Compliance with the subtilisins PEL is assessed by sampling with a high volume sampler (600–800 liters per m
[f] For those substances for which a full standard exists, exposure limits are provided in the table for inform
[g] For sectors excluded from 1910.1028 the limit is 10 ppm TWA and see 1910.1000, Table Z-2 for ceiling limits
[h] Where OSHA has published a proposal for a substance but has not issued a final rule, the proposal is refere
placed in the proposed final limit column. However, this is presented just for information purposes, and the substa

—	5	20	—	—	—	—	—
(1)	1	—	5	—	—	—	—
—	10	60	—	—	—	—	X
—	1	4	—	—	—	—	—
—	100	480	—	—	—	—	—
—	200	1350	490	1800	—	—	—
—	—	0.1	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	8	—	10	—	—	—
—	—	2.5	—	—	—	—	—
—	100	435	190	655	—	—	—
—	—	—	—	—	—	0.1	X
X	2	10	—	—	—	—	X
—	—	1	—	—	—	—	—
—	—	1	—	2	—	—	—
(2)	—	—	—	—	—	0.1	—
—	—	5	—	10	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	5	—	10	—	—	—

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en listed with a ppm entry, it is approximate.

overing more than one metal compound, measured as the metal, the CAS number for the metal is given—

per minute) for at least 60 minutes.
informational purposes, and the binding requirements are as set forth in the single-substance standard.
limits.
referenced and the existing limit is published in the transitional limit column, and the proposed final limit is
substances are not being considered in this rulemaking.

III. OSHA proposes to amend 29 CFR part 1918 as follows:

PART 1918—LONGSHORING

1. It is proposed to amend the authority citation for Part 1918 to read as follows:

Authority: Sec. 41, Longshore and Harbor Workers Compensation Act (33 U.S.C. 941); secs. 4, 6, 8, Occupational Safety and Health Act of 1970 (29 U.S.C. 653, 655, 657); Secretary of Labor's Order No. 12–71 (36 FR 8754), 8–76 (41 FR 25059), 9–83 (48 FR 35736) or 1–90 (55 FR 9033), as applicable.

Sections 1918.90 and 1918.92 also issued under 5 U.S.C. 553 and 29 CFR Part 1911.

2. It is proposed to amend § 1918.93 by revising paragraph (a)(1)(ii), and removing paragraphs (e) and (f) as follows:

§ 1918.93 Ventilation and atmospheric conditions.

(a) Ventilation requirements with respect to carbon monoxide:

(1) (i) * * *

(ii) The carbon monoxide content of the atmosphere in a compartment, room, building, hold, vehicle, railcar or any enclosed space shall be maintained at not more than 35 ppm per million (0.0035%) as an 8-hour time weighted average and employees shall be removed from the enclosed space if the carbon monoxide concentration exceeds 100 parts per million (0.01%). The short term exposure limit in outdoors, non-enclosed spaces shall be 200 ppm (0.02%) measured over a 5 minute period.

* * * * *

Subparts K–Y—[Reserved]

3. It is proposed to reserve subparts K–Y and to add a new subpart Z consisting of § 1918.1000 to read as follows:

Subpart Z—Toxic and Hazardous Substances

§ 1918.1000 Air contaminants.

(a) *Exposure limits.* An employees exposure to any substance listed in Table Z, Longshoring & Marine Terminals (located at 29 CFR 1917.1000) shall be limited in accordance with the requirements of the following paragraphs of this section:

(1) Final Rule Limits Columns. An employee's exposure to any substance listed in Table Z, Longshoring & Marine Terminals shall not exceed the Time Weighted Average (TWA), Short Term Exposure Limit (STEL) and Ceiling Limit specified for that substance in Table Z—Longshoring and Marine Terminals under the Final Rule Limits columns.

(2) Skin Designation. To prevent or reduce skin absorption, an employee's skin exposure to substances listed in Table Z, Marine Terminals & Longshoring with an "X" in one or both of the Skin Designation columns following the substances name shall be prevented or reduced to the extent necessary in the circumstances through the use of gloves, coveralls, goggles, or other appropriate personal protective equipment, engineering controls or work practices.

(3) Definitions. The following definitions are applicable to Table Z, Longshoring & Marine Terminals:

(i) *Time weighted average (TWA)* is the employee's average airborne exposure in any 8-hour work shift of a 40-hour work week which shall not be exceeded.

(ii) *Short term exposure limit (STEL)* is the employee's 15-minute time weighted average exposure which shall not be exceeded at any time during a work day unless another time limit is specified in a parenthetical notation below the limit. If another time period is specified, the time weighted average exposure over that time period shall not be exceeded at any time during the working day.

(iii) *Ceiling* is the employee's exposure which shall not be exceeded during any part of the work day. If instantaneous monitoring is not feasible, then the ceiling shall be assessed as a 15-minute time weighted average exposure which shall not be exceeded at my time over a working day.

(b) *Computation formulae.* The computation formula which shall apply to employee exposure to more than one substance for which 8-hour time weighted averages are listed in subpart Z of 29 CFR Part 1918 in order to determine whether an employee is exposed over the regulatory limit is as follows:

(1)(i) The cumulative exposure for an 8-hour work shift shall be computed as follows:

$$E=(C_aT_a+C_bT_b+ \ldots C_nT_n)\div 8$$

Where.

E is the equivalent exposure for the working shift.

C is the concentration during any period of time T where the concentration remains constant.

T is the duration in hours of the exposure at the concentration C.

The value of E shall not exceed the 8-hour time weighted average specified in Subpart Z or 29 CFR Part 1918 for the material involved.

(ii) To illustrate the formula prescribed in paragraph (d)(1)(i) of this section, assume that Substance A has an 8-hour time weighted average limit of 100 ppm noted in Table Z. Assume that an employee is subject to the following exposure:

Two hours exposure at 150 ppm

Two hours exposure at 75 ppm

Four hours exposure at 50 ppm

Substituting this information in the formula, we have

$$(2\times 150+2\times 75+4\times 50)\div 8=81.25 \text{ ppm}$$

Since 81.25 ppm is less than 100 ppm, the 8-hour time weighted average limit, the exposure is acceptable.

(2)(i) in case of a mixture of air contaminants an employer shall compute the equivalent exposure as follows:

$$E_m=(C_1\div L_a+C_2\div L_a)+ \ldots (C_n\div L_n)$$

Where:

E_m is the equivalent exposure for the mixture.

C is the concentration of a particular contaminant.

L is the exposure limit for that substance specified in Subpart Z of 29 CFR Part 1918.

The value of E_m shall not exceed unity (1).

(ii) To illustrate the formula prescribed in paragraph (d)(2)(i) of this section, consider the following exposures:

Substance	Actual concentration of 8 hour exposure (ppm)	8 hr. TWA PEL (ppm)
B	500	1,000
C	45	200
D	40	200

Substituting in the formula we have:

$$Em=500\div 1,000+45\div 200+40\div 200$$
$$Em=0.500+0.225+0.200$$
$$Em=0.925$$

Since Em is less than unity (1), the exposure combination is within acceptable limits.

(c) *Methods of compliance.* To achieve compliance with paragraph (a) of this section administrative or engineering controls must first be implemented whenever feasible. When such controls are not feasible to achieve full compliance, protective equipment or other protective measures shall be used to keep the exposure of employees to air contaminants within the limits prescribed in this section. Any equipment and technical measures used for this purpose must first be approved for each particular use by a competent industrial hygienist or other technically qualified person.

(d) *Sealed containers.* (1) When a substance or cargo is contained within a sealed, intact means of packaging or containment complying with Department of Transportation or International Maritime Organization requirements then the rest of this section is not applicable.

(2) When sealed, intact means of packaging complying with Department of Transportation or International Maritime Organization requirements containing substances or cargo break, leak or are damaged so that there is a reasonable possibility of leakage, the exposure limits specified 29 CFR Part 1918, Subpart Z are applicable. These exposure limits shall be met by my reasonable combination of engineering controls, work practices and personal protective equipment.

(e) *Effective and start-up dates.* (1) *Effective date.* The effective date for the permissible exposure limits specified in the Final Rule Limits columns of Table Z, Longshoring & Marine Terminals is [90 days after date of publication in the **Federal Register**].

(2) Start-up dates. (i) The permissible exposure limits specified in the Final Rule Limits columns of Table Z, Longshoring & Marine Terminals shall be achieved by any reasonable combination of engineering controls, work practices and personal protective equipment from the effective date through [4 years after date of publication in the **Federal Register**].

(ii) The permissible exposure limits specified in the Final Rule Limits columns of Table Z, Longshoring & Marine Terminals shall be achieved by the methods of compliance specified in paragraph (c) of this section commencing [4 years after date of publication in the **Federal Register**].

(iii) The skin designation in the Final Rule Limits columns shall be complied with commencing on the effective date.

(3) Transitional provision. The permissible exposure limits specified in the Transitional Limits columns of Table Z, Longshoring & Marine Terminals (which are Time Weighted Averages unless proceeded by a "C" in which case they are ceilings) shall continue to be achieved by the methods of compliance specified in paragraph (c) of this section through [4 years after date of publication in the **Federal Register**].

(4) If any new or amended provisions or new or revised limits for any substance or substances are either administratively stayed or judicially stayed or vacated, then the existing provisions or limits for those substances specified in the Transitional Limits columns of Table Z, Longshoring & Marine Terminals shall remain in effect until such stay is lifted, or indefinitely if the limit is vacated.

IV. OSHA proposes to amend 29 CFR part 1926 as follows:

PART 1926—CONSTRUCTION

1. It is proposed to revise the authority citation for 29 CFR part 1926, subpart D as follows:

Authority: Sec. 107, Contract Work Hours and Safety Standards Act (Construction Safety Act) (40 U.S.C. 333); secs. 4, 6, 8, Occupational Safety and Health Act of 1970 (29 U.S.C. 653, 655, 657); Secretary of Labor's Order No. 12–71 (36 FR 8754), 8–76 (41 FR 25059), 9–83 (48 FR 35736) or 1–90 (55 FR 9033) as applicable.

Sections 1926.55 and 1926.59 also issued under 5 U.S.C. 553 and 29 CFR Part 1911.

2. It is proposed to revise § 1926.55 to read as follows:

§ 1926.55 Air contaminants.

(a) *Exposure limits.* Employers shall prevent exposure of employees to inhalation of substances above the exposure limits specified in Table Z, Construction and minimize skin absorption as follows:

(1) An employee's exposure to any substance in Table Z, Construction under the Transitional Limits columns, the exposure limit of which is preceded by a "C" shall at no time exceed the exposure limit given for that substance in Table Z, Construction under the Transitional Limits columns.

(2) An employee's exposure to any substance in Table Z, Construction under the Transitional Limits columns, the exposure limit of which is not preceded by a "C", shall not exceed the 8-hour time Weighted Average given for that substance in Table Z, Construction under the Transitional Limits columns in any 8-hour work shift of a 40-hour work week.

(3) An employee's exposure to any substance listed in Table Z, Construction shall not exceed the Time Weighted Average (TWA), Short Term Exposure Limit (STEL) and Ceiling Limit specified for that substance in Table Z, Construction under the Final Rule Limits columns.

(4) To prevent or reduce skin absorption, an employee's skin exposure to substances listed in Table Z, Construction with an "X" in one or both of the Skin Designation columns following the substance name shall be prevented or reduced to the extent necessary in the circumstances through the use of gloves, coveralls, goggles, or other appropriate personal protective equipment, engineering controls or work practices.

(5) The following definitions are applicable to the Final Rule Limits columns of Table Z, Construction:

(i) *Time weighted average (TWA)* is the employee's average airborne exposure in any 8-hour work shift of a 40-hour work week which shall not be exceeded.

(ii) *Short term exposure limit (STEL)* is the employee's 15-minute time weighted average exposure which shall not be exceeded at any time during a work day unless another time limit is specified in a parenthetical notation below the limit. If another time period is specified, the time weighted average exposure over that time period shall not be exceeded at any time during the working day.

(iii) *Ceiling* is the employee's exposure which shall not be exceeded during any part of the work day. If instantaneous monitoring is not feasible, then the ceiling shall be assessed as a 15-minute time weighted average exposure which shall not be exceeded at any time over a working day..

(b) *Methods of compliance.* To achieve compliance with paragraph (a) of this section, administrative or engineering controls must first be implemented whenever feasible. When such controls are not feasible to achieve full compliance, protective equipment or other protective measures shall be used to keep the exposure of employees to air contaminants within the limits prescribed in this section. Any equipment and technical measures used for this purpose must first be approved for each particular use by a competent industrial hygienist or other technically qualified person. Whenever respirators are used, their use shall comply with § 1926.103.

(c) *Computation formulae.* The computation formula which shall apply to employee exposure to more than one substance for which 8-hour time weighted averages are listed in Table Z, Construction in order to determine whether an employee is exposed over the regulatory limit is as follows:

(1)(i) The cumulative exposure for an 8-hour work shift shall be computed as follows:

$$E=(C_aT_a+C_bT_b+\ldots C_nT_n)\div 8$$

Where:

E is the equivalent exposure for the working shift.

C is the concentration during any period of time T where the concentration remains constant.

T is the duration in hours of the exposure at the concentration C.

The value of E shall not exceed the 8-hour time weighted average specified in Table Z, Construction for the material involved.

(ii) To illustrate the formula prescribed in paragraph (d)(1)(i) of this section, assume that Substance A has an 8-hour time weighted average limit of 100 ppm noted in Table Z. Assume that an employee is subject to the following exposure:

Two hours exposure at 150 ppm
Two hours exposure at 75 ppm
Four hours exposure at 50 ppm

Substituting this information in the formula, we have:

$(2\times150+2\times75+4\times50)\div8=81.25$ ppm

Since 81.25 ppm is less than 100 ppm, the 8-hour time weighted average limit, the exposure is acceptable.

(2)(i) In case of a mixture of air contaminants an employer shall compute the equivalent exposure as follows:

$E_m=(C_1\div L_1+C_2\div L_2)+\ldots(C_n\div L_n)$

Where:

E_m is the equivalent exposure for the mixture.
C is the concentration of a particular contaminant.
L is the exposure limit for that substance specified in Table Z, Construction.

The value of E_m shall not exceed unity (1).

(ii) To illustrate the formula prescribed in paragraph (d)(2)(i) of this section, consider the following exposures:

Substance	Actual concentration of 8 hour exposure (ppm)	8 hr. TWA PEL (ppm)
B	500	1000
C	45	200
D	40	200

Substituting in the formula we have:

$Em=500\div1,000+45\div200+40\div200$
$Em=0.500+0.225+0.200$
$Em=0.925$

Since Em is less than unity (1), the exposure combination is within acceptable limits.

(d) *Effective and start-up dates—(1) Effective date.* The effective date for the permissible exposure limits specified in the Final Rule Limits columns of Table Z, Construction is [90 days after date of publication in the Federal Register].

(2) Start-up dates. (i) The permissible exposure limits specified in the Final Rule Limits columns of Table Z, Construction shall be achieved by any reasonable combination of engineering controls, work practices and personal protective equipment from the effective date through [4 years after date of publication in the Federal Register].

(ii) The permissible exposure limits specified in the Final Rule Limits columns of Table Z, Construction shall be achieved by the method of compliance specified in paragraph (b) of this section commencing [4 years after date of publication in the Federal Register].

(iii) The skin designations in the Final Rule Limits columns shall be complied with commencing on the effective date.

(3) Transitional provision. The permissible exposure limits specified in the Transitional Limits columns of Table Z, Construction shall continue to be achieved by the methods of compliance specified in paragraph (b) of this section through [4 years after the date of publication in the Federal Register].

(4) If any new or amended provisions or new or revised limits for any substance or substances are either administratively stayed or judicially stayed or vacated, then the existing provisions or limits for those substances specified in the Transitional Limits columns of Table Z, Construction shall remain in effect until such stay is lifted, or indefinitely if the limit is vacated.

TABLE Z.—Cons

Substance	CAS No.[a]	Transitional Limits[a] ppm[a]	(1970 TLVs) mg/m[3][b]	Skin Designation
Abate; see Temephos				
Acetaldehyde	75-07-0	200	360	—
Acetic acid	64-19-7	10	25	—
Acetic anhydride	108-24-7	5	20	—
Acetone	67-64-1	1000	2400	—
Acetonitrile	75-05-8	40	70	—
2-Acetylaminofluorine; see 1910.1014	53-96-3			
Acetylene	74-86-2	E		
Acetylene dichloride; see 1,2-Dichloroethylene				
Acetylene tetrabromide	79-27-6	1	14	—
Acetylsalicylic acid (Aspirin)	50-78-2	—	—	—
Acrolein	107-02-8	0.1	0.25	—
Acrylamide	79-06-1	—	0.3	X
Acrylic acid	79-10-7	—	—	—
Acrylonitrile [f]; see 1910.1045	107-13-1	(1)	(1)	X
Aldrin	309-00-2	—	0.25	X
Allyl alcohol	107-18-6	2	5	X
Allyl chloride	107-05-1	1	3	—
Allyl glycidyl ether (AGE)	106-92-3	(C)10	(C)45	—
Allyl propyl disulfide	2179-59-1	2	12	—
alpha-Alumina	1344-28-1			
Total dust		—	15	—
Respirable fraction		—	5	—
Aluminum (as Al)	7429-90-5			
Metal				
Total dust		—	15	—
Respirable fraction		—	5	—
Pyro powders		—	—	—
Welding fumes[c][e]		—	—	—
Soluble salts		—	—	—
Alkyls		—	—	—
Alundum; see alpha-Alumina				
4-Aminodiphenyl; see 1910.1011	92-67-1			
2-Aminoethanol; see Ethanolamine				
2-Aminopyridine	504-29-0	0.5	2	—
Amitrole	61-82-5	—	—	—
Ammonia	7664-41-7	50	35	—
Ammonium chloride fume	12125-02-9	—	—	—
Ammonium sulfamate	7773-06-0			
Total dust		—	15	—
Respirable fraction		—	5	—
n-Amyl acetate	628-63-7	100	525	—
sec-Amyl acetate	626-38-0	125	650	—
Aniline and homologs	62-53-3	5	19	X
Anisidine (o-, p-isomers)	29191-52-4	—	0.5	X
Antimony and compounds (as Sb)	7440-36-0	—	0.5	—
ANTU (alpha Naphthylthiourea)	86-88-4	—	0.3	—
Argon	7440-37-1	E	—	—
Arsenic, inorganic compounds (as As) [f]; see 1910.1018	7440-38-2	—	0.01	—
Arsenic, organic compounds (as As)	7440-38-2	—	0.5	—
Arsine	7784-42-1	0.05	0.2	—
Asbestos [f]; see 1926.58	Varies	(1)	(1)	(1)
Asphalt (Petroleum) fumes	8052-42-4	—	—	—
Atrazine	1912-24-9	—	—	—

Construction

Proposed Final Rule Limits						
TWA		STEL [a]		CEILING		Skin Designation
ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
100	180	150	270	—	—	—
10	25	—	—	—	—	—
—	—	—	—	5	20	—
750	1800	1000	2400	—	—	—
40	70	60	105	—	—	—
1	14	—	—	—	—	—
—	5	—	—	—	—	—
0.1	0.25	0.3	0.8	—	—	X
—	0.03	—	—	—	—	X
10	30	—	—	—	—	X
2	—	10	—	—	—	X
—	0.25	—	—	—	—	X
2	5	4	10	—	—	—
1	3	2	6	—	—	—
5	22	10	44	—	—	—
2	12	3	18	—	—	—
—	10	—	—	—	—	—
—	5	—	—	—	—	—
—	15	—	—	—	—	—
—	5	—	—	—	—	—
—	5	—	—	—	—	—
—	5	—	—	—	—	—
—	2	—	—	—	—	—
—	2	—	—	—	—	—
0.5	2	—	—	—	—	—
—	0.2	—	—	—	—	—
—	—	35	27	—	—	—
—	10	—	20	—	—	—
—	10	—	—	—	—	—
—	5	—	—	—	—	—
100	525	—	—	—	—	—
125	650	—	—	—	—	—
2	8	—	—	—	—	X
—	0.5	—	—	—	—	X
—	0.5	—	—	—	—	—
—	0.3	—	—	—	—	—
—	0.01	—	—	—	—	—
—	0.5	—	—	—	—	—
0.05	0.2	—	—	—	—	—
(1)	0.2 f/cc	—	1 f/cc (30 min.)	—	—	—
—	5	—	—	—	—	—
—	5	—	—	—	—	—

TABLE Z.—Constructio

Substance	CAS No.[4]	Transitional Limits[a] ppm [a]	(1970 TLVs) mg/m [3 b]	Skin Designation
Azinphos-methyl	86-50-0	—	0.2	X
Barium, soluble compounds (as Ba)	7440-39-3	—	0.5	—
Barium sulfate	7727-43-7			
Total dust		—	15	—
Respirable fraction		—	5	—
Benomyl	17804-35-2			
Total dust		—	15	—
Respirable fraction		—	5	—
Benzene [c, e]; see 1910.1028	71-43-2	([1])	([1])	([1])
Benzidine; see 1910.1010	92-87-5		A [1]	
p-Benzoquinone; see Quinone				
Benzo(a)pyrene; see Coal tar pitch volatiles				
Benzoyl peroxide	94-36-0	—	5	—
Benzyl chloride	100-44-7	1	5	—
Beryllium and beryllium compounds (as Be)	7440-41-7	—	0.002	—
Biphenyl; see Diphenyl				
Bismuth telluride, Undoped	1304-82-1			
Total dust		—	15	—
Respirable fraction		—	5	—
Bismuth telluride, Se-doped	1304-82-1	—	—	—
Bisphenol A; see Diglycidyl ether				
Borates, tetra, sodium salts				
Anhydrous	1330-43-4	—	—	—
Decahydrate	1303-96-4	—	—	—
Pentahydrate	12179-04-3	—	—	—
Boron oxide	1303-86-2			
Total dust		—	15	—
Boron tribromide	10294-33-4	1	10	—
Boron trifluoride	7637-07-2	(C)1	(C)3	—
Bromacil	314-40-9	—	—	—
Bromine	7726-95-6	0.1	0.7	—
Bromine pentafluoride	7789-30-2	0.1	0.7	—
Bromoform	75-25-2	0.5	5	X
Butadiene (1,3-Butadiene) [b]; see 55 FR 32736	106-99-0	1000	2200	—
Butane	106-97-8	—	—	—
Butanethiol; see Butyl mercaptan				
2-Butanone (Methyl ethyl ketone)	78-93-3	200	590	—
2-Butoxyethanol	111-76-2	50	240	X
n-Butyl-acetate	123-86-4	150	710	—
sec-Butyl acetate	105-46-4	200	950	—
tert-Butyl acetate	540-88-5	200	950	—
Butyl acrylate	141-32-2	—	—	—
n-Butyl alcohol	71-36-3	100	300	—
sec-Butyl alcohol	78-92-2	150	450	—
tert-Butyl alcohol	75-65-0	100	300	—
Butylamine	109-73-9	(C)5	(C)15	X
tert-Butyl chromate (as CrO_3)	1189-85-1	—	(C)0.1	X
n-Butyl glycidyl ether (BGE)	2426-08-6	50	270	—
n-Butyl lactate	138-22-7	—	—	—
Butyl mercaptan	109-79-5	0.5	1.5	—
o-sec-Butylphenol	89-72-5	—	—	—
p-tert-Butyltoluene	98-51-1	10	60	—

...action—Continued

	Proposed Final Rule Limits						
	TWA		STEL [c]		CEILING		Skin Designation
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
X	—	0.2	—	—	—	—	X
	—	0.5	—	—	—	—	
	—	10	—	—	—	—	
	—	5	—	—	—	—	
	—	10	—	—	—	—	
	—	5	—	—	—	—	
[1])	1	—	5	—	—	—	
	—	5	—	—	—	—	
	1	5	—	—	—	—	
	—	0.002	—	0.025 (30 min.)	—	—	
	—	15	—	—	—	—	
	—	5	—	—	—	—	
	5	—	—	—	—	—	
	—	10	—	—	—	—	
	—	10	—	—	—	—	
	—	10	—	—	—	—	
	—	10	—	—	1	10	
	—	—	—	—	1	3	
	1	10	—	—	—	—	
	0.1	0.7	0.3	2	—	—	
	0.1	0.7	—	—	—	—	
X	0.5	5	—	—	—	—	X
	2	—	10	—	—	—	
	800	1900	—	—	—	—	
	200	590	300	885	—	—	
X	25	120	—	—	—	—	X
	150	710	200	950	—	—	
	208	958	—	—	—	—	
	200	960	—	—	—	—	
	10	55	—	—	—	—	X
	—	—	—	—	50	150	
	100	365	—	—	—	—	
	100	300	150	450	—	—	
X	—	—	—	—	5	15	X
X	—	—	—	—	—	0.1	X
	25	135	—	—	—	—	
	5	25	—	—	—	—	
	0.5	1.5	—	—	—	—	X
	5	30	—	—	—	—	
	10	60	20	120	—	—	

Cadmium fume (as Cd) [b]; see 55 FR 4052	7440-43-9	—	(C)0.1	—
Cadmium dust (as Cd) [b]; see 55 FR 4052	7440-43-9	—	0.2	—
Calcium carbonate	1317-65-3	—		
Total dust		—	15	—
Respirable fraction		—	5	—
Calcium cyanamide	156-62-7	—	—	—
Calcium hydroxide	1305-62-0	—	—	—
Calcium hydroxide				
Total dust		—	15	—
Respirable fraction		—	5	—
Calcium oxide	1305-78-8	—	5	—
Calcium silicate	1344-95-2			
Total dust		—	15	—
Respirable fraction		—	5	—
Calcium sulfate	7778-18-9			
Total dust		—	15	—
Respirable fraction		—	5	—
Camphor, synthetic	76-22-2	—	2	—
Caprolactam	105-60-2			
Dust		—	—	—
Vapor		—	—	—
Captafol (Difolatan)	2425-06-1	—	—	—
Captan	133-06-2	—	—	—
Carbaryl (Sevin)	63-25-2	—	5	—
Carbofuran (Furadan)	1563-66-2	—	—	—
Carbon black	1333-86-4	—	3.5	—
Carbon dioxide	124-38-9	5000	30000	—
Carbon disulfide	75-15-0	20	60	X
Carbon monoxide	630-08-0	50	55	—
Carbon tetrabromide	558-13-4	—	—	—
Carbon tetrachloride	56-23-5	10	25	X
Carbonyl fluoride	353-50-4	—	—	—
Catechol (Pyrocatechol)	120-80-9	—	—	—
Cellulose	9004-34-6			
Total dust		—	15	—
Respirable fraction		—	5	—
Cesium hydroxide	21351-79-1	—	—	—
Chlordane	57-74-9	—	0.5	X
Chlorinated camphene	8001-35-2	—	0.5	X
Chlorinated diphenyl oxide	55720-99-5	—	0.5	—
Chlorine	7782-50-5	1	3	—
Chlorine dioxide	10049-04-4	0.1	0.3	—
Chlorine trifluoride	7790-91-2	(C)0.1	(C)0.4	—
Chloroacetaldehyde	107-20-0	(C)1	(C)3	—
a-Chloroacetophenone (Phenacyl chloride)	532-27-4	0.05	0.3	—
Chloroacetyl chloride	79-04-9	—	—	—
Chlorobenzene	108-90-7	75	350	—
o-Chlorobenzylidene malononitrile	2698-41-1	0.05	0.4	—
Chlorobromomethane	74-97-5	200	1050	—
2-Chloro-1,3-butadiene; see beta-Chloroprene				
Chlorodifluoromethane	75-45-6	—	—	—
Chlorodiphenyl (42% Chlorine) (PCB)	53469-21-9	—	1	X
Chlorodiphenyl (54% Chlorine) (PCB)	11097-69-1	—	0.5	X
1-Chloro,2,3-epoxypropane; see Epichlorohydrin				
2-Chloroethanol; see Ethylene chlorohydrin				
Chloroethylene; see Vinyl chloride				
Chloroform (Trichloromethane)	67-66-3	50	240	—
bis(Chloromethyl) ether; see 1910.1008	542-88-1			
Chloromethyl methyl ether; see 1910.1006	107-30-2			
1-Chloro-1-nitropropane	600-25-9	20	100	—

| | | |

TABLE Z.—Construct

Substance	Transitional Limits[a]		(1970 TLVs)	Skin Designation
	CAS No.[d]	ppm[e]	mg/m³[b]	
Chloropentafluoroethane	76-15-3	—	—	—
Chloropicrin	76-06-2	0.1	0.7	—
beta-Chloroprene	126-99-8	25	90	X
o-Chlorostyrene	2039-87-4	—	—	—
o-Chlorotoluene	95-49-8	—	—	—
2-Chloro-6-(trichloromethyl) pyridine	1929-82-4			
Total dust		—	15	—
Respirable fraction		—	5	—
Chlorpyrifos	2921-88-2	—	—	—
Chromic acid and chromates				
(as CrO_3)	Varies with compound	—	0.1	—
Chromium (II) compounds				
(as Cr)	7440-47-3	—	0.5	—
Chromium (III) compounds				
(as Cr)	7440-47-3	—	0.5	—
Chromium metal and insol. salts (as Cr)	7440-47-3	—	1	—
Chrysene; see Coal tar pitch volatiles				
Clopidol	2971-90-6			
Total dust		—	15	—
Respirable fraction		—	5	—
Coal dust (less than 5% SiO_2), Respirable fraction		—	—	—
Coal dust (greater than or equal to 5% SiO_2), Respirable quartz fraction		—	—	—
Coal tar pitch volatiles (benzene soluble fraction), anthracene, BaP, phenanthrene, acridine, chrysene, pyrene	65065-93-2	—	0.2	—
Cobalt metal, dust, and fume (as Co)	7440-48-4	—	0.1	—
Cobalt carbonyl (as Co)	10210-68-1	—	—	—
Cobalt hydrocarbonyl (as Co)	16842-03-8	—	—	—
Coke oven emissions[f]; see 1910.1029		—	0.15	—
Copper	7440-50-8			
Fume (as Cu)		—	0.1	—
Dusts and mists (as Cu)		—	1	—
Corundum; see Emery				
Cotton dust[f]; see 1910.1043		—	1	—
This 8-hour TWA applies to respirable dust as measured by a vertical elutriator cotton dust sampler or equ respirable dust personal sampler may be used. In these circumstances the exposure limit is 1 mg/m³ respirab				
Crag herbicide (Sesone)	136-78-7			
Total dust		—	15	—
Respirable fraction		—	5	—
Cresol, all isomers	1319-77-3	5	22	X
Crotonaldehyde	123-73-9; 4170-30-3	2	6	—
Crufomate	299-86-5	—	—	—
Cumene	98-82-8	50	245	X
Cyanamide	420-04-2	—	—	—
Cyanides (as CN)	Varies with Compound	—	5	—
Cyanogen	460-19-5	10	—	—
Cyanogen chloride	506-77-4	—	—	—
Cyclohexane	110-82-7	300	1050	—
Cyclohexanol	108-93-0	50	200	—
Cyclohexanone	108-94-1	50	200	—
Cyclohexene	110-83-8	300	1015	—
Cyclohexylamine	108-91-8	—	—	—
Cyclonite	121-82-4	—	1.5	X

struction—Continued

kin sig- tion	Proposed Final Rule Limits						Skin Desig- nation
	TWA		STEL [c]		CEILING		
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
—	1000	6320	—	—	—	—	—
—	0.1	0.7	—	—	—	—	—
X	10	35	—	—	—	—	X
—	50	285	75	428	—	—	—
—	50	250	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	0.2	—	—	—	—	X
—	—	—	—	—	—	0.1	—
—	—	0.5	—	—	—	—	—
—	—	0.5	—	—	—	—	—
—	—	1	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	2	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	0.2	—	—	—	—	—
—	—	0.05	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	0.15	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	1	—	—	—	—	—
—	—	0.5	—	—	—	—	—

or equivalent instrument. For the Transitional Limit and where it is not feasible to use a vertical elutriator, a espirable dust, personal sampler

—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
X	5	22	—	—	—	—	X
—	2	6	—	—	—	—	—
—	—	5	—	—	—	—	—
X	50	245	—	—	—	—	X
—	—	2	—	—	—	—	—
—	—	5	—	—	—	—	—
—	10	20	—	—	—	—	—
—	—	—	—	—	0.3	0.6	—
—	300	1050	—	—	—	—	—
—	50	200	—	—	—	—	X
—	25	100	—	—	—	—	X
—	300	1015	—	—	—	—	—
—	10	40	—	—	—	—	—
X	—	1.5	—	—	—	—	X

Cyclopentadiene	542-92-7	75	200	—
Cyclopentane	287-92-3	—	—	—
Cyhexatin	13121-70-5	—	—	—
2,4-D (Dichlorophenoxyacetic acid)	94-75-7	—	10	—
Decaborane	17702-41-9	0.05	0.3	X
Demeton (Systox)	8065-48-3	—	0.1	X
Diacetone alcohol (4-Hydroxy-4-methyl-2-pentanone)	123-42-2	50	240	—
1,2-Diaminoethane; see Ethylenediamine				
Diazinon	333-41-5	—	—	—
Diazomethane	334-88-3	0.2	0.4	—
Diborane	19287-45-7	0.1	0.1	—
1,2-Dibromo-3-chloropropane (CBCP) [f]; see 1910.1044	96-12-8	0.001	—	X
1,2-Dibromoethane; see Ethylene dibromide				
2-N-Dibutylaminoethanol	102-81-8	—	—	—
Dibutyl phosphate	107-66-4	1	5	—
Dibutyl phthalate	84-74-2	—	5	—
Dichloroacetylene	7572-29-4	(C)0.1	(C)0.4	—
o-Dichlorobenzene	95-50-1	(C)50	(C)300	—
p-Dichlorobenzene	106-46-7	75	450	—
3,3'-Dichlorobenzidine; see 1910.1007	91-94-1			
Dichlorodifluoromethane	75-71-8	1000	4950	—
1,3-Dichloro-5,5-dimethyl hydantoin	118-52-5	—	0.2	—
Dichlorodiphenyltrichloroethane (DDT)	50-29-3	—	1	X
1,1-Dichloroethane	75-34-3	100	400	—
1,2-Dichloroethane; see Ethylene dichloride				
1,2-Dichloroethylene	540-59-0	200	790	—
Dichloroethyl ether	111-44-4	(C)15	(C)90	X
Dichloromethane; see Methylene chloride				
Dichloromonofluoromethane	75-43-4	1000	4200	—
1,1-Dichloro-1-nitroethane	594-72-9	(C)10	(C)60	—
1,2-Dichloropropane; see Propylene dichloride				
1,3-Dichloropropene	542-75-6	—	—	—
2,2-Dichloropropionic acid	75-99-0	—	—	—
Dichlorotetrafluoroethane	76-14-2	1000	7000	—
Dichlorvos (DDVP)	62-73-7	—	1	X
Dicrotophos	141-66-2	—	—	—
Dicyclopentadiene	77-73-6	—	—	—
Dicyclopentadienyl iron	102-54-5			
Total dust		—	15	—
Respirable fraction		—	5	—
Dieldrin	60-57-1	—	0.25	X
Diethanolamine	111-42-2	—	—	—
Diethylamine	109-89-7	25	75	—
2-Diethylaminoethanol	100-37-8	10	50	X
Diethylene triamine	111-40-0	(C)10	(C)42	X
Diethyl ether; see Ethyl ether				
Diethyl ketone	96-22-0	—	—	—
Diethyl phthalate	84-66-2	—	—	—
Difluorodibromomethane	75-61-6	100	860	—
Diglycidyl ether (DGE)	2238-07-5	(C)0.5	(C)2.8	—
Dihydroxybenzene; see Hydroquinone				
Diisobutyl ketone	108-83-8	50	290	—
Diisopropylamine	108-18-9	5	20	X
4-Dimethylaminoazobenzene; see 1910.1015	60-11-7			
Dimethoxymethane; see Methylal				
Dimethyl acetamide	127-19-5	10	35	X
Dimethylamine	124-40-3	10	18	—
Dimethylaminobenzene; see Xylidine				
Dimethylaniline (N,N-Dimethylaniline)	121-69-7	5	25	X
Dimethylbenzene; see Xylene				
Dimethyl-1,2-dibromo- 2,2-dichloroethyl phosphate	300-76-5	—	3	—
Dimethylformamide	68-12-2	10	30	X
2,6-Dimethyl-4-heptanone; see Diisobutyl ketone				
1,1-Dimethylhydrazine	57-14-7	0.5	1	X

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TABLE Z.—Constructio

Substance	CAS No.[c]	Transitional Limits[a] (1970 TLVs) ppm[a]	mg/m³[b]	Skin Designation
Dimethylphthalate	131-11-3	—	5	—
Dimethyl sulfate	77-78-1	1	5	X
Dinitolmide (3,5- Dinitro-o-toluamide)	148-01-6	—	—	—
Dinitrobenzene (all isomers)				
(ortho)	528-29-0	—	1	X
(meta)	99-65-0			
(para)	100-25-4			
Dinitro-o-cresol	534-52-1	—	0.2	X
Dinitrotoluene	25321-14-6	—	1.5	X
Dioxane (Diethylene dioxide)	123-91-1	100	360	X
Dioxathion (Delnav)	78-34-2	—	—	—
Diphenyl (Biphenyl)	92-52-4	0.2	1	—
Diphenylamine	122-39-4	—	10	—
Diphenylmethane diisocyanate; see Methylene bisphenyl isocyanate				
Dipropylene glycol methyl ether	34590-94-8	100	600	X
Dipropyl ketone	123-19-3	—	—	—
Diquat	85-00-7	—	—	—
Di-sec octyl phthalate (Di-(2-ethylhexyl) phthalate)	117-81-7	—	5	—
Disulfiram	97-77-8	—	—	—
Disulfoton	298-04-4	—	—	—
2,6-Di-tert-butyl-p-cresol	128-37-0	—	—	—
Diuron	330-54-1	—	—	—
Divinyl benzene	1321-74-0	—	—	—
Emery	12415-34-8	—		
Total dust		—	15	—
Respirable fraction		—	5	—
Endosulfan	115-29-7	—	0.1	X
Endrin	72-20-8	—	0.1	X
Epichlorohydrin	106-89-8	5	19	X
EPN	2104-64-5	—	0.5	X
1,2-Epoxypropane; see Propylene oxide				
2,3-Epoxy-1-propanol; see Glycidol				
Ethane	74-84-0	E		
Ethanethiol; see Ethyl mercaptan				
Ethanolamine	141-43-5	3	6	—
Ethion	563-12-2	—	—	—
2-Ethoxyethanol (Cellosolve)	110-80-5	200	740	X
2-Ethoxyethyl acetate (Cellosolve acetate)	111-15-9	100	540	X
Ethyl acetate	141-78-6	400	1400	—
Ethyl acrylate	140-88-5	25	100	X
Ethyl alcohol (Ethanol)	64-17-5	1000	1900	—
Ethylamine	75-04-7	10	18	—
Ethyl amyl ketone (5-Methyl-3-heptanone)	541-85-5	25	130	—
Ethyl benzene	100-41-4	100	435	—
Ethyl bromide	74-96-4	200	890	—
Ethyl butyl ketone (3-Heptanone)	106-35-4	50	230	—
Ethyl chloride	75-00-3	1000	2600	—
Ethyl ether	60-29-7	400	1200	—
Ethyl formate	109-94-4	100	300	—
Ethyl mercaptan	75-08-1	0.5	1	—
Ethyl silicate	78-10-4	100	850	—
Ethylene	74-85-1	E		
Ethylene chlorohydrin	107-07-3	5	16	X
Ethylenediamine	107-15-3	10	25	—
Ethylene dibromide	106-93-4	(C)25	(C)190	X
Ethylene dichloride (1,2-Dichloroethane)	107-06-2	50	200	1

ruction—Continued

n g- on	Proposed Final Rule Limits						Skin Desig-nation
	TWA		STEL [c]		CEILING		
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
—	—	5	—	—	—	—	—
X	0.1	0.5	—	—	—	—	X
—	—	5	—	—	—	—	—
X	—	1	—	—	—	—	X
X	—	0.2	—	—	—	—	X
X	—	1.5	—	—	—	—	X
X	25	90	—	—	—	—	X
—	—	0.2	—	—	—	—	X
—	0.2	1	—	—	—	—	—
—	—	10	—	—	—	—	—
X	100	600	150	900	—	—	X
—	50	235	—	—	—	—	—
—	—	0.5	—	—	—	—	—
—	—	5	—	10	—	—	—
—	—	2	—	—	—	—	—
—	—	0.1	—	—	—	—	X
—	—	10	—	—	—	—	—
—	—	10	—	—	—	—	—
—	10	50	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
X	—	0.1	—	—	—	—	X
X	—	0.1	—	—	—	—	X
X	2	8	—	—	—	—	X
X	—	0.5	—	—	—	—	X
—	3	8	6	15	—	—	—
—	—	0.4	—	—	—	—	X
X	200	740	—	—	—	—	X
X	100	540	—	—	—	—	X
—	400	1400	—	—	—	—	—
X	5	20	25	100	—	—	X
—	1000	1900	—	—	—	—	—
—	10	18	—	—	—	—	—
—	25	130	—	—	—	—	—
—	100	435	125	545	—	—	—
—	200	890	250	1110	—	—	—
—	50	230	—	—	—	—	—
—	1000	2600	—	—	—	—	—
—	400	1200	500	1500	—	—	—
—	100	300	—	—	—	—	—
—	0.5	1	—	—	—	—	—
—	10	85	—	—	—	—	—
X	—	—	—	—	1	3	X
—	10	25	—	—	—	—	—
X	—	—	—	—	25	190	X
1	4	2	8	—	—	—	—

Ethylene glycol	107-21-1	—	—	—
Ethylene glycol dinitrate	628-96-6	(C)0.2	(C)1	X
Ethylene glycol methyl acetate; see Methyl cellosolve acetate				
Ethyleneimine; see 1910.1012	151-56-4	0.5	1	X
Ethylene oxide [f]; see 1910.1047	75-21-8	(1)	(1)	(1)
Ethylidene chloride; see 1,1-Dichloroethane				
Ethylidene norbornene	16219-75-3	—	—	—
N-Ethylmorpholine	100-74-3	20	94	X
Fenamiphos	22224-92-6	—	—	—
Fensulfothion (Dasanit)	115-90-2	—	—	—
Fenthion	55-38-9	—	—	—
Ferbam	14484-64-1			
Total dust		—	15	—
Ferrovanadium dust	12604-58-9	—	1	—
Fibrous Glass				
Total dust			15	—
Respirable fraction		—	5	—
Fluorides (as F)	Varies with compound	—	2.5	—
Fluorine	7782-41-4	0.1	0.2	—
Fluorotrichloromethane (Trichlorofluoromethane)	75-69-4	1000	5600	—
Fonofos	944-22-9	—	—	—
Formaldehyde [f, b]; see 1910.1048; and 56 FR 32302	50-00-0	1	(2 ppm STEL)	—
Formamide	75-12-7	—	—	—
Formic acid	64-18-6	5	9	—
Furfural	98-01-1	5	20	X
Furfuryl alcohol	98-00-0	50	200	—
Gasoline	8006-61-9		A [3]	
Germanium tetrahydride	7782-65-2	—	—	—
Glutaraldehyde	111-30-8	—	—	—
Glycerin (mist)	56-81-5			
Total dust		—	15	—
Respirable fraction		—	5	—
Glycidol	556-52-5	50	150	—
Glycol monoethyl ether; see 2-Ethoxyethanol				
Grain dust (oat, wheat, barley)		—	—	—
Graphite, natural, respirable dust	7782-42-5	(2)	(2)	(2)
Graphite, synthetic		—		
Total dust		—	15	—
Respirable fraction		—	5	—
Guthion; see Azinphos methyl				
Gypsum	13397-24-5			
Total dust		—	15	—
Respirable fraction		—	5	—
Hafnium	7440-58-6	—	0.5	—
Helium	7440-59-7	E		
Heptachlor	76-44-8	—	0.5	M
Heptane (n-Heptane)	142-82-5	500	2000	—
Hexachlorobutadiene	87-68-3	—	—	—
Hexachlorocyclopentadiene	77-47-4	—	—	—
Hexachloroethane	67-72-1	1	10	X
Hexachloronaphthalene	1335-87-1	—	0.2	X
Hexafluoroacetone	684-16-2	—	—	—
n-Hexane	110-54-3	500	1800	—
Hexane isomers	Varies with compound	—	—	—
2-Hexanone (Methyl n-butyl ketone)	591-78-6	100	410	—
Hexone (Methyl isobutyl ketone)	108-10-1	100	410	—
sec-Hexyl acetate	108-84-9	50	300	—
Hexylene glycol	107-41-5	—	—	—
Hydrazine	302-01-2	1	1.3	X
Hydrogen	1333-74-0	E		
Hydrogenated terphenyls	61788-32-7	—	—	—

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TABLE Z.—Constructio

Substance	CAS No.[a]	Transitional Limits[a] ppm[a]	(1970 TLVs) mg/m³[b]	Skin Designation
Hydrogen bromide	10035-10-6	3	10	—
Hydrogen chloride	7647-01-0	(C)5	(C)7	—
Hydrogen cyanide	74-90-8	10	11	X
Hydrogen fluoride (as F)	7664-39-3	3	2	—
Hydrogen peroxide	7722-84-1	1	1.4	—
Hydrogen selenide (as Se)	7783-07-5	0.05	0.2	—
Hydrogen sulfide	7783-06-4	10	15	—
Hydroquinone	123-31-9	—	2	—
2-Hydroxypropyl acrylate	999-61-1	—	—	—
Indene	95-13-6	10	45	—
Indium and compounds (as In)	7440-74-6	—	0.1	—
Iodine	7553-56-2	(C)0.1	(C)1	—
Iodoform	75-47-8	—	—	—
Iron oxide fume	1309-37-1	—	10	—
Iron pentacarbonyl (as Fe)	13463-40-6	—	—	—
Iron salts (soluble) (as Fe)	Varies with compound	—	1	—
Isoamyl acetate	123-92-2	100	525	—
Isoamyl alcohol (primary and secondary)	123-51-3	100	360	—
Isobutyl acetate	110-19-0	150	700	—
Isobutyl alcohol	78-83-1	100	300	—
Isooctyl alcohol	26952-21-6	—	—	—
Isophorone	78-59-1	25	140	—
Isophorone diisocyanate	4098-71-9	—	—	—
2-Isopropoxyethanol	109-59-1	—	—	—
Isopropyl acetate	108-21-4	250	950	—
Isopropyl alcohol	67-63-0	400	980	—
Isopropylamine	75-31-0	5	12	—
N-Isopropylaniline	768-52-5	—	—	—
Isopropyl ether	108-20-3	500	2100	—
Isopropyl glycidyl ether (IGE)	4016-14-2	50	240	—
Kaolin	1332-58-7			
Total dust		—	15	—
Respirable fraction		—	5	—
Ketene	463-51-4	0.5	0.9	—
Lead, inorganic (as Pb)	7439-92-1	—	0.2	—
Limestone	1317-65-3			
Total dust		—	15	—
Respirable fraction		—	5	—
Lindane	58-89-9	—	0.5	X
Lithium hydride	7580-67-8	—	0.025	—
L.P.G. (Liquefied petroleum gas)	68476-85-7	1000	1800	—
Magnesite	546-93-0			
Total dust		—	15	—
Respirable fraction		—	5	—
Magnesium oxide fume	1309-48-4			
Total particulate		15	—	—
Malathion	121-75-5			
Total dust		—	15	X
Maleic anhydride	108-31-6	0.25	1	0.25
Manganese compounds (as Mn)	7439-96-5	—	(C)5	—
Manganese fume (as Mn)	7439-96-5	—	(C)5	—
Manganese cyclopentadienyl tricarbonyl (as Mn)	12079-65-1	—	—	—
Manganese tetroxide (as Mn)	1317-35-7	—	—	—
Marble	1317-65-3			
Total dust		—	15	—
Respirable fraction		—	5	—

truction—Continued

Skin Designation	Proposed Final Rule Limits						Skin Designation
	TWA		STEL [c]		CEILING		
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
—	—	—	—	—	3	10	—
—	—	—	—	—	5	7	—
X	—	—	4.7	5	X		
—	3	—	6	—	—	—	—
—	1	1.4	—	—	—	—	—
—	0.05	0.2	—	—	—	—	—
—	10	14	15	21	—	—	—
—	—	2	—	—	—	—	—
—	0.5	3	—	—	—	—	X
—	10	45	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	—	—	—	0.1	1	—
—	0.6	10	—	—	—	—	—
—	—	10	—	—	—	—	—
—	0.1	0.8	0.2	1.6	—	—	—
—	—	1	—	—	—	—	—
—	100	525	—	—	—	—	—
—	100	360	125	450	—	—	—
—	150	700	—	—	—	—	—
—	50	150	—	—	—	—	—
—	50	270	—	—	—	—	X
—	4	23	—	—	—	—	—
—	0.005	—	0.02	—	—	—	X
—	25	105	—	—	—	—	—
—	250	950	310	1185	—	—	—
—	400	980	500	1225	—	—	—
—	5	12	10	24	—	—	—
—	2	10	—	—	—	—	X
—	500	2100	—	—	—	—	—
—	50	240	75	360	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	0.5	0.9	1.5	3	—	—	—
—	—	0.05	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
X	—	0.5	—	—	—	—	X
—	—	0.025	—	—	—	—	—
—	1000	1800	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	10	—	—	—	—	—	
X	—	10	—	—	—	—	X
0.25	1	—	—	—	—	—	—
—	—	—	—	—	—	5	—
—	—	1	—	3	—	—	—
—	—	0.1	—	—	—	—	X
—	—	1	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—

Mercury (aryl and inorganic)(as Hg)	7439-97-6		0.1	X
Mercury (organo) alkyl compounds (as Hg)	7439-97-6	—	0.01	X
Mercury (vapor) (as Hg)	7439-97-6	—	0.1	X
Mesityl oxide	141-79-7	25	100	—
Methacrylic acid	79-41-4	—	—	—
Methane	74-82-8	E		
Methanethiol; see Methyl mercaptan				
Methomyl (Lannate)	16752-77-5	—	—	—
Methoxychlor	72-43-5			
Total dust		—	15	—
2-Methoxyethanol (Methyl cellosolve)	109-86-4	25	80	X
2-Methoxyethyl acetate (Methyl cellosolve acetate)	110-49-6	25	120	X
4-Methoxyphenol	150-76-5	—	—	—
Methyl acetate	79-20-9	200	610	—
Methyl acetylene (Propyne)	74-99-7	1000	1650	—
Methyl acetylene-propadiene mixture (MAPP)		1000	1800	—
Methyl acrylate	96-33-3	10	35	X
Methylacrylonitrile	126-98-7	—	—	—
Methylal (Dimethoxymethane)	109-87-5	1000	3100	—
Methyl alcohol	67-56-1	200	260	—
Methylamine	74-89-5	10	12	—
Methyl amyl alcohol; see Methyl isobutyl carbinol				
Methyl n-amyl ketone	110-43-0	100	465	—
Methyl bromide	74-83-9	(C)20	(C)80	X
Methyl butyl ketone; see 2-Hexanone				
Methyl cellosolve; see 2-Methoxyethanol				
Methyl cellosolve acetate; see 2-Methoxyethyl acetate				
Methyl chloride	74-87-3	100	210	—
Methyl chloroform (1,1,1-Trichloroethane)	71-55-6	350	1900	—
Methyl 2-cyanoacrylate	137-05-3	—	—	—
Methylcyclohexane	108-87-2	500	2000	—
Methylcyclohexanol	25639-42-3	100	470	—
o-Methylcyclohexanone	583-60-8	100	460	X
Methylcyclopentadienyl manganese tricarbonyl (as Mn)	12108-13-3	—	—	—
Methyl demeton	8022-00-2	—	—	—
4,4'-Methylene bis (2-chloroaniline) (MBOCA)	101-14-4	—	—	—
Methylene bis (4-cyclohexylisocyanate)	5124-30-1	—	—	—
Methylene chloride [R]; see 56 FR 57036	75-09-2	500	1740	—
Methyl ethyl ketone (MEK); see 2-Butanone				
Methyl ethyl ketone peroxide (MEKP)	1338-23-4	—	—	—
Methyl formate	107-31-3	100	250	—
Methyl hydrazine (Monomethyl hydrazine)	60-34-4	(C)0.2	(C)0.35	X
Methyl iodide	74-88-4	5	28	Y
Methyl isoamyl ketone	110-12-3	100	475	—
Methyl isobutyl carbinol	108-11-2	25	100	X
Methyl isobutyl ketone; see Hexone				
Methyl isocyanate	624-83-9	0.02	0.05	X
Methyl isopropyl ketone	563-80-4	—	—	—
Methyl mercaptan	74-93-1	0.5	1	—
Methyl methacrylate	80-62-6	100	410	100
Methyl parathion	298-00-0	—	—	—
Methyl propyl ketone; see 2-Pentanone				
Methyl silicate	681-84-5	5	30	—
alpha-Methyl styrene	98-83-9	(C)100	(C)480	—
Methylene bisphenyl isocyanate (MDI)	101-68-8	(C)0.02	(C)0.2	—
Metribuzin	21087-64-9	—	—	—
Mica; see Silicates				
Mineral wool				
Total dust		—	15	—
Respirable dust		—	5	—
Molybdenum (as Mo)	7439-98-7			
Soluble compounds		—	5	—
Insoluble compounds				
Total dust		—	15	—

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TABLE Z.—Constructi

Substance	CAS No.[c]	Transitional Limits[a] ppm[a]	(1970 TLVs) mg/m³[b]	Skin Designation
Monocrotophos (Azodrin)	6923-22-4	—	—	—
Monomethyl aniline	100-61-8	2	9	X
Monomethyl hydrazine; see Methyl hydrazine				
Morpholine	110-91-8	20	70	X
Naphtha (Coal tar)	8030-30-6	100	400	—
Naphthalene	91-20-3	10	50	—
alpha-Naphthylamine; see 1910.1004	134-32-7			
beta-Naphthylamine; see 1910.1009	91-59-8		A[1]	
Neon	7440-01-9	E		
Nickel carbonyl (as Ni)	13463-39-3	0.001	0.007	—
Nickel, metal and insoluble compounds (as Ni)	7440-02-0	—	1	—
Nickel, soluble compounds (as Ni)	7440-02-0	—	1	—
Nicotine	54-11-5	—	0.5	X
Nitric acid	7697-37-2	2	5	—
Nitric oxide	10102-43-9	25	30	—
p-Nitroaniline	100-01-6	1	6	X
Nitrobenzene	98-95-3	1	5	X
p-Nitrochlorobenzene	100-00-5	—	1	X
4-Nitrodiphenyl; see 1910.1003	92-93-3			
Nitroethane	79-24-3	100	310	—
Nitrogen	7727-37-9	E		
Nitrogen dioxide	10102-44-0	(C)5	(C)9	—
Nitrogen trifluoride	7783-54-2	10	29	—
Nitroglycerin	55-63-0	(C)0.2	(C)2	X
Nitromethane	75-52-5	100	250	—
1-Nitropropane	108-03-2	25	90	—
2-Nitropropane	79-46-9	25	90	—
N-Nitrosodimethylamine; see 1910.1016	62-79-9		A[1]	
Nitrotoluene (all isomers)		5	30	X
o-isomer	88-72-2;			
m-isomer	99-08-1;			
p-isomer	99-99-0			
Nitrotrichloromethane; see Chloropicrin				
Nitrous oxide	10024-97-2	E		
Nonane	111-84-2	—	—	—
Octachloronaphthalene	2234-13-1	—	0.1	X
Octane	111-65-9	400	1900	—
Oil mist, mineral	8012-95-1	—	5	—
Osmium tetroxide (as Os)	20816-12-0	—	0.002	—
Oxalic acid	144-62-7	—	1	—
Oxygen difluoride	7783-41-7	0.05	0.1	—
Ozone	10028-15-6	0.1	0.2	—
Paraffin wax fume	8002-74-2	—	—	—
Paraquat, respirable dust	4685-14-7; 1910-42-5; 2074-50-2	—	0.5	X
Parathion	56-38-2	—	0.1	X
Particulates not otherwise regulated				
Total dust		—	15	—
Respirable fraction		—	5	—
PCB; see Chlorodiphenyl (42% and 54% chlorine)				
Pentaborane	19624-22-7	0.005	0.01	—
Pentachloronaphthalene	1321-64-8	—	0.5	X
Pentachlorophenol	87-86-5	—	0.5	X
Pentaerythritol	115-77-5			
Total dust		—	15	—
Respirable fraction		—	5	—

tion—Continued

	Proposed Final Rule Limits						
	TWA		STEL [c]		CEILING		Skin Designation
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
	—	0.25	—	—	—	—	X
X	0.5	2	—	—	—	—	—
X	20	70	30	105	—	—	—
	100	400	—	—	—	—	—
—	10	50	15	75	—	—	—
—	0.001	0.007	—	—	—	—	—
—	—	1	—	—	—	—	—
—	—	0.1	—	—	—	—	X
X	—	0.5	—	—	—	—	—
—	2	5	4	10	—	—	—
—	25	20	—	—	—	—	—
X	—	3	—	—	—	—	—
X	1	5	—	—	—	—	—
—	—	1	—	—	—	—	—
—	100	310	—	—	—	—	—
—	—	—	1	1.8	—	—	—
—	10	29	—	0.1	—	—	—
X	—	—	—	—	—	—	—
—	100	250	—	—	—	—	—
—	25	90	—	—	—	—	—
—	10	35	—	—	—	—	X
X	2	11	1	—	—	—	—
—	200	1060	—	0.3	—	—	—
X	—	0.1	375	1800	—	—	—
—	300	1450	—	—	—	—	—
—	—	5	—	0.006	—	—	—
—	0.0002	0.002	0.0006	2	—	—	—
—	—	1	—	—	0.05	0.1	—
—	0.1	0.2	0.3	0.6	—	—	—
—	—	2	—	—	—	—	—
X	—	0.1	—	—	—	—	—
—	—	0.1	—	—	—	—	X
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	0.005	0.01	0.015	0.03	—	—	X
X	—	0.5	—	—	—	—	—
X	—	0.5	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—

Pentane	109-66-0	500	1500	—	—	—
2-Pentanone (Methyl propyl ketone)	107-87-9	200	700	—	—	—
Perchloroethylene (Tetrachloroethylene)	127-18-4	100	670	—	—	—
Perchloromethyl mercaptan	594-42-3	0.1	0.8	—	—	—
Perchloryl fluoride	7616-94-6	3	13.5	—	—	—
Perlite	93763-70-3					
Total dust		—	15	—	—	—
Respirable fraction		—	5	—	—	—
Petroleum distillates (Naphtha)(Rubber Solvent)			A [8]			
Phenol	108-95-2	5	19	—	—	X
Phenothiazine	92-84-2	—	—	—	—	—
p-Phenylene diamine	106-50-3	—	0.1	—	—	X
Phenyl ether, vapor	101-84-8	1	7	—	—	—
Phenyl ether-biphenyl mixture, vapor		1	7	—	—	—
Phenylethylene; see Styrene						
Phenyl glycidyl ether (PGE)	122-60-1	10	60	—	—	—
Phenylhydrazine	100-63-0	5	22	—	—	X
Phenyl mercaptan	108-98-5	—	—	—	—	—
Phenylphosphine	638-21-1	—	—	—	—	—
Phorate	298-02-2	—	—	—	—	—
Phosdrin (Mevinphos)	7786-34-7	—	0.1	—	—	X
Phosgene (Carbonyl chloride)	75-44-5	0.1	0.4	—	—	—
Phosphine	7803-51-2	0.3	0.4	—	—	—
Phosphoric acid	7664-38-2	—	1	—	—	—
Phosphorus (yellow)	7723-14-0	—	0.1	—	—	—
Phosphorus oxychloride	10025-87-3	—	—	—	—	—
Phosphorus pentachloride	10026-13-8	—	1	—	—	—
Phosphorus pentasulfide	1314-80-3	—	1	—	—	—
Phosphorus trichloride	7719-12-2	0.5	2	—	—	—
Phthalic anhydride	85-44-9	2	12	—	—	—
m-Phthalodinitrile	626-17-5	—	—	—	—	—
Picloram	1918-02-1					
Total dust		—	15	—	—	—
Respirable fraction		—	5	—	—	—
Picric acid	88-89-1	—	0.1	—	—	X
Piperazine dihydrochloride	142-64-3	—	—	—	—	—
Pindone (2-Pivalyl-1,3-indandione)	83-26-1	—	0.1	—	—	—
Plaster of Paris	26499-65-0					
Total dust		—	15	—	—	—
Respirable fraction		—	5	—	—	—
Platinum (as Pt)	7440-06-4					
Metal		—	—	—	—	—
Soluble salts		—	0.002	—	—	—
Polytetrafluoroethylene decomposition products			A [3]			
Portland cement	65997-15-1					
Total dust		15	—	—	—	10
Respirable fraction		5	—	—	—	—
Potassium hydroxide	1310-58-3	—	—	—	—	—
Propargyl alcohol	107-19-7	1	—	—	—	X
beta-Propiolactone; see 1910.1013	57-57-8		A [1]			
Propionic acid	79-09-4	—	—	—	—	—
Propoxur (Baygon)	114-26-1	—	—	—	—	—
n-Propyl acetate	109-60-4	200	840	—	—	—
n-Propyl alcohol	71-23-8	200	500	—	—	—
n-Propyl nitrate	627-13-4	25	110	—	—	—
Propylene dichloride	78-87-5	75	350	—	—	—
Propylene glycol dinitrate	6423-43-4	—	—	—	—	—
Propylene glycol monomethyl ether	107-98-2	—	—	—	—	—
Propylene imine	75-55-8	2	5	—	—	X
Propylene oxide	75-56-9	100	240	—	—	—
Propyne; see Methyl acetylene						
Pyrethrum	8003-34-7	—	5	—	—	—
Pyridine	110-86-1	5	15	—	—	—
Quinone	106-51-4	0.1	0.4	—	—	—
RDX; see Cyclonite						
Resorcinol	108-46-3	—	—	—	—	—

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TABLE Z.—Constructi

Substance	CAS No.[4]	Transitional Limits[a] ppm[a]	(1970 TLVs) mg/m³[b]	Skin Designation
Rhodium (as Rh), metal fume and insoluble compounds	7440-16-6	—	0.1	—
Rhodium (as Rh), soluble compounds	7440-16-6	—	0.001	—
Ronnel	299-84-3	—	10	—
Rosin core solder pyrolysis products, as formaldehyde		—	—	—
Rotenone	83-79-4	—	5	—
Rouge		—		
Total dust		—	15	—
Respirable fraction		—	5	—
Selenium compounds (as Se)	7782-49-2	—	0.2	—
Selenium hexafluoride (as Se)	7783-79-1	0.05	0.4	—
Silica, amorphous, precipitated and gel	112926-00-8	(2)	(2)	(2)
Silica, amorphous, diatomaceous earth, containing less than 1% crystalline silica	61790-53-2	(2)	(2)	(2)
Silica, crystalline cristobalite, respirable dust	14464-46-1	(2)	(2)	(2)
Silica, crystalline quartz, respirable dust	14808-60-7	(2)	(2)	(2)
Silica, crystalline tripoli (as quartz), respirable dust	1317-95-9	(2)	(2)	(2)
Silica, crystalline tridymite, respirable dust	15468-32-3	(2)	(2)	(2)
Silica, fused, respirable dust	60676-86-0	(2)	(2)	(2)
Silicates (less than 1% crystalline silica)				
Mica (respirable dust)	12001-26-2	(2)	(2)	(2)
Soapstone, total dust	—	(2)	(2)	(2)
Soapstone, respirable dust	—	(2)	(2)	(2)
Talc (containing asbestos)	—	(3)	(3)	(3)
Talc (containing no asbestos), respirable dust	14807-96-6	(2)	(2)	(2)
Tremolite		(1)	(1)	(1)
Silicon	7440-21-3			
Total dust		—	15	—
Respirable fraction		—	5	—
Silicon carbide	409-21-2			
Total dust		—	15	—
Respirable fraction		—	5	—
Silicon tetrahydride	7803-62-5	—	—	—
Silver, metal and soluble compounds (as Ag)	7440-22-4	—	0.01	—
Soapstone; see Silicates				
Sodium azide	26628-22-8			
(as HN_3)		—	—	—
(as NaN_3)		—	—	—
Sodium bisulfite	7631-90-5	—	—	—
Sodium fluoroacetate	62-74-8	—	0.05	X
Sodium hydroxide	1310-73-2	—	2	—
Sodium metabisulfite	7681-57-4	—	—	—
Starch	9005-25-8			
Total dust		—	15	—
Respirable fraction		—	5	—
Stibine	7803-52-3	0.1	0.5	—
Stoddard solvent	8052-41-3	500	1150	—
Strychnine	57-24-9	—	0.15	—
Styrene	100-42-5	100	420	50
Subtilisins (Proteolytic enzymes)	9014-01-1	—	—	—
Sucrose	57-50-1			
Total dust		—	15	—

ruction—Continued

n g- on	Proposed Final Rule Limits						Skin Desig-nation
	TWA		STEL [c]		CEILING		
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
—	—	0.1	—	—	—	—	—
—	—	0.001	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	0.1	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	0.2	—	—	—	—	—
—	0.05	0.4	—	—	—	—	—
[3]	—	6	—	—	—	—	—
[3]	—	6	—	—	—	—	—
[3]	—	0.05	—	—	—	—	—
[3]	—	0.1	—	—	—	—	—
[3]	—	0.1	—	—	—	—	—
[3]	—	0.05	—	—	—	—	—
[3]	—	0.1	—	—	—	—	—
[3]	—	3	—	—	—	—	—
[3]	—	6	—	—	—	—	—
[3]	—	3	—	—	—	—	—
[3]	—	0.2 f/cc	—	1 f/cc (30 min.)	—	—	—
[2]	—	2	—	—	—	—	—
[1]	[4]	[4]	[4]	[4]	[4]	[4]	[4]
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	5	7	—	—	—	—	—
—	—	0.01	—	—	—	—	—
—	—	—	—	—	0.1	—	X
—	—	—	—	—	—	0.3	X
—	—	5	—	—	—	—	—
X	—	0.05	—	0.15	—	—	X
—	—	—	—	—	—	2	—
—	—	5	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	0.1	0.5	—	—	—	—	—
—	100	525	—	—	—	—	—
—	—	0.15	—	—	—	—	—
50	215	100	425	—	—	—	—
—	—	—	—	0.00006 [e] (60 min.)	—	—	—
—	—	15	—	—	—	—	—

Respirable fraction		—	5	—
Sulfur dioxide	7446-09-5	5	13	—
Sulfur hexafluoride	2551-62-4	1000	6000	—
Sulfuric acid	7664-93-9	—	1	—
Sulfur monochloride	10025-67-9	1	6	—
Sulfur pentafluoride	5714-22-7	0.025	0.25	—
Sulfur tetrafluoride	7783-60-0	—	—	—
Sulfuryl fluoride	2699-79-8	5	20	—
Sulprofos	35400-43-2	—	—	—
Systox, see Demeton				
2,4,5-T (2,4,5-trichlorophenoxyacetic acid)	93-76-5	—	10	—
Talc; see Silicates				
Tantalum, metal and oxide dust	7440-25-7	—	5	—
TEDP (Sulfotep)	3689-24-5	—	0.2	X
Teflon decomposition products		A2		
Tellurium and compounds (as Te)	13494-80-9	—	0.1	—
Tellurium hexafluoride (as Te)	7783-80-4	0.02	0.2	—
Temephos	3383-96-8			
Total dust		—	15	—
Respirable fraction		—	5	—
TEPP (Tetraethyl pyrophosphate)	107-49-3	—	0.05	X
Terphenyls	26140-60-3	(C)1	(C)9	—
1,1,1,2-Tetrachloro-2,2-difluoroethane	76-11-9	500	4170	—
1,1,2,2-Tetrachloro-1,2-difluoroethane	76-12-0	500	4170	—
1,1,2,2-Tetrachloroethane	79-34-5	5	35	X
Tetrachloroethylene; see Perchloroethylene				
Tetrachloromethane; see Carbon tetrachloride				
Tetrachloronaphthalene	1335-88-2	—	2	X
Tetraethyl lead (as Pb)	78-00-2	—	0.1	X
Tetrahydrofuran	109-99-9	200	590	—
Tetramethyl lead, (as Pb)	75-74-1	—	0.15	X
Tetramethyl succinonitrile	3333-52-6	0.5	3	X
Tetranitromethane	509-14-8	1	6	—
Tetrasodium pyrophosphate	7722-88-5	—	—	—
Tetryl (2,4,6-Trinitrophenylmethylnitramine)	479-45-8	—	1.5	X
Thallium, soluble compounds (as Tl)	7440-28-0	—	0.1	X
4,4'-Thiobis (6-tert, Butyl-m-cresol)	96-69-5			
Total dust		—	15	—
Respirable fraction		—	5	—
Thioglycolic acid	68-11-1	—	—	—
Thionyl chloride	7719-09-7	—	—	—
Thiram	137-26-8	—	5	—
Tin, inorganic compounds (except oxides) (as Sn)	7440-31-5	—	2	—
Tin, organic compounds (as Sn)	7440-31-5	—	0.1	—
Tin oxide (as Sn)	21651-19-4	—	—	—
Total dust		—	15	—
Respirable fraction		—	5	—
Titanium dioxide	13463-67-7			
Total dust		—	15	—
Toluene	108-88-3	200	750	100
Toluene-2,4-diisocyanate (TDI)	584-84-9	(C)0.02	(C)0.14	—
m-Toluidine	108-44-1	—	—	—
o-Toluidine	95-53-4	5	22	X
p-Toluidine	106-49-0	—	—	—
Toxaphene; see Chlorinated camphene				
Tremolite; see Silicates				
Tributyl phosphate	126-73-8	—	5	—
Trichloroacetic acid	76-03-9	—	—	—
1,2,4-Trichlorobenzene	120-82-1	—	—	—
1,1,1-Trichloroethane; see Methyl chloroform				
1,1,2-Trichloroethane	79-00-5	10	45	X
Trichloroethylene	79-01-6	100	535	—
Trichloromethane; see Chloroform				
Trichloronaphthalene	1321-65-9	—	5	X
1,2,3-Trichloropropane	96-18-4	50	300	—
1,1,2-Trichloro-1,2,2-trifluoroethane	76-13-1	1000	7600	—

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Substance	CAS No.[a]	Transitional Limits[a] ppm[a]	(1970 TLVs) mg/m³[b]	Skin Designation
Triethylamine	121-44-8	25	100	—
Trifluorobromomethane	75-63-8	1000	6100	—
Trimellitic anhydride	552-30-7	—	—	—
Trimethylamine	75-50-3	—	—	—
Trimethyl benzene	25551-13-7	25	120	—
Trimethyl phosphite	121-45-9	—	—	—
2,4,6-Trinitrophenyl; see Picric acid				
2,4,6-Trinitrophenylmethylnitramine; see Tetryl				
2,4,6-Trinitrotoluene (TNT)	118-96-7	—	1.5	X
Trior thocresyl phosphate	78-30-8	—	0.1	—
Triphenyl amine	603-34-9	—	—	—
Triphenyl phosphate	115-86-6	—	3	—
Tungsten (as W)	7440-33-7			
Insoluble compounds		—	5	—
Soluble compounds		—	1	—
Turpentine	8006-64-2	100	560	—
Uranium (as U)	7440-61-1			
Soluble compounds		—	0.2	—
Insoluble compounds		—	0.2	—
n-Valeraldehyde	110-62-3	—	—	—
Vanadium	1314-62-1			
Respirable dust (as V_2O_5)		—	(C)0.5	—
Fume (as V_2O_5)		—	(C)0.1	—
Vegetable oil mist	—			
Total dust		—	15	—
Respirable fraction		—	5	—
Vinyl acetate	108-05-4	—	—	—
Vinyl benzene; see Styrene				
Vinyl bromide	593-60-2	—	—	—
Vinyl chloride[c]; see 1910.1017	75-01-4	(1)	(1)	(1)
Vinyl cyanide; see Acrylonitrile				
Vinyl cyclohexene dioxide	106-87-6	—	—	—
Vinylidene chloride (1,1Dichloroethylene)	75-35-4	—	—	—
Vinyl toluene	25013-15-4	100	480	—
VM & P Naphtha	8032-32-4	—	—	—
Warfarin	81-81-2	—	0.1	—
Welding fumes (total particulate)**		—	—	—
Wood dust, all soft and hard woods, except Western red cedar		—	—	—
Wood dust, Western red cedar		—	—	—
Xylenes (o-, m-, p-isomers)	1330-20-7	100	435	—
m-Xylene alpha, alpha'-diamine	1477-55-0	—	—	—
Xylidine	1300-73-8	5	25	X
Yttrium	7440-65-5	—	1	—
Zinc chloride fume	7646-85-7	—	1	—
Zinc chromate (as CrO_3)	13530-65-9	—	—	—
Zinc oxide fume	1314-13-2	—	5	—
Zinc oxide	1314-13-2			
Total dust		—	15	—
Respirable fraction		—	5	—
Zinc stearate	557-05-1			
Total dust		—	15	—
Respirable fraction		—	5	—
Zirconium compounds (as Zr)	7440-67-7	—	5	—

Footnotes:
[1] Same as Final Rule Limits.

struction—Continued

kin sig- ion	Proposed Final Rule Limits						Skin Desig- nation
	TWA		STEL [c]		CEILING		
	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
—	10	40	15	60	—	—	—
—	1000	6100	—	—	—	—	—
—	0.005	0.04	—	—	—	—	—
—	10	24	15	36	—	—	—
—	25	125	—	—	—	—	—
—	2	10	—	—	—	—	—
X	—	0.5	—	—	—	—	X
—	—	0.1	—	—	—	—	X
—	—	5	—	—	—	—	—
—	—	3	—	—	—	—	—
—	—	5	—	10	—	—	—
—	—	1	—	3	—	—	—
—	100	560	—	—	—	—	—
—	—	0.05	—	—	—	—	—
—	—	0.2	—	0.6	—	—	—
—	50	175	—	—	—	—	—
—	—	0.05	—	—	—	—	—
—	—	0.05	—	—	—	—	—
—	—	15	—	—	—	—	—
—	—	5	—	—	—	—	—
—	10	30	20	60	—	—	—
—	5	20	—	—	—	—	—
[1]	1	—	5	—	—	—	—
—	10	60	—	—	—	—	X
—	1	4	—	—	—	—	—
—	100	480	—	—	—	—	—
—	300	1350	400	1800	—	—	—
—	—	0.1	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	5	—	10	—	—	—
—	—	2.5	—	—	—	—	—
—	100	435	150	655	—	—	—
X	—	—	—	—	—	0.1	X
—	2	10	—	—	—	—	X
—	—	1	—	—	—	—	—
—	—	1	—	2	—	—	—
—	—	—	—	—	—	0.1	—
—	—	5	—	10	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	10	—	—	—	—	—
—	—	5	—	—	—	—	—
—	—	5	—	10	—	—	—

[5] See Mineral Dusts Table.
[6] Use Asbestos Limit 1910.1001.
[7] See 1910.1001 for asbestiform; for non-asbestiform see 1910.1101.
* The transitional PELs are 8-hour TWAs unless otherwise noted; a (C) designation denotes a ceiling limit.
** As determined from breathing-zone air samples.
[a] Parts of vapor or gas per million parts of contaminated air by volume at 25°C and 760 torr.
[b] Milligrams of substance per cubic meter of air. When entry is in this column only, the value is exact; when listed wi
[c] Duration is for 15 minutes, unless otherwise noted.
[d] The CAS number is for information only. Enforcement is based on the substance name. For an entry covering mor
CAS numbers for the individual compounds.
[e] Compliance with the subtilisins PEL is assessed by sampling with a high volume sampler (600-800 liters per minute
[f] For those substances for which a full standard exists, exposure limits are provided in the table for information
[g] For sectors excluded from 1910.1028 the limit is 10 ppm TWA and see 1910.1000, Table Z-2 for ceiling limits.
[h] Where OSHA has published a proposal for a substance separately from this rulemaking but has not issued a fi
columns. The proposed new limit is published in the Final Rule Limits columns.
The 1970 TLV uses letter designations instead of a numerical value as follows:
A [1] Because of the high incidence of cancer, either in man or in animals, no exposure or contact by any route, respi
A [2] Polytetrafluoroethylene decomposition products. Because these products decompose in part by hydrolysis in a
exposure. No TLV is recommended pending determination of the toxicity of the products, but air concentrations should
A [3] Gasoline and/or Petroleum Distillates. The composition of these materials varies greatly and thus a single TLV
and additives should be determined to arrive at the appropriate TLV.
E Simple asphyxiants.

ted with a ppm entry, it is approximate.

g more than one metal compound, measured as the metal, the CAS number for the metal is given--not

minute) for at least 60 minutes.
mational purposes, and the binding requirements are as set forth in the single substance standard.
ts.
d a final rule, the proposal is referenced and the existing limit is published in the Transitional Limits

, respiratory, oral, or skin, should be permitted.
is in alkaline solution, they can be quantitatively determined in air as fluoride to provide an index of
should be minimal.
e TLV for all types of these materials is no longer applicable. The content of benzene, other aromatics

V. OSHA proposes to amend 29 CFR part 1928 as follows:

PART 1928—AGRICULTURE

1. It is proposed to revise the authority citation for part 1928 to read as follows:

Authority: Secs. 6, 8 Occupational Safety and Health Act, 29 U.S.C. 655, 657; Secretary of Labor's Order, No. 12–7 (36 FR 8754), 8–76 (41 FR 25059), 9–83 (48 FR 35736) or 1–90 (55 FR 9033) as applicable; and 29 CFR Part 1911.

Subparts J–Y—[Reserved]

2. It is proposed to reserve Subparts J–Y and to add a new Subpart Z consisting of § 1928.1000 to read as follows:

Subpart Z—Toxic and Hazardous Substances

§ 1928.1000 Air contaminants.

(a) *Exposure limits.* An employee's exposure to any substance listed in Table Z, Agriculture shall be limited in accordance with the requirements of the following paragraphs of this section:

(1) Final Rule Limit Columns. An employee's exposure to any substance listed in Table Z, Agriculture shall not exceed the Time Weighted Average (TWA), Short Term Exposure Limit (STEL) and Ceiling Limit specified for that substance in Table Z, Agriculture under the Final Rule Limits columns.

(2) Skin Designation. To prevent or reduce skin absorption, an employee's skin exposure to substances listed in Table Z, Agriculture with an "X" in the Skin Designation column following the substance name shall be prevented or reduced to the extent necessary in the circumstances through the use of gloves, coveralls, goggles, or other appropriate personal protective equipment, engineering controls or work practices.

(3) Definitions. The following definitions are applicable to the Final Rule Limits columns of Table Z, Agriculture:

(i) *Time weighted average (TWA)* is the employee's average airborne exposure in any 8-hour work shift of a 40-hour work week which shall not be exceeded.

(ii) *Short term exposure limit (STEL)* is the employee's 15-minute time weighted average exposure which shall not be exceeded at any time during a work day unless another time limit is specified in a parenthetical notation below the limit. If another time period is specified, the time weighted average exposure over that time period shall not be exceeded at any time during the working day.

(iii) Ceiling is the employee's exposure which shall not be exceeded during any part of the work day. If instantaneous monitoring is not feasible, then the ceiling shall be assessed as a 15-minute time weighted average exposure which shall not be exceeded at any time over a working day.

(b) *Computation formulae.* The computation formula which shall apply to employee exposure to more than one substance for which 8-hour time weighted averages are listed in subpart Z of 29 CFR Part 1918 in order to determine whether an employee is exposed over the regulatory limit is as follows:

(1)(i) The cumulative exposure for an 8-hour work shift shall be computed as follows:

$$E=(C_aT_a+C_bT_b+\ldots C_nT_n)\div 8$$

Where:

E is the equivalent exposure for the working shift.

C is the concentration during any period of time T where the concentration remains constant.

T is the duration in hours of the exposure at the concentration C.

The value of E shall not exceed the 8-hour time weighted average specified in Subpart Z or 29 CFR Part 1918 for the material involved.

(ii) To illustrate the formula prescribed in paragraph (d)(1)(i) of this section, assume that Substance A has an 8-hour time weighted average limit of 100 ppm noted in Table Z. Assume that an employee is subject to the following exposure:

Two hours exposure at 150 ppm
Two hours exposure at 75 ppm
Four hours exposure at 50 ppm

Substituting this information in the formula, we have:

$$(2\times 150+2\times 75+4\times 50)\div 8=81.25 \text{ ppm}$$

Since 81.25 ppm is less than 100 ppm, the 8-hour time weighted average limit, the exposure is acceptable.

(2)(i) in case of a mixture of air contaminants an employer shall compute the equivalent exposure as follows:

$$Em=(C_1\div L_1+C_2\div L_2)+\ldots(C_n\div L_n)$$

Where:

E_m is the equivalent exposure for the mixture.

C is the concentration of a particular contaminant.

L is the exposure limit for that substance specified in subpart Z of 29 CFR part 1918.

The value of E_m shall not exceed unity (1).

(ii) To illustrate the formula prescribed in paragraph (d)(2)(i) of this section, consider the following exposures:

Substance	Actual concentration of 8 hour exposure (ppm)	8 hr. TWA PEL (ppm)
B	500	1000
C	45	200
D	40	200

Substituting in the formula we have:

$$E_m=500\div 1{,}000+45\div 200+40\div 200$$
$$E_m=0.500+0.225+0.200$$
$$E_m=0.925$$

Since Em is less than unity (1), the exposure combination is within acceptable limits.

(c) *Methods of compliance.* To achieve compliance with paragraph (a) of this section administrative, work practice or engineering controls must first be implemented whenever feasible. When such controls are not feasible, respirators or other protective measures shall be used to keep the exposure of employees to air contaminants within the limits prescribed in this section. Whenever respirators are used, their use shall comply with 29 CFR § 1910.134.

(d) *Effective and Start-up dates*—(1) *Effective date.* The effective date for the permissible exposure limits specified in the Final Rule Limits columns of Table Z, Agriculture is [90 days after date of publication in the **Federal Register**].

(2) Start-up dates (i) The permissible exposure limits specified in the Final Rule Limits columns of Table Z, Agriculture shall be achieved by any reasonable combination of engineering controls, administrative controls, work practices and personal protective equipment effective [1 year after date of publication in the **Federal Register**].

(ii) The permissible exposure limits specified in the Final Rule Limits columns of Table Z, Agriculture shall be achieved by the method of compliance specified in paragraph (b) of this section effective [4 years after date of publication in the **Federal Register**].

(iii) The skin designations in the Final Rule Limits columns shall be complied with commencing [1 year after date of publication in the **Federal Register**].

TABLE Z.—Agriculture

Substance	CAS No.[a]	Proposed Final Rule Limits						Skin Designation
		TWA		STEL		CEILING		
		ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	
Acetaldehyde	75-07-0	100	180	150	270	—	—	—
Acetic acid	64-19-7	10	25	—	—	—	—	—
Acetic anhydride	108-24-7	—	—	—	—	5	20	—
Acetone	67-64-1	750	1800	1000	2400	—	—	—
Acetonitrile	75-05-8	40	70	60	105	—	—	—
Acetylene tetrabromide	79-27-6	1	14	—	—	—	—	—
Acetylsalicylic acid (Aspirin)	50-78-2	—	5	—	—	—	—	—
Acrolein	107-02-8	0.1	0.25	0.3	0.8	—	—	—
Acrylamide	79-06-1	—	0.03	—	—	—	—	X
Acrylic acid	79-10-7	10	30	—	—	—	—	X
Acrylonitrile [c]; see 1910.1045	107-13-1	2	—	10	—	—	—	X
Aldrin	309-00-2	—	0.25	—	—	—	—	X
Allyl alcohol	107-18-6	2	5	4	10	—	—	X
Allyl chloride	107-05-1	1	3	2	6	—	—	—
Allyl glycidyl ether (AGE)	106-92-3	5	22	10	44	—	—	—
Allyl propyl disulfide	2179-59-1	2	12	3	18	—	—	—
alpha-Alumina	1344-28-1							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Aluminum (as Al)	7429-90-5							
Metal								
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Pyro powders		—	5	—	—	—	—	—
Welding fumes[d][e]		—	5	—	—	—	—	—
Soluble salts		—	2	—	—	—	—	—
Alkyls		—	2	—	—	—	—	—
2-Aminoethanol; see Ethanolamine								
2-Aminopyridine	504-29-0	0.5	2	—	—	—	—	—
Amitrole	61-82-5	—	0.2	—	—	—	—	—
Ammonia	7664-41-7	—	—	35	27	—	—	—
Ammonium chloride fume	12125-02-9	—	10	—	20	—	—	—
Ammonium sulfamate	7773-06-0							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
n-Amyl acetate	628-63-7	100	525	—	—	—	—	—
sec-Amyl acetate	626-38-0	125	650	—	—	—	—	—
Aniline and homologs	62-53-3	2	8	—	—	—	—	X
Anisidine (o-, p-isomers)	29191-52-4	—	0.5	—	—	—	—	X
Antimony and compounds (as Sb)	7440-36-0	—	0.5	—	—	—	—	—
ANTU (alpha Naphthylthiourea)	86-88-4	—	0.3	—	—	—	—	—
Arsenic, inorganic compounds (as As) [c]; see 1910.1018	7440-38-2	—	0.01	—	—	—	—	—
Arsenic, organic compounds (as As)	7440-38-2	—	0.5	—	—	—	—	—
Arsine	7784-42-1	0.05	0.2	—	—	—	—	—
Asbestos [c]; see 1910.1001 and 1910.1101	Varies		0.2 f/cc		1 f/cc (30 min.)			
Asphalt (petroleum) fumes	8052-42-4	—	5	—	—	—	—	—
Atrazine	1912-24-9	—	5	—	—	—	—	—
Azinphos-methyl	86-50-0	—	0.2	—	—	—	—	X
Barium, soluble compounds (as Ba)	7440-39-3	—	0.5	—	—	—	—	—
Barium sulfate	7727-43-7							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Benomyl	17804-35-2							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Benzene [c][e]; see 1910.1028	71-43-22	1	—	5	—	—	—	—
p-Benzoquinone; see Quinone								
Benzo(a)pyrene; see Coal tar pitch volatiles								
Benzoyl peroxide	94-36-0	—	5	—	—	—	—	—
Benzyl chloride	100-44-7	1	5	—	—	—	—	—
Beryllium and beryllium compounds (as Be)	7440-41-7	0.002	0.025 (30 min.)					
Biphenyl; see Diphenyl								
Bismuth telluride, Undoped	1304-82-1							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Bismuth telluride, Se-doped	1304-82-1	—	5	—	—	—	—	—
Borates, tetra, sodium salts								
Anhydrous	1330-43-4	—	10	—	—	—	—	—
Decahydrate	1303-96-4	—	10	—	—	—	—	—
Pentahydrate	12179-04-3	—	10	—	—	—	—	—
Boron oxide	1303-86-2							
Total dust		—	10	—	—	—	—	—

TABLE Z.—Agriculture—Continued

Substance	CAS No.[a]	Proposed Final Rule Limits						Skin Designation
		TWA		STEL		CEILING		
		ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	
Boron tribromide	10294-33-4	—	—	—	—	1	10	—
Boron trifluoride	7637-07-2	—	—	—	—	1	3	—
Bromacil	314-40-9	1	10	—	—	—	—	—
Bromine	7726-95-6	0.1	0.7	0.3	2	—	—	—
Bromine pentafluoride	7789-30-2	0.1	0.7	—	—	—	—	—
Bromoform	75-25-2	0.5	5	—	—	—	—	X
Butadiene (1,3-Butadiene)[h]; see 55 FR 32736	106-99-0	2	—	10	—	—	—	—
Butane	106-97-8	800	1900	—	—	—	—	—
Butanethiol; see Butyl mercaptan								
2-Butanone (Methyl ethyl ketone)	78-93-3	200	590	300	885	—	—	—
2-Butoxyethanol	111-76-2	25	120	—	—	—	—	X
n-Butyl acetate	123-86-4	150	710	200	950	—	—	—
sec-Butyl acetate	105-46-4	200	950	—	—	—	—	—
tert-Butyl acetate	540-88-5	200	950	—	—	—	—	—
Butyl acrylate	141-32-2	10	55	—	—	—	—	—
n-Butyl alcohol	71-36-3	—	—	—	—	50	150	X
sec-Butyl alcohol	78-92-2	100	305	—	—	—	—	—
tert-Butyl alcohol	75-65-0	100	300	150	450	—	—	—
Butylamine	109-73-9	—	—	—	—	5	15	X
tert-Butyl chromate (as CrO_3)	1189-85-1	—	—	—	—	—	0.1	X
n-Butyl glycidyl ether (BGE)	2426-08-6	25	135	—	—	—	—	—
n-Butyl lactate	138-22-7	5	27	—	—	—	—	—
Butyl mercaptan	109-79-5	0.5	1.5	—	—	—	—	—
o-sec-Butylphenol	89-72-5	5	30	—	—	—	—	X
p-tert-Butyltoluene	98-51-1	10	60	20	120	—	—	—
Cadmium fume and dust (as Cd)[f]; see 55 FR 4052	7440-43-9	—	0.001 or 0.005	—	0.005 or 0.025			
Calcium carbonate	1317-65-3							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Calcium cyanamide	156-62-7	—	0.5	—	—	—	—	—
Calcium hydroxide	1305-62-0							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Calcium oxide	1305-78-8	—	5	—	—	—	—	—
Calcium silicate	1344-95-2							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Calcium sulfate	7778-18-9							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Camphor, synthetic	76-22-2	—	2	—	—	—	—	—
Caprolactam	105-60-2							
Dust		—	1	—	3	—	—	—
Vapor		5	20	10	40	—	—	—
Captafol (Difolatan)	2425-06-1	—	0.1	—	—	—	—	—
Captan	133-06-2	—	5	—	—	—	—	—
Carbaryl (Sevin)	63-25-2	—	5	—	—	—	—	—
Carbofuran (Furadan)	1563-66-2	—	0.1	—	—	—	—	—
Carbon black	1333-86-4	—	3.5	—	—	—	—	—
Carbon dioxide	124-38-9	10,000	18,000	30,000	54,000	—	—	—
Carbon disulfide	75-15-0	4	12	12	36	—	—	X
Carbon monoxide	630-08-0	35	40	—	—	200	229	—
Carbon tetrabromide	558-13-4	0.1	1.4	0.3	4	—	—	—
Carbon tetrachloride	56-23-5	2	12.6	—	—	—	—	
Carbonyl fluoride	353-50-4	2	5	5	15	—	—	—
Catechol (Pyrocatechol)	120-80-9	5	20	—	—	—	—	X
Cellulose	9004-34-6							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Cesium hydroxide	21351-79-1	—	2	—	—	—	—	—
Chlordane	57-74-9	—	0.5	—	—	—	—	X
Chlorinated camphene	8001-35-2	—	0.5	—	1	—	—	X
Chlorinated diphenyl oxide	55720-99-5	—	0.5	—	—	—	—	—
Chlorine	7782-50-5	0.5	1.5	1	3	—	—	
Chlorine dioxide	10049-04-4	0.1	0.3	0.3	0.9	—	—	—
Chlorine trifluoride	7790-91-2	—	—	—	—	0.1	0.4	
Chloroacetaldehyde	107-20-0	—	—	—	—	1	3	—
a-Chloroacetophenone (Phenacyl chloride)	532-27-4	0.05	0.3	—	—	—	—	—
Chloroacetyl chloride	79-04-9	0.05	0.2	—	—	—	—	—
Chlorobenzene	108-90-7	75	350	—	—	—	—	—
o-Chlorobenzylidene malononitrile	2698-41-1	—	—	—	—	0.05	0.4	X
Chlorobromomethane	74-97-5	200	1060	—	—	—	—	—

TABLE Z.—Agriculture—Continued

Substance	CAS No.[d]	Proposed Final Rule Limits						Skin Designation
		TWA		STEL		CEILING		
		ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	
2-Chloro-1,3-butadiene; see beta-Chloroprene								
Chlorodifluoromethane	75-45-6	1000	3500	—	—	—	—	—
Chlorodiphenyl (42% Chlorine) (PCB)	53469-21-9	—	1	—	—	—	—	X
Chlorodiphenyl (54% Chlorine) (PCB)	11097-69-1	—	0.5	—	—	—	—	X
1-Chloro,2,3-epoxypropane; see Epichlorohydrin								
2-Chloroethanol; see Ethylene chlorohydrin								
Chloroethylene; see Vinyl chloride								
Chloroform (Trichloromethane)	67-66-3	2	9.78	—	—	—	—	—
1-Chloro-1-nitropropane	600-25-9	2	10	—	—	—	—	—
Chloropentafluoroethane	76-15-3	1000	6320	—	—	—	—	—
Chloropicrin	76-06-2	0.1	0.7	—	—	—	—	—
beta-Chloroprene	126-99-8	10	35	—	—	—	—	X
o-Chlorostyrene	2039-87-4	50	285	75	428	—	—	—
o-Chlorotoluene	95-49-8	50	250	—	—	—	—	—
2-Chloro-6-(trichloromethyl) pyridine	1929-82-4							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Chlorpyrifos	2921-88-2	—	0.2	—	—	—	—	X
Chromic acid and chromates (as CrO_3)	Varies with compound	—	—	—	—	0.1	—	
Chromium (II) compounds (as Cr)	7440-47-3	—	0.5	—	—	—	—	—
Chromium (III) compounds (as Cr)	7440-47-3	—	0.5	—	—	—	—	—
Chromium metal (as Cr)	7440-47-3	—	1	—	—	—	—	—
Chrysene; see Coal tar pitch volatiles	—							
Clopidol	2971-90-6							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Coal dust (less than 5% SiO_2), Respirable fraction		—	2	—	—	—	—	
Coal dust (greater than or equal to 5% SiO_2), Respirable quartz fraction		—	0.1	—	—	—	—	
Coal tar pitch volatiles (benzene soluble fraction), anthracene, BaP, phenanthrene, acridine, chrysene, pyrene	65996-93-2	—	0.2	—	—	—	—	—
Cobalt metal, dust, and fume (as Co)	7440-48-4	—	0.05	—	—	—	—	—
Cobalt carbonyl (as Co)	10210-68-1	—	0.1	—	—	—	—	—
Cobalt hydrocarbonyl (as Co)	16842-03-8	—	0.1	—	—	—	—	—
Copper	7440-50-8							
Fume (as Cu)		—	0.1	—	—	—	—	—
Dusts and mists (as Cu)		—	1	—	—	—	—	—
Cotton dust[f]; see 1910.1043		—	0.5	—	—	—	—	—
This 8-hour TWA applies to respirable dust as measured by a vertical elutriator cotton dust sampler or equivalent instrument. Where it is not feasible to use vertical elutriator, a respirable dust personal sampler may be used. In these circumstances the exposure limit is 1 mg/m³ respirable dust, personal sampler.								
Crag herbicide (Sesone)	136-78-7							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Cresol, all isomers	1319-77-3	5	22	—	—	—	—	X
Crotonaldehyde	123-73-9; 4170-30-3	2	6	—	—	—	—	—
Crufomate	299-86-5	—	5	—	—	—	—	—
Cumene	98-82-8	50	245	—	—	—	—	X
Cyanamide	420-04-2	—	2	—	—	—	—	—
Cyanides (as CN)	Varies with Compound	—	5	—	—	—	—	—
Cyanogen	460-19-5	10	20	—	—	—	—	—
Cyanogen chloride	506-77-4	—	—	—	—	0.3	0.6	—
Cyclohexane	110-82-7	300	1050	—	—	—	—	—
Cyclohexanol	108-93-0	50	200	—	—	—	—	X
Cyclohexanone	108-94-1	25	100	—	—	—	—	X
Cyclohexene	110-83-8	300	1015	—	—	—	—	—
Cyclohexylamine	108-91-8	10	40	—	—	—	—	—
Cyclonite	121-82-4	—	1.5	—	—	—	—	X
Cyclopentadiene	542-92-7	75	200	—	—	—	—	—
Cyclopentane	287-92-3	600	1720	—	—	—	—	—
Cyhexatin	13121-70-5	—	5	—	—	—	—	—
2,4-D (Dichlorophenoxyacetic acid)	94-75-7	—	10	—	—	—	—	—
Decaborane	17702-41-9	0.05	0.3	0.15	0.9	—	—	X
Demeton (Systox)	8065-48-3	—	0.1	—	—	—	—	X
Diacetone alcohol (4-Hydroxy-4-methyl-2-pentanone)	123-42-2	50	240	—	—	—	—	—
1,2-Diaminoethane; see Ethylenediamine								
Diazinon	333-41-5	—	0.1	—	—	—	—	X
Diazomethane	334-88-3	0.2	0.4	—	—	—	—	—
Diborane	19287-45-7	0.1	0.1	—	—	—	—	—
1,2-Dibromo-3-chloropropane (DBCP)[f]; see 1910.1044	96-12-8	0.001	—	—	—	—	—	X
2-N-Dibutylaminoethanol	102-81-8	2	14	—	—	—	—	—
Dibutyl phosphate	107-66-4	1	5	2	10	—	—	—
Dibutyl phthalate	84-74-2	—	5	—	—	—	—	—
Dichloroacetylene	7572-29-4	—	—	—	—	0.1	0.4	—

TABLE Z.—Agriculture—Continued

Substance	CAS No.[a]	Proposed Final Rule Limits						Skin Designation
		TWA		STEL		CEILING		
		ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	
o-Dichlorobenzene	95-50-1	—	—	—	—	50	[illegible]	—
p-Dichlorobenzene	106-46-7	75	[illegible]	110	675	—	—	—
Dichlorodifluoromethane	75-71-8	[illegible]	4950	—	—	—	—	—
1,3-Dichloro-5,5-dimethyl hydantoin	118-52-5	—	0.2	—	0.4	—	—	—
Dichlorodiphenyltrichloroethane (DDT)	50-29-3	—	[illegible]	—	—	—	—	X
1,1-Dichloroethane	75-34-3	[illegible]	[illegible]	—	—	—	—	—
1,2-Dichloroethylene	540-59-0	[illegible]	790	—	—	—	—	—
Dichloroethyl ether	111-44-4	5	30	10	[illegible]	—	—	X
Dichloromethane; see Methylene chloride								
Dichloromonofluoromethane	75-43-4	10	40	—	—	—	—	—
1,1-Dichloro-1-nitroethane	594-72-9	[illegible]	10	—	—	—	—	—
1,2-Dichloropropane; see Propylene dichloride								
1,3-Dichloropropene	542-75-6	[illegible]	[illegible]	—	—	—	—	X
2,2-Dichloropropionic acid	75-99-0	[illegible]	6	—	—	—	—	—
Dichlorotetrafluoroethane	76-14-2	1000	7000	—	—	—	—	—
Dichlorvos (DDVP)	62-73-7	—	[illegible]	—	—	—	—	X
Dicrotophos	141-66-2	—	0.25	—	—	—	—	X
Dicyclopentadiene	77-73-6	[illegible]	30	—	—	—	—	—
Dicyclopentadienyl iron	102-54-5							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	[illegible]	—	—	—	—	—
Dieldrin	60-57-1	—	0.25	—	—	—	—	X
Diethanolamine	111-42-2	3	15	—	—	—	—	—
Diethylamine	109-89-7	10	30	25	75	—	—	—
2-Diethylaminoethanol	100-37-8	10	[illegible]	—	—	—	—	X
Diethylene triamine	111-40-0	[illegible]	4	—	—	—	—	—
Diethyl ether; see Ethyl ether								
Diethyl ketone	96-22-0	[illegible]	705	—	—	—	—	—
Diethyl phthalate	84-66-2	—	5	—	—	—	—	—
Difluorodibromomethane	75-61-6	100	[illegible]	—	—	—	—	—
Diglycidyl ether (DGE)	2238-07-5	0.1	0.5	—	—	—	—	—
Dihydroxybenzene; see Hydroquinone								
Diisobutyl ketone	108-83-8	[illegible]	150	—	—	—	—	—
Diisopropylamine	108-18-9	5	[illegible]	—	—	—	—	X
Dimethoxymethane; see Methylal								
Dimethyl acetamide	127-19-5	10	[illegible]	—	—	—	—	X
Dimethylamine	124-40-3	10	[illegible]	—	—	—	—	—
Dimethylaminobenzene; see Xylidine								
Dimethylaniline (N-Dimethylaniline)	121-69-7	5	[illegible]	10	50	—	—	X
Dimethylbenzene; see Xylene								
Dimethyl-1,2-dibromo-2,2-dichloroethyl phosphate	300-76-5	—	[illegible]	—	—	—	—	X
Dimethylformamide	68-12-2	10	[illegible]	—	—	—	—	X
2,6-Dimethyl-4-heptanone; see Diisobutyl ketone								
1,1-Dimethylhydrazine	57-14-7	0.5	[illegible]	—	—	—	—	X
Dimethylphthalate	131-11-3	—	[illegible]	—	—	—	—	—
Dimethyl sulfate	77-78-1	0.1	0.5	—	—	—	—	X
Dinitolmide (3,5-Dinitro-o-toluamide)	148-01-6	—	[illegible]	—	—	—	—	—
Dinitrobenzene (all isomers)		—	[illegible]	—	—	—	—	X
(ortho)	528-29-0							
(meta)	[illegible]							
(para)	100-25-4							
Dinitro-o-cresol	534-52-1	—	0.2	—	—	—	—	X
Dinitrotoluene	25321-14-6	—	1.5	—	—	—	—	X
Dioxane (Diethylene dioxide)	123-91-1	[illegible]	[illegible]	—	—	—	—	X
Dioxathion (Delnav)	78-34-2	—	0.2	—	—	—	—	X
Diphenyl (Biphenyl)	92-52-4	0.2	[illegible]	—	—	—	—	—
Diphenylamine	122-39-4	—	10	—	—	—	—	—
Diphenylmethane diisocyanate; see Methylene bisphenyl isocyanate								
Dipropylene glycol methyl ether	34590-94-8	100	[illegible]	150	[illegible]	—	—	X
Dipropyl ketone	123-19-3	50	[illegible]	—	—	—	—	—
Diquat	85-00-7	—	0.5	—	—	—	—	—
Di-sec octyl phthalate (Di-(2-ethylhexyl) phthalate)	117-81-7	—	5	—	10	—	—	—
Disulfiram	97-77-8	—	[illegible]	—	—	—	—	—
Disulfoton	298-04-4	—	0.1	—	—	—	—	X
2,6-Di-tert-butyl-p-cresol	128-37-0	—	10	—	—	—	—	—
Diuron	330-54-1	—	10	—	—	—	—	—
Divinyl benzene	1321-74-0	10	[illegible]	—	—	—	—	—
Emery	12415-34-8							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Endosulfan	115-29-7	—	0.1	—	—	—	—	X
Endrin	72-20-8	—	0.1	—	—	—	—	X
Epichlorohydrin	106-89-8	2	[illegible]	—	—	—	—	X
EPN	2104-64-5	—	0.5	—	—	—	—	X
1,2-Epoxypropane; see Propylene oxide								

TABLE Z.—Agriculture—Continued

Substance	CAS No.[d]	Proposed Final Rule Limits						Skin Designation
		TWA		STEL		CEILING		
		ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	
2,3-Epoxy-1-propanol; see Glycidol								
Ethanethiol; see Ethyl mercaptan								
Ethanolamine	141-43-5	3	8	6	15	—	—	—
Ethion	563-12-2	—	0.4	—	—	—	—	X
2-Ethoxyethanol (Cellosolve)	110-80-5	200	740	—	—	—	—	X
2-Ethoxyethyl acetate (Cellosolve acetate)	111-15-9	100	540	—	—	—	—	X
Ethyl acetate	141-78-6	400	1400	—	—	—	—	—
Ethyl acrylate	140-88-5	5	20	25	100	—	—	X
Ethyl alcohol (Ethanol)	64-17-5	1000	1900	—	—	—	—	—
Ethylamine	75-04-7	10	18	—	—	—	—	—
Ethyl amyl ketone (5-Methyl-3-heptanone)	541-85-5	25	130	—	—	—	—	—
Ethyl benzene	100-41-4	100	435	125	545	—	—	—
Ethyl bromide	74-96-4	200	890	250	1110	—	—	—
Ethyl butyl ketone (3-Heptanone)	106-35-4	50	270	—	—	—	—	—
Ethyl chloride	75-00-3	1000	2600	—	—	—	—	—
Ethyl ether	60-29-7	400	1200	500	1500	—	—	—
Ethyl formate	109-94-4	100	300	—	—	—	—	—
Ethyl mercaptan	75-08-1	0.5	1	—	—	—	—	—
Ethyl silicate	78-10-4	10	85	—	—	—	—	—
Ethylene chlorohydrin	107-07-3	—	—	—	—	1	3	X
Ethylenediamine	107-15-3	10	25	—	—	—	—	—
Ethylene dibromide	106-93-4	20	—	50 (5 min.)	—			
Ethylene dichloride	107-06-2	1	4	2	8	—	—	—
Ethylene glycol	107-21-1	—	—	—	—	50	125	—
Ethylene glycol dinitrate	628-96-6	—	—	—	0.1	—	—	X
Ethylene glycol methyl acetate; see Methyl cellosolve acetate								
Ethylene oxide [f]; see 1910.1047	75-21-8	1	—	5	—	—	—	—
Ethylidene chloride; see 1,1-Dichloroethane								
Ethylidene norbornene	16219-75-3	—	—	—	—	5	25	—
N-Ethylmorpholine	100-74-3	5	23	—	—	—	—	X
Fenamiphos	22224-92-6	—	0.1	—	—	—	—	X
Fensulfothion (Dasanit)	115-90-2	—	0.1	—	—	—	—	—
Fenthion	55-38-9	—	0.2	—	—	—	—	X
Ferbam	14484-64-1							
Total dust		—	10	—	—	—	—	—
Ferrovanadium dust	12604-58-9	—	1	—	3	—	—	—
Fibrous glass		—	1 f/cc	—	—	—	—	—
Fluorides (as F)	Varies with compound	—	2.5	—	—	—	—	—
Fluorine	7782-41-4	0.1	0.2	—	—	—	—	—
Fluorotrichloromethane (Trichlorofluoromethane)	75-69-4	—	—	—	—	1000	5600	—
Fonofos	944-22-9	—	0.1	—	—	—	—	X
Formaldehyde [f, h]; see 19190.1048; 56 FR 32302	50-00-0	0.75	—	2	—	—	—	—
Formamide	75-12-7	20	30	30	45	—	—	—
Formic acid	64-18-6	5	9	—	—	—	—	—
Furfural	98-01-1	2	8	—	—	—	—	X
Furfuryl alcohol	98-00-0	10	40	15	60	—	—	X
Gasoline	8006-61-9	300	900	500	1500	—	—	—
Germanium tetrahydride	7782-65-2	0.2	0.6	—	—	—	—	—
Glutaraldehyde	111-30-8	—	—	—	—	0.2	0.8	—
Glycerin (mist)	56-81-5							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Glycidol	556-52-5	25	75	—	—	—	—	—
Glycol monoethyl ether; see 2-Ethoxyethanol								
Grain dust (oat, wheat, barley)		—	10	—	—	—	—	—
Graphite, natural, respirable dust	7782-42-5	—	2.5	—	—	—	—	—
Graphite, synthetic	7782-42-5							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Guthion; see Azinphos methyl								
Gypsum	13397-24-5							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Hafnium	7440-58-6	—	0.5	—	—	—	—	—
Heptachlor	76-44-8	—	0.5	—	—	—	—	X
Heptane (n-Heptane)	142-82-5	400	1600	500	2000	—	—	—
Hexachlorobutadiene	87-68-3	0.02	0.24	—	—	—	—	—
Hexachlorocyclopentadiene	77-47-4	0.01	0.1	—	—	—	—	—
Hexachloroethane	67-72-1	1	10	—	—	—	—	X
Hexachloronaphthalene	1335-87-1	—	0.2	—	—	—	—	X
Hexafluoroacetone	684-16-2	0.1	0.7	—	—	—	—	X
n-Hexane	110-54-3	50	180	—	—	—	—	—

TABLE Z.—Agriculture—Continued

Substance	CAS No.[a]	Proposed Final Rule Limits						Skin Designation
		TWA		STEL		CEILING		
		ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	
Hexane isomers	Varies with compound	500	1800	1000	3600	—	—	—
2-Hexanone (Methyl n-butyl ketone)	591-78-6	5	20	—	—	—	—	—
Hexone (Methyl isobutyl ketone)	108-10-1	50	205	75	300	—	—	—
sec-Hexyl acetate	108-84-9	50	300	—	—	—	—	—
Hexylene glycol	107-41-5	—	—	—	—	25	125	—
Hydrazine	302-01-2	0.1	0.1	—	—	—	—	X
Hydrogenated terphenyls	61788-32-7	0.5	5	—	—	—	—	—
Hydrogen bromide	10035-10-6	—	—	—	—	3	10	—
Hydrogen chloride	7647-01-0	—	—	—	—	5	7	—
Hydrogen cyanide	74-90-8	—	—	4.7	5	—	—	X
Hydrogen fluoride (as F)	7664-39-3	3	—	6	—	—	—	—
Hydrogen peroxide	7722-84-1	1	1.4	—	—	—	—	—
Hydrogen selenide (as Se)	7783-07-5	0.05	0.2	—	—	—	—	—
Hydrogen sulfide	7783-06-4	10	14	15	21	—	—	—
Hydroquinone	123-31-9	—	2	—	—	—	—	—
2-Hydroxypropyl acrylate	999-61-1	0.5	3	—	—	—	—	X
Indene	95-13-6	10	45	—	—	—	—	—
Indium and compounds (as In)	7440-74-6	—	0.1	—	—	—	—	—
Iodine	7553-56-2	—	—	—	—	0.1	1	—
Iodoform	75-47-8	0.6	10	—	—	—	—	—
Iron oxide fume	1309-37-1	—	10	—	—	—	—	—
Iron pentacarbonyl (as Fe)	13463-40-6	0.1	0.8	0.2	1.6	—	—	—
Iron salts (soluble) (as Fe)	Varies with compound	—	1	—	—	—	—	—
Isoamyl acetate	123-92-2	100	525	—	—	—	—	—
Isoamyl alcohol (primary and secondary)	123-51-3	100	360	125	450	—	—	—
Isobutyl acetate	110-19-0	150	700	—	—	—	—	—
Isobutyl alcohol	78-83-1	50	150	—	—	—	—	—
Isooctyl alcohol	26952-21-6	50	270	—	—	—	—	X
Isophorone	78-59-1	4	23	—	—	—	—	—
Isophorone diisocyanate	4098-71-9	0.005	—	0.02	—	—	—	X
2-Isopropoxyethanol	109-59-1	25	105	—	—	—	—	—
Isopropyl acetate	108-21-4	250	950	310	1185	—	—	—
Isopropyl alcohol	67-63-0	400	980	500	1225	—	—	—
Isopropylamine	75-31-0	5	12	10	24	—	—	—
N-Isopropylaniline	768-52-5	2	10	—	—	—	—	X
Isopropyl ether	108-20-3	500	2100	—	—	—	—	—
Isopropyl glycidyl ether (IGE)	4016-14-2	50	240	75	360	—	—	—
Kaolin	1332-58-7							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Ketene	463-51-4	0.5	0.9	1.5	3	—	—	—
Lead, inorganic (as Pb)[f]; see 1910.1025	7439-92-1	—	0.05	—	—	—	—	—
Limestone	1317-65-3							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Lindane	58-89-9	—	0.5	—	—	—	—	X
Lithium hydride	7580-67-8	—	0.025	—	—	—	—	—
L.P.G. (Liquefied petroleum gas)	68476-85-7	1000	1800	—	—	—	—	—
Magnesite	546-93-0							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Magnesium oxide fume	1309-48-4							
Total particulate		—	10	—	—	—	—	—
Malathion	121-75-5							
Total dust		—	10	—	—	—	—	X
Maleic anhydride	108-31-6	0.25	1	—	—	—	—	—
Manganese compounds (as Mn)	7439-96-5	—	—	—	—	—	5	—
Manganese fume (as Mn)	7439-96-5	—	1	—	3	—	—	—
Manganese cyclopentadienyl tricarbonyl (as Mn)	12079-65-1	—	0.1	—	—	—	—	X
Manganese tetroxide (as Mn)	1317-35-7	—	1	—	—	—	—	—
Marble	1317-65-3							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Mercury (aryl and inorganic) (as Hg)	7439-97-6	—	—	—	—	—	0.1	X
Mercury (organo) alkyl compounds (as Hg)	7439-97-6	—	0.01	—	0.03	—	—	X
Mercury (vapor) (as Hg)	7439-97-6	—	0.05	—	—	—	—	X
Mesityl oxide	141-79-7	15	60	25	100	—	—	—
Methacrylic acid	79-41-4	20	70	—	—	—	—	X
Methanethiol; see Methyl mercaptan								
Methomyl (Lannate)	16752-77-5	—	2.5	—	—	—	—	—
Methoxychlor	72-43-5							
Total dust		—	10	—	—	—	—	—
2-Methoxyethanol (Methyl cellosolve)	109-86-4	20	60	—	—	—	—	X
2-Methoxyethanol acetate (Methyl cellosolve acetate)	110-49-6	25	120	—	—	—	—	X

TABLE Z.—Agriculture—Continued

Substance	CAS No.[a]	Proposed Final Rule Limits						Skin Designation
		TWA		STEL		CEILING		
		ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	
4-Methoxyphenol	150-76-5	—	5	—	—	—	—	—
Methyl acetate	79-20-9	200	610	250	760	—	—	—
Methyl acetylene (Propyne)	74-99-7	1000	1650	—	—	—	—	—
Methyl acetylene-propadiene mixture (MAPP)	—	1000	1800	1250	2250	—	—	—
Methyl acrylate	96-33-3	10	35	—	—	—	—	X
Methylacrylonitrile	126-98-7	1	3	—	—	—	—	X
Methylal (Dimethoxymethane)	109-87-5	1000	3100	—	—	—	—	—
Methyl alcohol	67-56-1	200	260	250	310	—	—	X
Methylamine	74-89-5	10	12	—	—	—	—	—
Methyl amyl alcohol; see Methyl isobutyl carbinol								
Methyl n-amyl ketone	110-43-0	100	465	—	—	—	—	—
Methyl bromide	74-83-9	5	20	—	—	—	—	X
Methyl butyl ketone; see 2-Hexanone								
Methyl cellosolve; see 2-Methoxyethanol								
Methyl cellosolve acetate; see 2-Methoxyethyl acetate								
Methyl chloride	74-87-3	50	105	100	210	—	—	—
Methyl chloroform (1,1,1-Trichloroethane)	71-55-6	350	1900	450	2450	—	—	—
Methyl 2-cyanoacrylate	137-05-3	2	8	4	16	—	—	—
Methylcyclohexane	108-87-2	400	1600	—	—	—	—	—
Methylcyclohexanol	25639-42-3	50	235	—	—	—	—	—
o-Methylcyclohexanone	583-60-8	50	230	75	345	—	—	X
Methylcyclopentadienyl manganese tricarbonyl (as Mn)	12108-13-3	—	0.2	—	—	—	—	X
Methyl demeton	8022-00-2	—	0.5	—	—	—	—	X
4,4'-Methylene bis (2-chloroaniline) (MBOCA)	101-14-4	0.02	0.22	—	—	—	—	X
Methylene bis (4-cyclo-hexylisocyanate)	5124-30-1	—	—	—	—	0.01	0.11	—
Methylene chloride [h]; see 56 FR 57036	75-09-22	25	—	125	—	—	—	—
Methyl ethyl ketone (MEK); see 2-Butanone								
Methyl ethyl ketone peroxide (MEKP)	1338-23-4	—	—	—	—	0.7	5	—
Methyl formate	107-31-3	100	250	150	375	—	—	—
Methyl hydrazine (Monomethyl hydrazine)	60-34-4	—	—	—	—	0.2	0.35	X
Methyl iodide	74-88-4	2	10	—	—	—	—	X
Methyl isoamyl ketone	110-12-3	50	240	—	—	—	—	—
Methyl isobutyl carbinol	108-11-2	25	100	40	165	—	—	X
Methyl isobutyl ketone; see Hexone								
Methyl isocyanate	624-83-9	0.02	0.05	—	—	—	—	X
Methyl isopropyl ketone	563-80-4	200	705	—	—	—	—	—
Methyl mercaptan	74-93-1	0.5	1	—	—	—	—	—
Methyl methacrylate	80-62-6	100	410	—	—	—	—	
Methyl parathion	298-00-0	—	0.2	—	—	—	—	X
Methyl propyl ketone; see 2-Pentanone								
Methyl silicate	681-84-5	1	6	—	—	—	—	—
alpha-Methyl styrene	98-83-9	50	240	100	485	—	—	—
Methylene bisphenyl isocyanate (MDI)	101-68-8	—	—	—	—	0.02	0.2	—
Metribuzin	21087-64-9	—	5	—	—	—	—	—
Mica; see Silicates								
Mineral wool		—	1 f/cc	—	—	—	—	
Molybdenum (as Mo)	7439-98-7							
Soluble compounds		—	5	—	—	—	—	—
Insoluble compounds								
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Monocrotophos (Azodrin)	6923-22-4	—	0.25	—	—	—	—	—
Monomethyl aniline	100-61-8	0.5	2	—	—	—	—	X
Morpholine	110-91-8	20	70	30	105	—	—	X
Naphtha (Coal tar)	8030-30-6	100	400	—	—	—	—	—
Naphthalene	91-20-3	10	50	15	75	—	—	—
Nickel, metal and insoluble compounds (as Ni)	7440-02-0	—	1	—	—	—	—	—
Nickel, soluble compounds (as Ni)	7440-02-0	—	0.1	—	—	—	—	—
Nicotine	54-11-5	—	0.5	—	—	—	—	X
Nitric acid	7697-37-2	2	5	4	10	—	—	—
Nitric oxide	10102-43-9	25	30	—	—	—	—	—
p-Nitroaniline	100-01-6	—	3	—	—	—	—	X
Nitrobenzene	98-95-3	1	5	—	—	—	—	X
p-Nitrochlorobenzene	100-00-5	—	1	—	—	—	—	X
Nitroethane	79-24-3	100	310	—	—	—	—	—
Nitrogen dioxide	10102-44-0	—	—	1	1.8	—	—	—
Nitrogen trifluoride	7783-54-2	10	29	—	—	—	—	—
Nitroglycerin	55-63-0	—	—	—	0.1	—	—	X
Nitromethane	75-52-5	100	250	—	—	—	—	—
1-Nitropropane	108-03-2	25	90	—	—	—	—	—
2-Nitropropane	79-46-9	10	35	—	—	—	—	—
N-Nitrosodimethylamine; see 1910.1016	62-79-9							
Nitrotoluene (all isomers)		2	11	—	—	—	—	X
o-isomer	88-72-2							
m-isomer	99-08-1							
p-isomer	99-99-0							

TABLE Z.—Agriculture—Continued

Substance	CAS No.[4]	Proposed Final Rule Limits						Skin Designation
		TWA		STEL		CEILING		
		ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	
Nitrotrichloromethane; see Chloropicrin								
Nonane	111-84-2	200	1050	—	—	—	—	—
Octachloronaphthalene	2234-13-1	—	0.1	—	0.3	—	—	X
Octane	111-65-9	300	1450	375	1800	—	—	—
Oil mist, mineral	8012-95-1	—	5	—	—	—	—	—
Osmium tetroxide (as Os)	20816-12-0	0.0002	0.002	0.0006	0.006	—	—	—
Oxalic acid	144-62-7	—	1	—	2	—	—	—
Oxygen difluoride	7783-41-7	—	—	—	—	0.05	0.1	—
Ozone	10028-15-6	0.1	0.2	0.3	0.6	—	—	—
Paraffin wax fume	8002-74-2	—	2	—	—	—	—	—
Paraquat, respirable dust	4685-14-7 1910-42-5 2074-50-2	—	0.1	—	—	—	—	X
Parathion	56-38-2	—	0.1	—	—	—	—	X
Particulates not otherwise regulated								
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
PCB; see chlorodiphenyl (42% and 54% chlorine)								
Pentaborane	19624-22-7	0.005	0.01	0.015	0.03	—	—	—
Pentachloronaphthalene	1321-64-8	—	0.5	—	—	—	—	X
Pentachlorophenol	87-86-5	—	0.5	—	—	—	—	X
Pentaerythritol	115-77-5							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Pentane	109-66-0	600	1800	750	2250	—	—	—
2-Pentanone (Methyl propyl ketone)	107-87-9	200	700	250	875	—	—	—
Perchloroethylene (Tetrachloroethylene)	127-18-4	25	170	—	—	—	—	—
Perchloromethyl mercaptan	594-42-3	0.1	0.8	—	—	—	—	—
Perchloryl fluoride	7616-94-6	3	14	6	28	—	—	—
Perlite	93763-70-3							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Petroleum distillates (Naphtha)		400	1600	—	—	—	—	—
Phenol	108-95-2	5	19	—	—	—	—	X
Phenothiazine	92-84-2	—	5	—	—	—	—	X
p-Phenylene diamine	106-50-3	—	0.1	—	—	—	—	X
Phenyl ether, vapor	101-84-8	1	7	—	—	—	—	—
Phenyl ether-biphenyl mixture, vapor		1	7	—	—	—	—	—
Phenylethylene; see Styrene								
Phenyl glycidyl ether (PGE)	122-60-1	1	6	—	—	—	—	—
Phenylhydrazine	100-63-0	5	20	10	45	—	—	X
Phenyl mercaptan	108-98-5	0.5	2	—	—	—	—	—
Phenylphosphine	638-21-1	—	—	—	—	0.05	0.25	—
Phorate	298-02-2	—	0.05	—	0.2	—	—	X
Phosdrin (Mevinphos)	7786-34-7	0.01	0.1	0.03	0.3	—	—	X
Phosgene (Carbonyl chloride)	75-44-5	0.1	0.4	—	—	—	—	—
Phosphine	7803-51-2	0.3	0.4	1	1	—	—	—
Phosphoric acid	7664-38-2	—	1	—	3	—	—	—
Phosphorus (yellow)	7723-14-0	—	0.1	—	—	—	—	—
Phosphorus oxychloride	10025-87-3	0.1	0.6	—	—	—	—	—
Phosphorus pentachloride	10026-13-8	—	1	—	—	—	—	—
Phosphorus pentasulfide	1314-80-3	—	1	—	3	—	—	—
Phosphorus trichloride	7719-12-2	0.2	1.5	0.5	3	—	—	—
Phthalic anhydride	85-44-9	1	6	—	—	—	—	—
m-Phthalodinitrile	626-17-5	—	5	—	—	—	—	—
Picloram	1918-02-1							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Picric acid	88-89-1	—	0.1	—	—	—	—	X
Piperazine dihydrochloride	142-64-3	—	5	—	—	—	—	—
Pindone (2-Pivalyl-1,3-indandione)	83-26-1	—	0.1	—	—	—	—	—
Plaster of Paris	26499-65-0							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Platinum (as Pt)	7440-06-4							
Metal		—	1	—	—	—	—	—
Soluble salts		—	0.002	—	—	—	—	—
Portland cement	65997-15-1							
Total dust		—	10	—	—	—	—	
Respirable fraction		—	5	—	—	—	—	
Potassium hydroxide	1310-58-3	—	—	—	—	—	2	—
Propargyl alcohol	107-19-7	1	2	—	—	—	—	X
beta-Propriolactone; see 1910.1013	57-57-8							
Propionic acid	79-09-4	10	30	—	—	—	—	—
Propoxur (Baygon)	114-26-1	—	0.5	—	—	—	—	—
n-Propyl acetate	109-60-4	200	840	250	1050	—	—	—

TABLE Z.—Agriculture—Continued

Substance	CAS No.[a]	Proposed Final Rule Limits						Skin Designation
		TWA		STEL		CEILING		
		ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	ppm [a]	mg/m [3] [b]	
n-Propyl alcohol	71-23-8	200	500	250	625	—	—	—
n-Propyl nitrate	627-13-4	25	105	40	170	—	—	—
Propylene dichloride	78-87-5	75	350	110	510	—	—	—
Propylene glycol dinitrate	6423-43-4	0.05	0.3	—	—	—	—	—
Propylene glycol monomethyl ether	107-98-2	100	360	150	540	—	—	—
Propylene imine	75-55-8	2	5	—	—	—	—	X
Propylene oxide	75-56-9	20	50	—	—	—	—	—
Propyne; see Methyl acetylene								
Pyrethrum	8003-34-7	—	5	—	—	—	—	—
Pyridine	110-86-1	5	15	—	—	—	—	—
Quinone	106-51-4	0.1	0.4	—	—	—	—	—
Resorcinol	108-46-3	10	45	20	90	—	—	—
Rhodium (as Rh), metal fume and insoluble compounds	7440-16-6	—	0.1	—	—	—	—	—
Rhodium (as Rh), soluble compounds	7440-16-6	—	0.001	—	—	—	—	—
Ronnel	299-84-3	—	10	—	—	—	—	—
Rosin core solder pyrolysis products, as formaldehyde		—	0.1	—	—	—	—	—
Rotenone	83-79-4	—	5	—	—	—	—	—
Rouge								
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Selenium compounds (as Se)	7782-49-2	—	0.2	—	—	—	—	—
Selenium hexafluoride (as Se)	7783-79-1	0.05	0.4	—	—	—	—	—
Silica, amorphous, precipitated and gel	112926-00-8	—	6	—	—	—	—	—
Silica, amorphous, diatomaceous earth, containing less than 1% crystalline silica	61790-53-2	—	6	—	—	—	—	
Silica, crystalline cristobalite, respirable dust	14464-46-1	—	0.05	—	—	—	—	—
Silica, crystalline quartz, respirable dust	14808-60-7	—	0.1	—	—	—	—	—
Silica, crystalline tripoli (as quartz), respirable dust	1317-95-9	—	0.1	—	—	—	—	—
Silica, crystalline tridymite, respirable dust	15468-32-3	—	0.05	—	—	—	—	—
Silica, fused, respirable dust	60676-86-0	—	0.1	—	—	—	—	—
Silicates (less than 1% crystalline silica)								
Mica (respirable dust)	12001-26	—	3	—	—	—	—	—
Soapstone, total dust		—	6	—	—	—	—	—
Soapstone, respirable dust		—	3	—	—	—	—	—
Talc (containing asbestos): use asbestos limit		—	0.2 f/cc	—	1 f/cc (30 min.)	—	—	—
Talc (containing no asbestos), respirable dust	14807-96-6	—	2	—	—	—	—	—
Tremolite; see 1910.1001 for asbestiform, and for non-asbestiform see 1910.1101								
Silicon	7440-21-3							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Silicon carbide	409-21-2							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Silicon tetrahydride	7803-62-5	5	7	—	—	—	—	—
Silver, metal and soluble compounds (as Ag)	7440-22-4	—	0.01	—	—	—	—	—
Soapstone; see Silicates								
Sodium azide	26628-22-8							
(as HN_3)		—	—	—	—	0.1	—	X
(as NaN_3)		—	—	—	—	—	0.3	X
Sodium bisulfite	7631-90-5	—	5	—	—	—	—	—
Sodium fluoroacetate	62-74-8	—	0.05	—	0.15	—	—	X
Sodium hydroxide	1310-73-2	—	—	—	—	—	2	—
Sodium metabisulfite	7681-57-4	—	5	—	—	—	—	—
Starch	9005-25-8							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Stibine	7803-52-3	0.1	0.5	—	—	—	—	—
Stoddard solvent	8052-41-3	100	525	—	—	—	—	—
Strychnine	57-24-9	—	0.15	—	—	—	—	—
Styrene	100-42-5-2	50	215	100	425	—	—	—
Subtilisins (Proteolytic enzymes)	9014-01-1	—	—	—	0.00006 [c] (60 min.)	—	—	—
Sucrose	57-50-1							
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Sulfur dioxide	7446-09-5	2	5	5	10	—	—	—
Sulfur hexafluoride	2551-62-4	1000	6000	—	—	—	—	—
Sulfuric acid	7664-93-9	—	1	—	—	—	—	—
Sulfur monochloride	10025-67-9	—	—	—	—	1	6	—
Sulfur pentafluoride	5714-22-7	—	—	—	—	0.01	0.1	—
Sulfur tetrafluoride	7783-60-0	—	—	—	—	0.1	0.4	—
Sulfuryl fluoride	2699-79-8	5	20	10	40	—	—	—

TABLE Z.—Agriculture—Continued

Substance	CAS No.[4]	Proposed Final Rule Limits						Skin Designation
		TWA		STEL		CEILING		
		ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	
Sulprofos	35400-43-2	—	1	—	—	-	—	—
Systox, see Demeton								
2,4,5-T (2,4,5-Trichlorophenoxyacetic acid)	93-76-5	—	10	—	—	—	—	—
Talc; see Silicates								
Tantalum, metal and oxide dust	7440-25-7	—	5	—	—	—	—	—
TEDP (Sulfotep)	3689-24-5	—	0.2	—	—	—	—	X
Tellurium and compounds (as Te)	13494-80-9	—	0.1	—	—	—	—	—
Tellurium hexafluoride (as Te)	7783-80-4	0.02	0.2	—	—	—	—	—
Temephos	3383-96-8							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
TEPP (Tetraethyl pyrophosphate)	107-49-3	—	0.05	—	—	—	—	X
Terphenyls	26140-60-3	—	—	—	—	0.5	5	—
1,1,1,2-Tetrachloro-2,2-difluoroethane	76-11-9	500	4170	—	—	—	—	—
1,1,2,2-Tetrachloro-1,2-difluoroethane	76-12-0	500	4170	—	—	—	—	—
1,1,2,2-Tetrachloroethane	79-34-5	1	7	—	—	—	—	X
Tetrachloroethylene; see Perchloroethylene								
Tetrachloromethane; see Carbon tetrachloride								
Tetrachloronaphthalene	1335-88-2	—	2	—	—	—	—	X
Tetraethyl lead (as Pb)	78-00-2	—	0.075	—	—	—	—	X
Tetrahydrofuran	109-99-9	200	590	250	735	—	—	—
Tetramethyl lead, (as Pb)	75-74-1	—	0.075	—	—	—	—	X
Tetramethyl succino-nitrile	3333-52-6	0.5	3	—	—	—	—	X
Tetranitromethane	509-14-8	1	8	—	—	—	—	—
Tetrasodium pyro-phosphate	7722-88-5	—	5	—	—	—	—	—
Tetryl (2,4,6-Trinitrophenylmethylnitramine)	479-45	—	1.5	—	—	—	—	X
Thallium, soluble compounds (as Tl)	7440-28-0	—	0.1	—	—	—	—	X
4,4'-Thiobis (6-tert, Butyl-m-cresol)	96-69-5							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Thioglycolic acid	68-11-1	1	4	—	—	—	—	X
Thionyl chloride	7719-09-7	—	—	—	—	1	5	—
Thiram	137-26-8	—	5	—	—	—	—	—
Tin, inorganic compounds (except oxides) (as Sn)	7440-31-5	—	2	—	—	—	—	—
Tin, organic compounds (as Sn)	7440-31-5	—	0.1	—	—	—	—	X
Tin oxide (as Sn)	21651-19-4	—	2	—	—	—	—	—
Titanium dioxide	13463-67-7							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Toluene	108-88-3	100	375	150	560	—	—	—
Toluene-2,4-diisocyanate (TDI)	584-84-9	0.005	0.04	0.02	0.15	—	—	—
m-Toluidine	108-44-1	2	9	—	—	—	—	X
o-Toluidine	95-53-4	5	22	—	—	—	—	X
p-Toluidine	106-49-0	2	9	—	—	—	—	X
Toxaphene; see Chlorinated camphene								
Tremolite; see Silicates								
Tributyl phosphate	126-73-8	0.2	2.5	—	—	—	—	—
Trichloroacetic acid	76-03-9	1	7	—	—	—	—	—
1,2,4-Trichlorobenzene	120-82-1	—	—	—	—	5	40	—
1,1,1-Trichloroethane; see Methyl chloroform								
1,1,2-Trichloroethane	79-00-5	10	45	—	—	—	—	X
Trichloroethylene	79-01-6-2	50	270	200	1080	—	—	—
Trichloromethane; see Chloroform								
Trichloronaphthalene	1321-65-9	—	5	—	—	—	—	X
1,2,3-Trichloropropane	96-18-4	10	60	—	—	—	—	—
1,1,2-Trichloro-1,2,2-trifluoroethane	76-13-1	1000	7600	1250	9500	—	—	—
Triethylamine	121-44-8	10	40	15	60	—	—	—
Trifluorobromomethane	75-63-8	1000	6100	—	—	—	—	—
Trimellitic anhydride	552-30-7	0.005	0.04	—	—	—	—	—
Trimethylamine	75-50-3	10	24	15	36	—	—	—
Trimethyl benzene	25551-13-7	25	125	—	—	—	—	—
Trimethyl phosphite	121-45-9	2	10	—	—	—	—	—
2,4,6-Trinitrophenyl; see Picric acid								
2,4,6-Trinitrophenylmethylnitramine; see Tetryl								
2,4,6-Trinitrotoluene (TNT)	118-96-7	—	0.5	—	—	—	—	X
Triorthocresyl phosphate	78-30-8	—	0.1	—	—	—	—	X
Triphenyl amine	603-34-9	—	5	—	—	—	—	—
Triphenyl phosphate	115-86-6	—	3	—	—	—	—	—
Tungsten (as W)	7440-33-7							
Insoluble compounds		—	5	—	10	—	—	—
Soluble compounds		—	1	—	3	—	—	—
Turpentine	8006-64-2	100	560	—	—	—	—	—
Uranium (as U)	7440-61-1							
Soluble compounds		—	0.05	—	—	—	—	—
Insoluble compounds		—	0.2	—	0.6	—	—	—
n-Valeraldehyde	110-62-3	50	175	—	—	—	—	—

TABLE Z.—Agriculture—Continued

Substance	CAS No.[d]	Proposed Final Rule Limits						Skin Designation
		TWA		STEL		CEILING		
		ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	ppm[a]	mg/m³[b]	
Vanadium	1314-62-1							
Respirable dust (as V_2O_5)		—	0.05	—	—	—	—	—
Fume (as V_2O_5)		—	0.05	—	—	—	—	—
Vegetable oil mist								
Total dust		—	15	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Vinyl acetate	108-05-4	10	30	20	60	—	—	—
Vinyl benzene; see Styrene								
Vinyl bromide	593-60-2	5	20	—	—	—	—	—
Vinyl chloride[c]; see 1910.1017	75-01-4	1	—	5	—	—	—	—
Vinyl cyanide; see Acrylonitrile								
Vinyl cyclohexene dioxide	106-87-6	10	60	—	—	—	—	X
Vinylidene chloride (1,1-Dichloroethylene)	75-35-4	1	4	—	—	—	—	—
Vinyl toluene	25013-15-4	100	480	—	—	—	—	—
VM & P Naphtha	8032-32-4	300	1350	400	1800	—	—	—
Warfarin	81-81-2	—	0.1	—	—	—	—	—
Welding fumes (total particulate)**		—	5	—	—	—	—	—
Wood dust, all soft and hard woods, except Western red cedar		—	5	—	10	—	—	—
Wood dust, Western red cedar		—	2.5	—	—	—	—	—
Xylenes (o-, m-, p- isomers)	1330-20-7	100	435	150	655	—	—	—
m-Xylene alpha, alpha'-diamine	1477-55-0	—	—	—	—	—	0.1	X
Xylidine	1300-73-8	2	10	—	—	—	—	X
Yttrium	7440-65-5	—	1	—	—	—	—	—
Zinc chloride fume	7646-85-7	—	1	—	2	—	—	—
Zinc chromate (as CrO_3)	13530-65-9	—	—	—	—		0.1	—
Zinc oxide fume	1314-13-2	—	5	—	10	—	—	—
Zinc oxide	1314-13-2							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Zinc stearate	557-05-1							
Total dust		—	10	—	—	—	—	—
Respirable fraction		—	5	—	—	—	—	—
Zirconium compounds (as Zr)	7440-67-7	—	5	—	10	—	—	—

Footnotes:

** As determined by breathing-zone air samples.

[a] Parts of vapor or gas per million parts of contaminated air by volume at 25°C and 760 torr.

[b] Milligrams of substance per cubic meter of air. When entry is in this column only, the value is exact; when listed with a ppm entry, it is approximate.

[c] Duration is for 15 minutes, unless otherwise noted.

[d] The CAS number is for information only. Enforcement is based on the substance name. For an entry covering more than one metal compound, measured as the metal, the CAS number for the metal is given—not CAS numbers for the individual compounds.

[e] Compliance with the subtilisins PEL is assessed by sampling with a high volume sampler (600–800 liters per minute) for at least 60 minutes.

[f] The reference to the comprehensive standard is for informational purposes and is not binding in agriculture. Only the exposure limits and 1928.1000 are binding in agriculture.

[g] For sectors excluded from 1910.1028, the limit is 10 ppm and see 1910.1000, Table Z-2 for ceiling limits.

[h] Where OSHA has published a proposal for a substance separate from this rulemaking but has not issued a final rule. The proposal is referenced, and the proposed final limit is placed in the table. However, this is presented just for information purposes, and the substances are not being considered in this rulemaking.

XI. Appendices

For "Sampling and Analytical Methods" see 54 FR 2961–2983, January 19, 1989. Additional and updated material on this issue will be found in the docket established for this rulemaking (H–020–A).

[FR Doc. 92–12903 Filed 6–11–92; 8:45 am]

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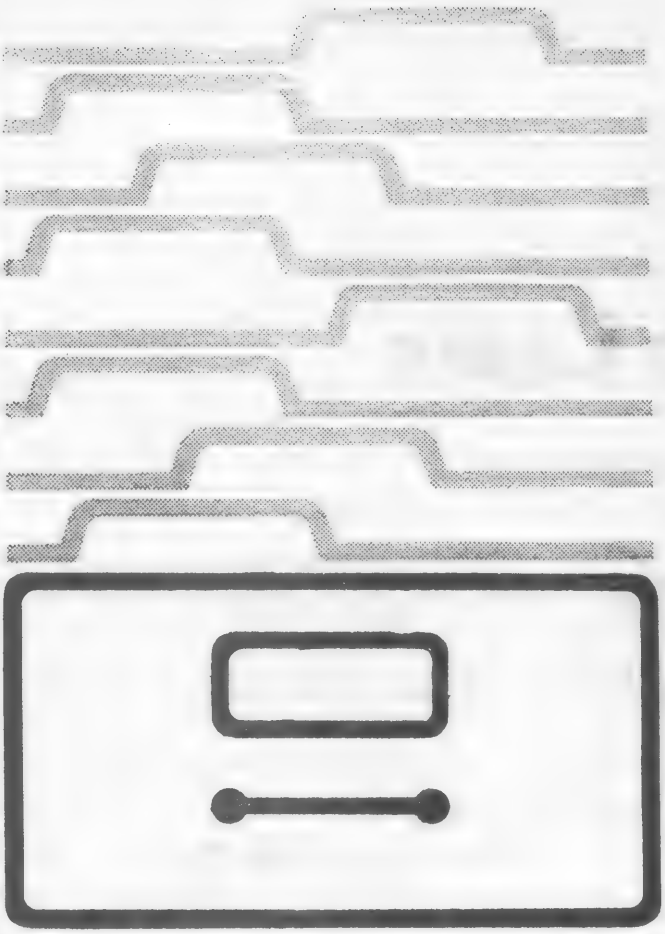

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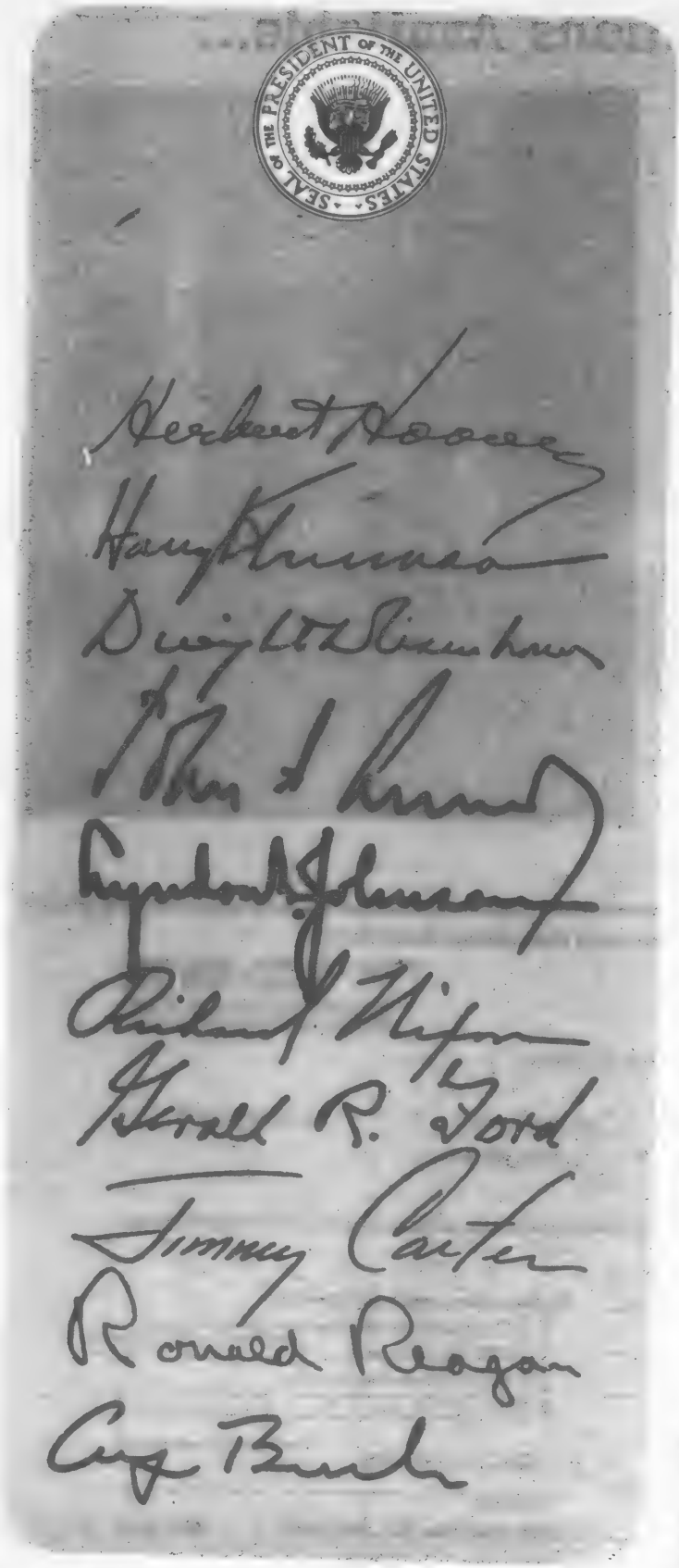

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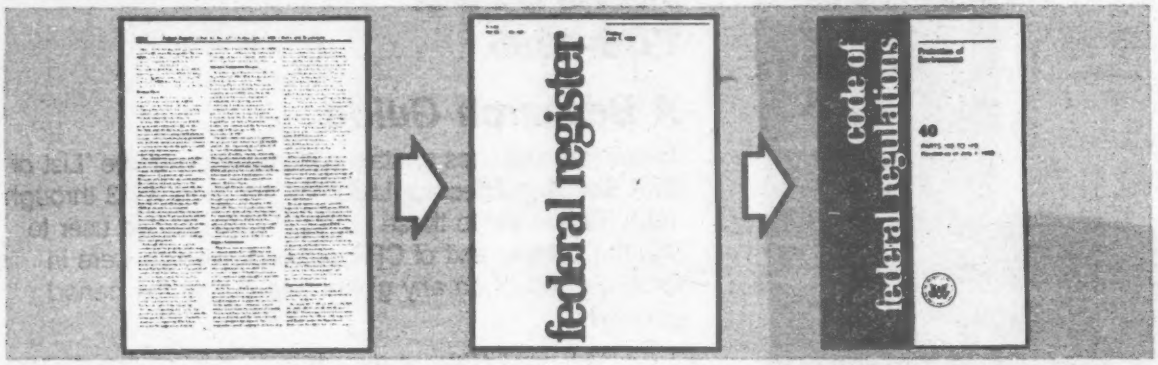

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